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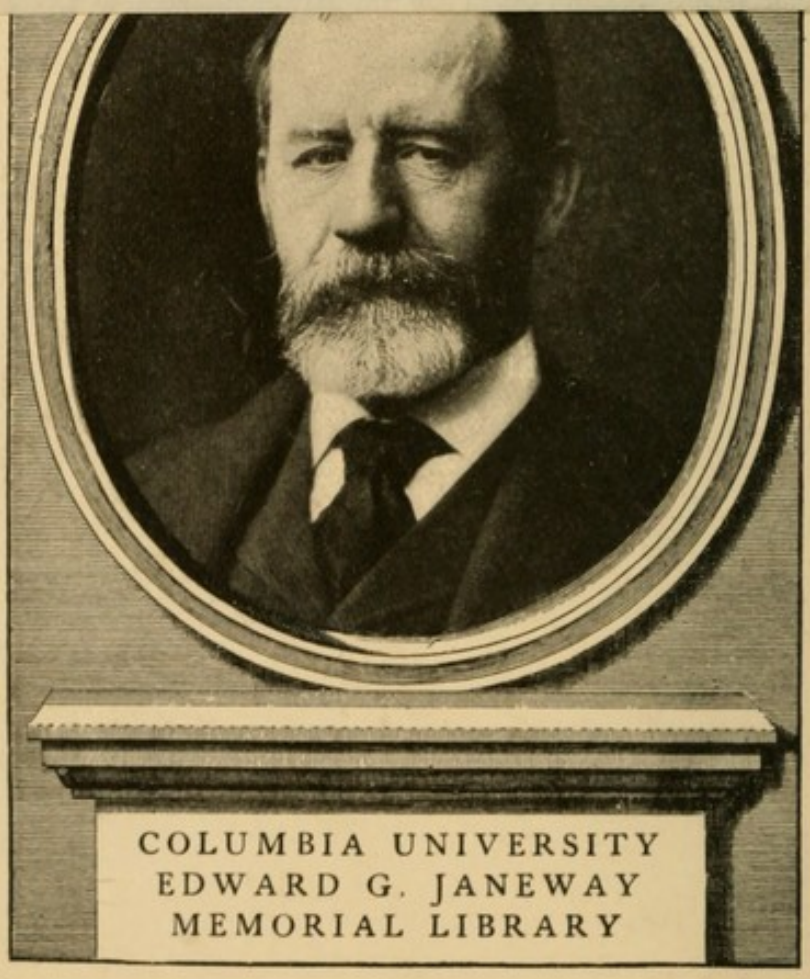
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DIAGNOSIS OF ORGANIC NERVOUS DISEASES

BY

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WITH 109 ILLUSTRATIONS

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PREFACE TO THE SECOND EDITION.

THE uninterrupted demand for the first edition of this little manual as a guide in the teaching of the diagnosis of nervous diseases has led me to believe that a revised and modernized edition would prove welcome. As my interest in medicine has gradually become concentrated on other fields than those of neurology, it has seemed wise to entrust the work of revision to one identified with the teaching and practice of neurology. Dr. L. Pierce Clark, who has undertaken the revision, has contributed much that is new to the edition now offered to the profession, without, however, altering or extending the scope of the earlier publication. Dr. Clark has rewritten in greater part the chapter on the anatomy and physiology of the nervous system, and has modernized the section dealing with the symptomatology of nervous diseases. The chapter on the examination of the patient has been wholly rewritten. These and the new illustrations added will be found, it is believed, to contribute a revision that is thorough without being destructive of the original plan of the book. It has been thought desirable to alter the title of the volume in order to express more plainly its preponderant relation to organic nervous diseases.

C. A. HERTER.

NEW YORK, Jan. 1907.

PREFACE.

THIS volume is designed to aid the student and general practitioner in the recognition of the commoner forms of nervous disease. While the book is in every respect elementary in character, it is believed that the subject of which it treats is presented with sufficient detail to make it serviceable.

The arrangement that has been followed in the presentation of facts is somewhat unusual and requires notice. In the chapter on the structure and functions of the nervous system no effort has been made to give a systematic outline of the anatomy of the brain and spinal cord. Indeed, little else has been attempted than the presentation of those facts regarding the motor and sensory paths that are of practical importance in diagnosis.

The chapter on symptomatology contains a description of the most important symptoms of nervous disease, and, in most cases, the chief facts regarding their diagnostic significance and pathology. In the chapter on localization many of the facts given under symptomatology have been restated. The justification of this repetition is found in the belief that it is very desirable to emphasize the more important relations between symptoms and lesions by changing the point of view.

In the fourth chapter, on the diagnosis of the nature of the lesion, an attempt is made, first, to give the main facts (from the standpoint of diagnosis) in the pathology

of the different classes of lesions, and, secondly, to show the manner of employing the indications that are derived from the onset, from etiology, and from the position of the lesion. In discussing the indications derived from etiology (causal indications) considerable space has been devoted to the enumeration of the varieties of disease that may result from different causes, in the belief that a knowledge of these facts will frequently help in forming a conclusion as to the nature of the lesion.

Next follows the chapter on clinical types, in which, while the types are necessarily somewhat rigidly drawn, an attempt is made to impress the fact that here, as in the classification of all natural phenomena, we must be prepared to recognize numerous variations in type and transitional forms.

No subject is more important or more difficult to treat satisfactorily than the distinction of functional and organic disease. An endeavor is made, in the sixth chapter, to present the chief points of distinction between different forms of functional disease and the organic conditions with which they may be confounded. It was considered advisable, on the whole, to make a rather sharp separation between the traumatic neurosis and hysteria.

The propriety of including in a work of so elementary a nature a short chapter on the examination of the patient will hardly be questioned.

The illustrations of diagnosis which are given in the eighth chapter are designed to aid the student in the application of the methods of diagnosis to actual cases of disease.

C. A. HERTER.

839 MADISON AVENUE,
March 7, 1892.

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DIAGNOSIS OF ORGANIC NERVOUS DISEASES

DIAGNOSIS OF ORGANIC NERVOUS DISEASES

CHAPTER I.

THE STRUCTURE AND FUNCTIONS OF THE NERVOUS SYSTEM.

Introductory Remarks.—Simple dissection of the cerebro-spinal axis in the fresh state reveals little beyond the coarse arrangement of the gray and white masses. But in specimens that have been prepared by careful maceration in alcohol or a solution of bichromate of potash, it is possible to remove with a forceps bundle after bundle of fibres from without inward, and with such nicety that the general course and relative disposition of many such bundles or tracts may be readily demonstrated. Indeed, in the hands of Meynert, Burdach, Foville, and others, this method of cleavage or teasing has yielded results of the highest importance. Still, for the elucidation of the more minute relations of the gray matter and the complicated systems of fibres, it is not wholly adequate. Nor is it possible, with the microscope and modern methods of staining cells and fibres, to determine precisely their connections or to

unravel the network of interlacing fibres, for at best only the structure of isolated elements is revealed by such means.

Fortunately, there are other methods of research. Disease in man and physiological experiment in the lower animals aid us in the investigation and differentiation of the more intricate and finer relations of gray matter and fibre tracts, and of the latter to one another. For when nerve-fibres are separated from their nutritive centres (the ganglion-cells), either by some morbid process, as frequently occurs in man, or by actual section of a nerve in a lower animal, such fibres degenerate in the portion cut off from the ganglion-cells by the lesion, and hence definite bundles of fibres may often be found altered in structure throughout their whole extent. On dissection the degenerated portion becomes visible to the naked eye by its change of color, and under the microscope the altered structure of the fibres is very marked. This process is known as secondary degeneration. The descending degeneration of the motor tract in the brain and in the lateral and anterior columns of the cord following coarse lesions in the cerebro-spinal axis is an example of this process, the fibres of the motor tract being separated from their nutritive ganglion-cells in the cerebral cortex.

Again, after tearing out accessible nerves and their end-organs, such as the optic nerve and the eye, in newly born animals, certain groups of ganglion-cells undergo a sort of retardation in development and an atrophy from non-use; and when, in other living animals, such groups of cells are experimentally removed, the fibres leading thence to the end-organ degenerate. This method of investigation is known as Gudden's atrophy method. While extremely useful, it is as yet rather restricted in

its application, since the lower animals only can be directly experimented upon, and the facts thus ascertained cannot always be properly transferred to human physiological anatomy.

Still another method of great value is one developed by Flechsig and originally suggested by Meynert. Certain tracts of nerve-fibres in the central nervous system have been found to develop their myeline sheaths at different periods of time. For instance, the fibres of the posterior nerve-roots receive their myeline sheaths as early as the seventh month of fœtal life, while those of the motor tract do not become perfected until after birth. The microscope easily differentiates such bundles, and even to the naked eye the medullated fibres, by their white color, contrast strongly in transverse section with the gray non-medullated fibres.

The functions of the central nervous system have been studied by experimental stimulation and destruction of certain portions of the brain and cord in lower animals, but the results of such experiments are not always applicable to man. Most of our knowledge of cerebral and spinal localization has been acquired by the careful observation of the symptoms of organic lesions in man, and their comparison with the pathological conditions observed at autopsies.

Investigations in the realm of histology have, in recent years, added much to our knowledge of the finer nervous anatomy. In this field Golgi and Ramon y Cajal have rendered inestimable service and have made possible a fuller conception of the anatomical structure of the central nervous system embodied in the neuron theory suggested by Waldeyer. This theory pictures the central nervous system as composed of various systems of units, the cell and fibre of the older parlance being combined in the

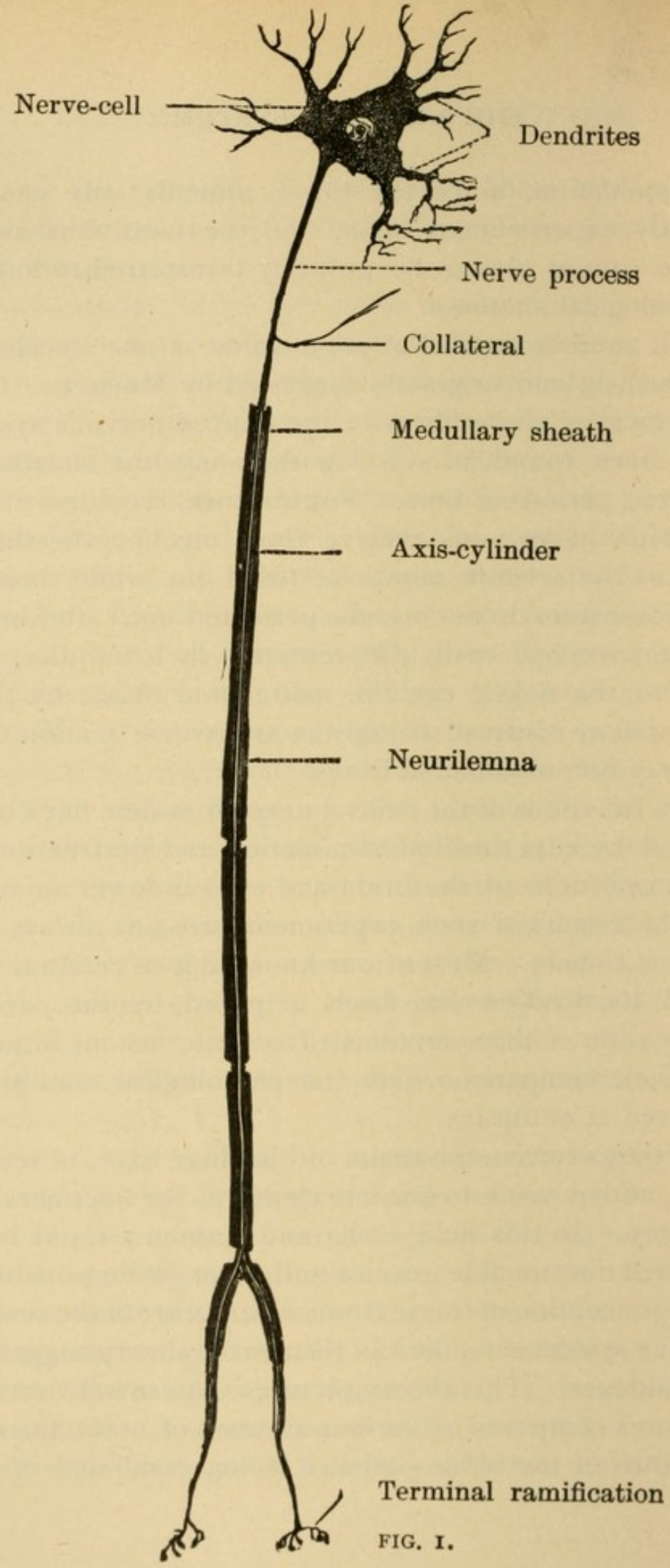


FIG. 1.

Diagram of a neuron. (Stohr.)

neuron of the new. In the neuron therefore are combined (a) the cell body containing various anatomical structures including the nucleus and nucleolus, (b) the axis-cylinder process which is the means of transmitting impulses from one set of neurons to another and the band by which the neurons are chained together into systems, and (c) the dendrites which are the other processes from the cell body whose function it probably is to perceive or apprehend impulses and carry them over from other neurons. The dendrites thus serve for the reception and diffusion of sensations while their combination and extension may be conceived as taking place in the cell body and their ultimate transmission from higher to lower groups of neurons is accomplished through the axis-cylinder process.

This conception of the nerve-cell and -fibre as constituting a single organ, the neuron, and of the central nervous system as being made up of groups of these organs which are in themselves separate and distinct, has not met with entire acceptance and corroboration, although its main features are well founded. Bethe and Apathy claim to have demonstrated histologically a fibre continuity between two separate neurons, which they think sufficiently demonstrates the non-existence of the neuron as an independent unit; while Held and more recently Nissl have put forward the theory of the great functional importance of the intercellular substance and have observed its minute fibrillary structure. Whether or not these views shall, in the future, be proven correct is immaterial for the present purpose. If established they may be accepted as corollaries to the neuron conception, while its principle will remain. For the purposes of this book, therefore, we shall accept the conceptions of the neuron theory.

Before considering the chief facts in the functional anatomy of the nervous system, certain general preliminary points should be reviewed.

The chief structural element underlying the physiological activity of the central nervous system is the

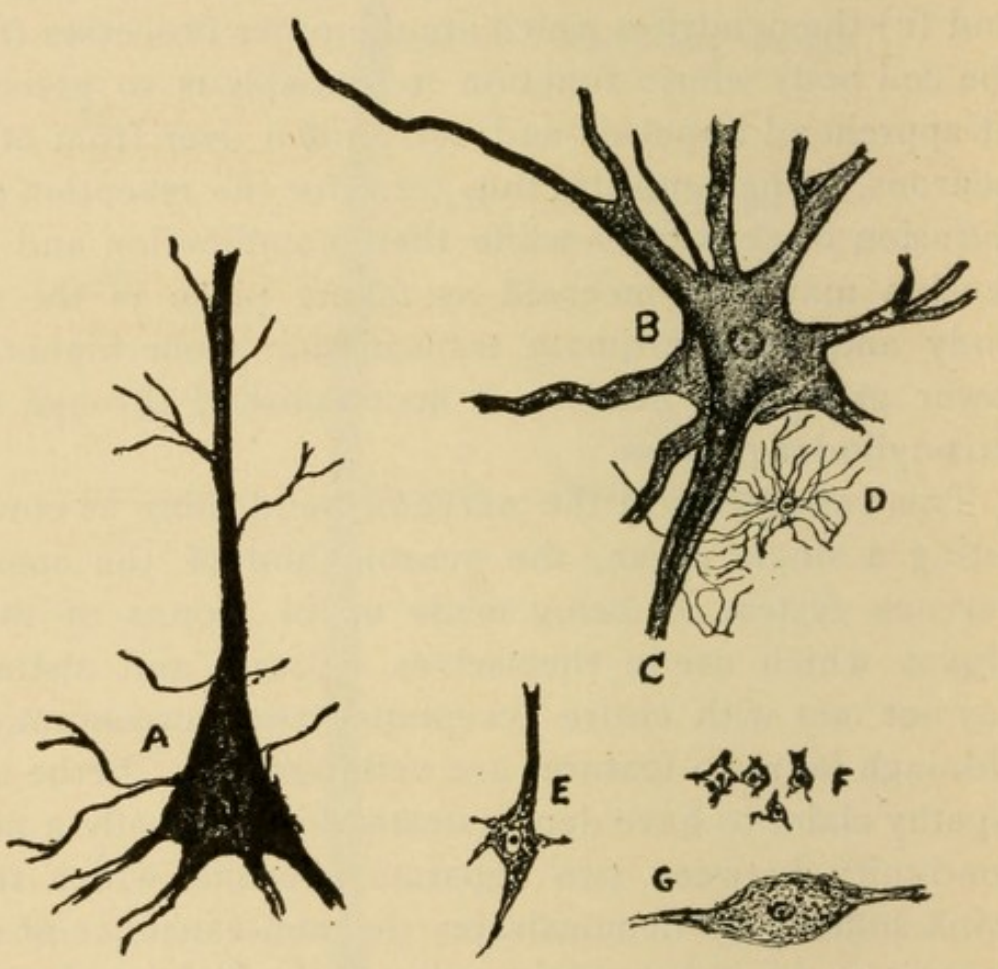


FIG. 2.

Diagram showing various forms of ganglion- or nerve-cells. A, pyramidal ganglion-cells from the human motor-cortex; B, multipolar ganglion-cell from anterior horn of spinal cord; C, axis-cylinder from B; D, neuroglia cell ("spider" cell); E, spindle-cell from human cortex; F, granule-cells from cortex; G, bipolar cells from spinal ganglion.

neuron consisting of a nerve-cell and its processes. The reception, perception, combination, correlation, and transmission of the impulse have been referred to. The great variations of the neuron in size and

shape are illustrated herewith. Many qualifying names have been applied to them, such as angular, granular, pyramidal, ganglion, multipolar, globose, round, spindle-shaped, etc., all of which are being superseded by terms applying more directly to a given neuron system. The nerve-fibre or axis-cylinder process of the neuron lacks

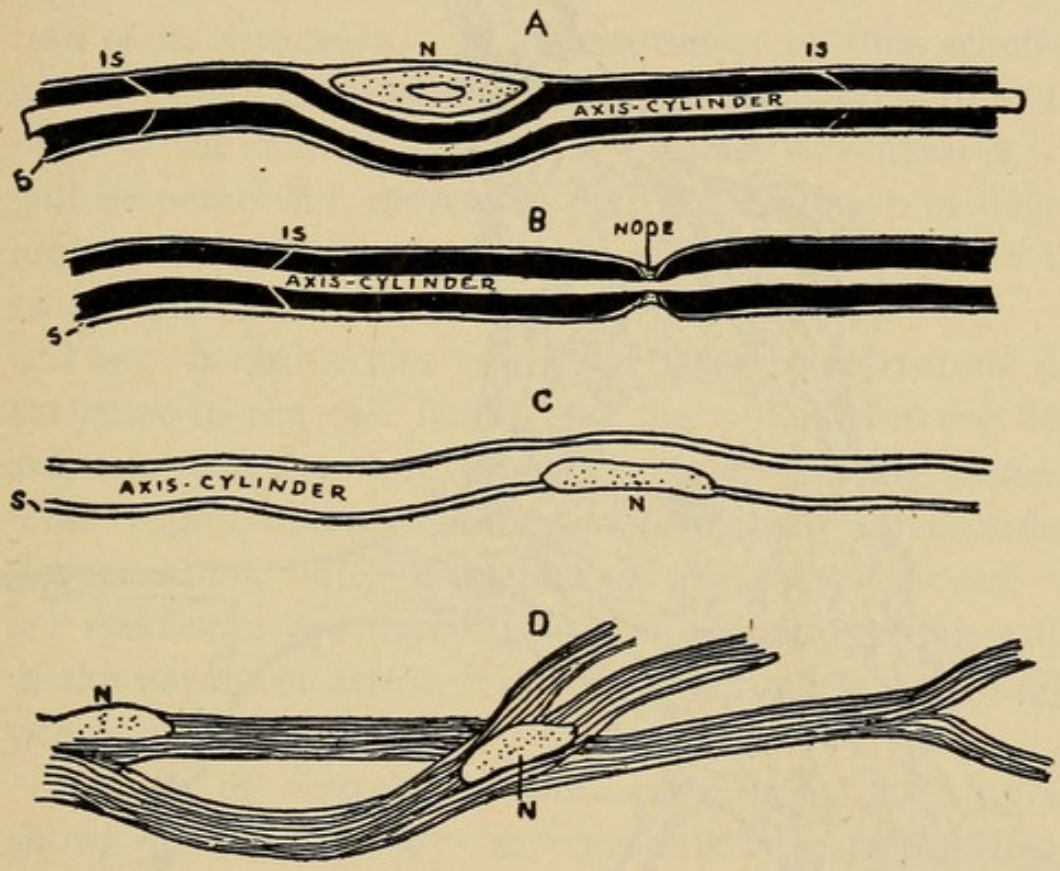


FIG. 3.

Diagram to illustrate the more important varieties of nerve-fibres. A and B, medullated nerve-fibres. S, sheath of Schwann. The myeline sheath lies between the sheath of Schwann and the axis-cylinder. N, nucleus of the sheath of Schwann. IS, incisures of Schmidt. Node, constriction separating two segments of a nerve-fibre. C, a nerve-fibre without a medullary sheath. D, fibre of Remak, from sympathetic.

uniformity of structure. It occurs either as a naked axis-cylinder, or an axis-cylinder clothed with a myeline sheath. Some of these forms of fibres are figured above, and the highest form of fibre is shown with its sheath of

Schwann, myeline sheath, axis-cylinder, Ranvier's nodes, and Schmidt's incisions (Fig. 3).

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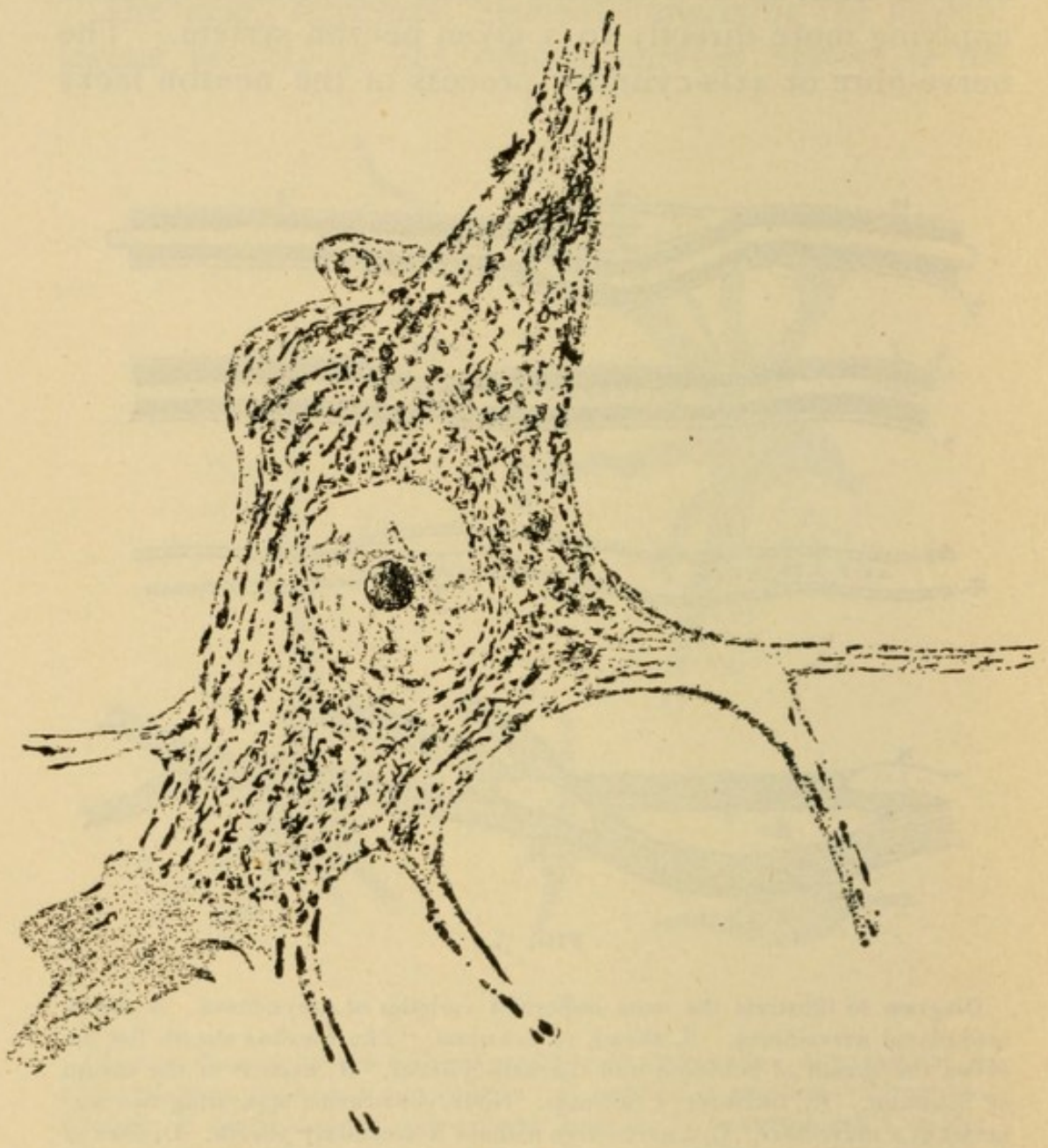


FIG. 4.

Normal Betz cell from the human paracentral lobule. (Meyer.)

neurons of varying types and their delicate appendages, there exists a tissue known as the neuroglia which is composed of branching cells and fibres derived from

these cells, whose function is probably a purely supportive one (see Fig 2). In addition to these there are other non-branching cells existing chiefly in the cerebral cortex and whose function is probably that of a support to the more delicate nervous structures.

Besides originating and receiving nervous impulses the neuron exercises a nutritional influence over every portion of its structure. In consequence of this, when an axis-cylinder process or a dendrite is severed from the body of the neuron, the distal portion degenerates. It will be perceived, therefore, that the direction of degeneration in a nerve-fibre does not necessarily follow the course and direction of the impulse.

There is also in the course of time some trophic disturbance in the cell itself, and the portion of the fibre still connected with it, especially in very young animals. This trophic change is allied histologically to secondary degeneration, but is not precisely the same process. It is a species of dystrophy from non-use, and partakes also of the nature of arrest of development when it occurs in young animals.

Groups of neurons of the larger type, such as are shown in Fig. 4, may be considered as subserving a motor function wherever met with. The figure represents one of the so-called Betz cells found in and characteristic of certain portions of the motor cortex, but similar neurons occur in the anterior horns of the spinal cord, here constituting the lower or second set of neurons in the motor system. Groups of neurons such, for example, as those above designated are commonly called "centres." This term is employed correctly only in a physiological sense. We mean thereby any group of neurons, not necessarily possessing well-defined topographical limits, which act conjointly in a functional

capacity. Sometimes such groups are widely separated, although functionally associated. They are therefore regarded as constituting a functional centre.

There is nothing in the anatomical features of nerve-fibres to give us a clue to the nature of their functional activity. Their axis-cylinders, apparently insulated by myeline sheaths, seem to be conductors of nerve force. The axis-cylinders vary in size, but the significance of such variation has not been satisfactorily explained. Many of the largest certainly belong to fibre systems of great length, but we are not yet warranted in assuming any definite relation of the breadth to the length of these elements.

The body of the neuron and its axis-cylinder process contribute respectively the gray and white matter of the cerebro-spinal axis. The gray matter completely envelops the hemispheres of the brain, forming a layer known as the cortex, the seat of the higher mental functions. There are also large masses of gray matter at the base of the brain in the basal ganglia, and the nuclear cells of the cranial nerves are scattered through the interior of the crura, pons, and medulla. In the cerebellum the gray matter has a very complex arrangement, while in the cord, on transverse section, it is seen to have an H-like contour as it lies imbedded in the midst of columns of white fibres.

The Brain.—The accompanying illustrations offer a readier means of acquiring a knowledge of the topographical distribution of the chief convolutions and fissures of the cerebrum than would a description in the text. The cortex varies in depth and structure in different regions, so that no less than eight distinct types of cortical lamination have been noted by some investigators. For instance, in some regions there are but three

layers in the cortex, in others seven. The neurons vary greatly in type and size in the various layers and regions of the cortex. The largest are found in the motor convolutions.

These differences of structure subserve differences in function, and modern research has localized, more or less perfectly, areas for voluntary motion, speech, cutaneous

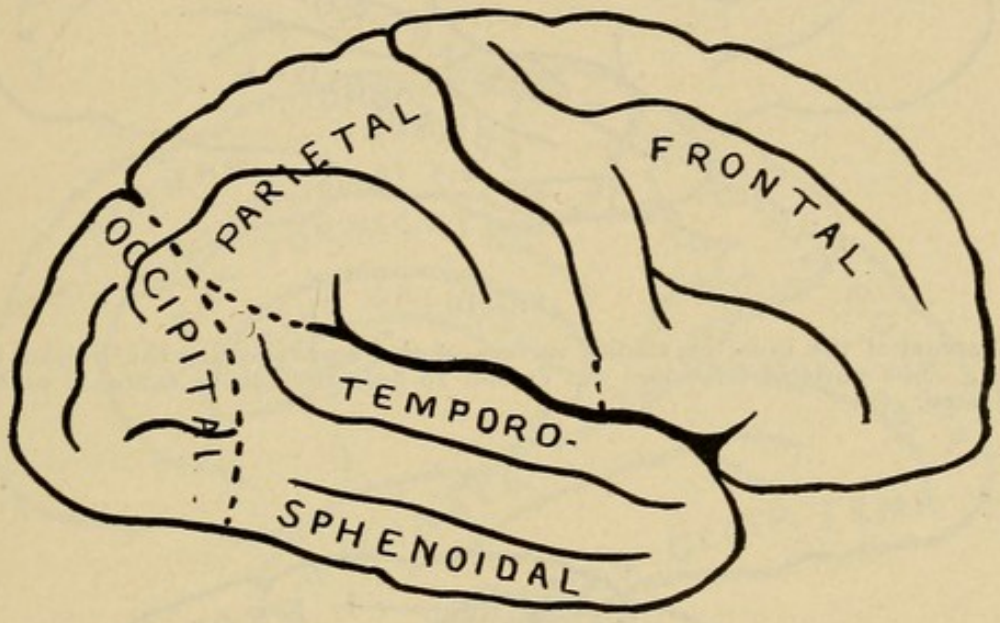


FIG. 5.

Diagram showing the division of the brain into lobes, outer surface, and of right hemisphere.

sensation, visual, auditory, and olfactory impressions, and has outlined with some degree of certainty regions which subserve the higher intellectual operations. The accompanying diagrams indicate with sufficient clearness the relative positions of these regions.

It is well to bear in mind that besides the fibres passing out from these cortical areas to the periphery of the body, there are numerous bundles of fibres which connect with one another different lobes and different convolutions, and join certain parts of the cortex with certain

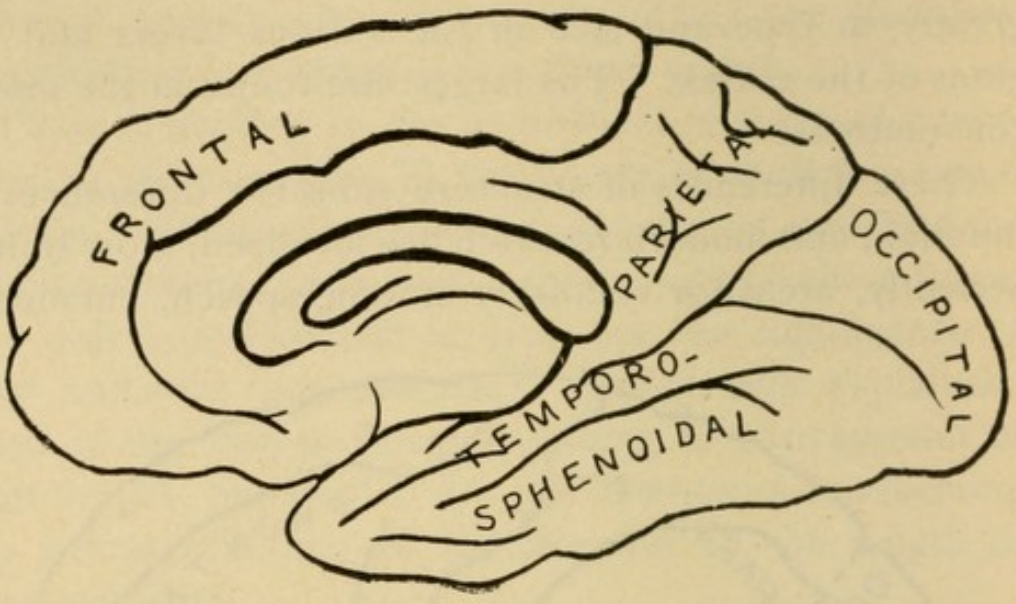


FIG. 6.

Diagram of the inner or medial surface of the brain showing the division into lobes. The parietal lobe does not extend so near the corpus callosum as here indicated.

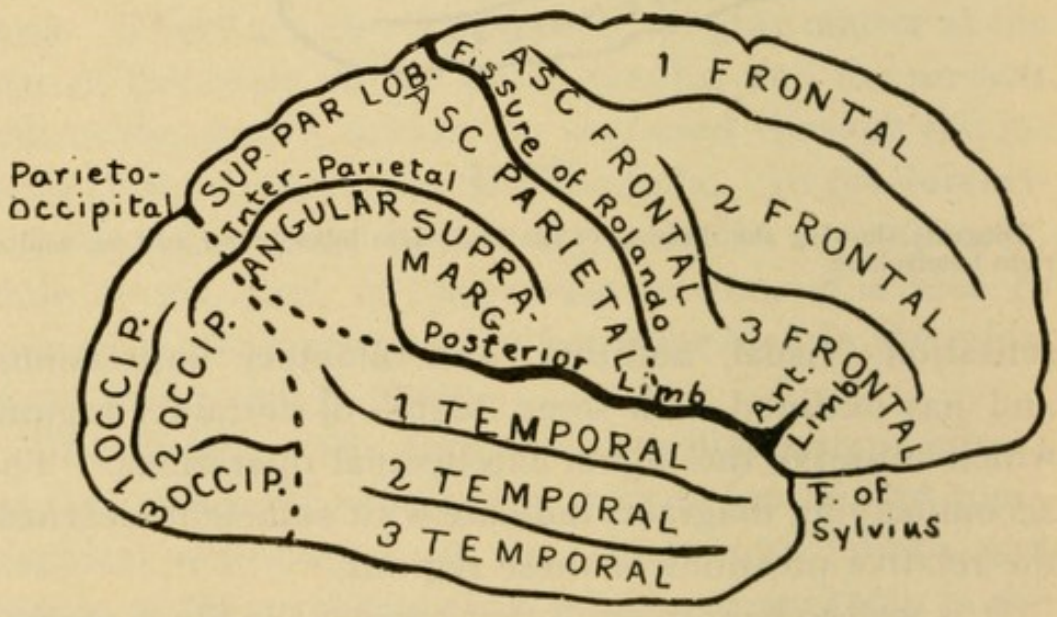


FIG. 7.

Diagram showing the chief convolutions and fissures on the outer surface of the right hemisphere.

of the basal ganglia and the cerebellum. The former fibres, which in a manner project the organism upon the cortex, as the world is projected upon a map, are often termed "projection" fibres; while most of the latter, which undoubtedly serve to bring various parts of the brain into conjoint and harmonious action, are known as "association" fibres (see Fig 15).

The two cerebral hemispheres are associated in func-

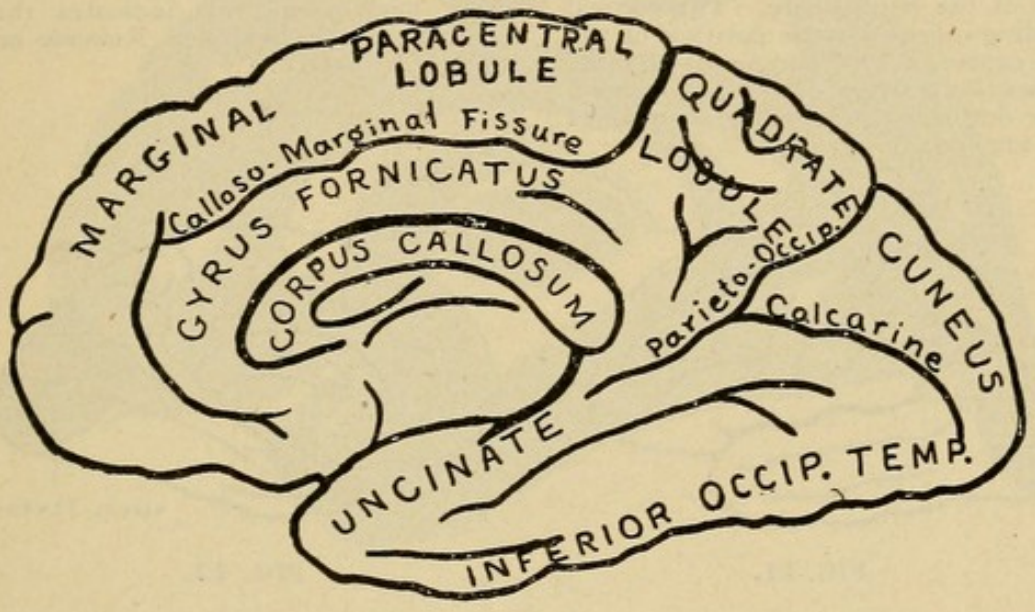


FIG. 8.

Diagram showing the chief convolutions and fissures on the inner or medial surface of the right hemisphere.

tion by the large bundles of fibres passing from the cortex of one side to that of the other, and constituting the corpus callosum. In addition to this great commissure there are three small ribands, known as the anterior, middle, and posterior commissures, in the third ventricle, which are to a certain extent bonds of union between the two halves of the brain. Of these commissures, the anterior is the most important, and is largely connected with the olfactory system, while the middle is of little or no importance.

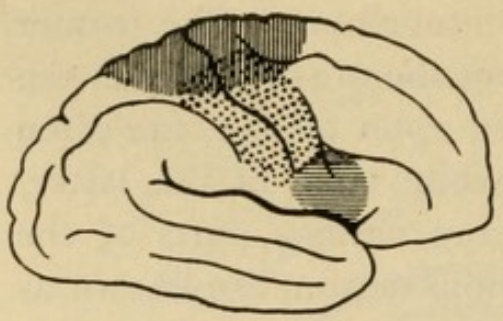


FIG. 9.

Diagram showing the position of the cortical motor area on the outer surface of the hemisphere. The vertical shading represents the position of the leg centre. The horizontal shading shows the position of the face centre. The dotted area shows the position of the arm centre.

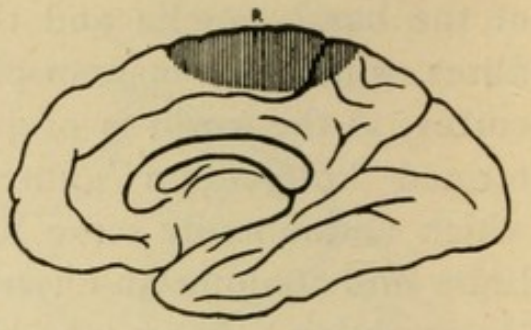


FIG. 10.

Diagram showing the position of the leg centre on the inner surface of the right hemisphere. R indicates the position of the fissure of Rolando on the outer surface.

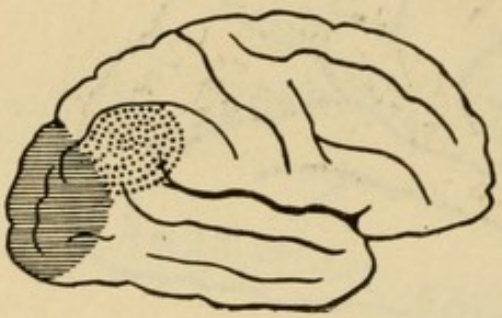


FIG. 11.

Diagram showing the cortical visual centres on the outer surface of the right hemisphere. The horizontal lines show the approximate position of the half-vision centre. The dotted areas show the position of the supposed higher visual centre.

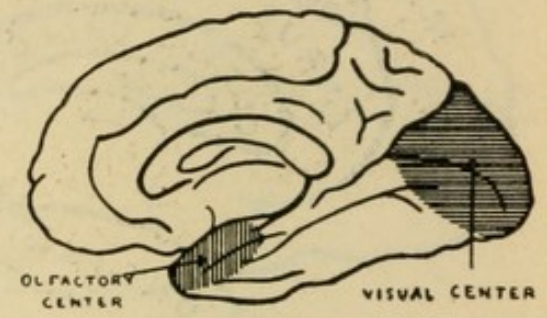


FIG. 12.

Diagram showing the position of the visual (half-vision) centre and the probable position of the olfactory centre on the inner surface of the right hemisphere.

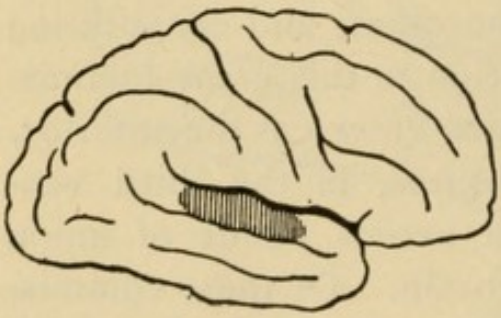


FIG. 13.

Diagram showing the position of the auditory centre in the first temporal convolution.

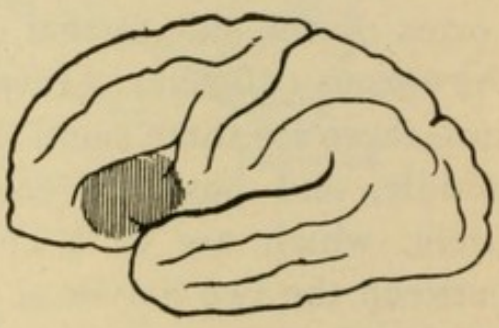


FIG. 14.

Diagram showing the position of the motor speech centre in the left hemisphere.

Very little is known concerning the functions of the caudate nuclei. The lenticular nucleus and the optic thalamus are of great importance as primary terminals of the great sensory systems from the periphery. The optic thalamus is of especial importance in this particular. Moreover, this mass forms a primary termination

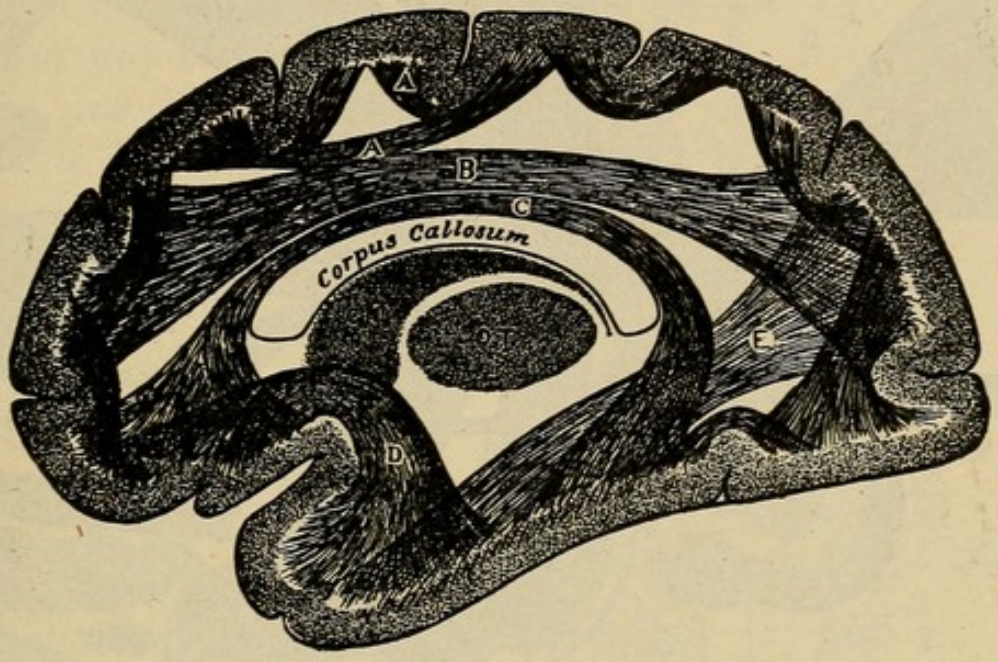


FIG. 15.

The association fibres. A, between adjacent convolutions; B, between frontal and occipital areas; C, between frontal and temporal areas, cingulum; D, between frontal and temporal areas, fasciculus uncinatus; E, between occipital and temporal areas, fasciculus longitudinalis inferior; C N, caudate nucleus; O T, optic thalamus. (Starr.)

for ocular and auditory fibres from the periphery and is connected with the visual and auditory areas of the cerebral cortex. It may, therefore, be regarded as the great way station for the entire sensory system. The anterior bodies of the corpora quadrigemina are associated with the function of sight, while the posterior are associated with the function of hearing. These bodies also have other functions not well known.

All the fibres connecting the periphery with the brain hemispheres pass through the crura cerebri (the olfactory

and optic tracts excepted). There are in addition special bundles of fibres connecting the cerebrum and cerebel-

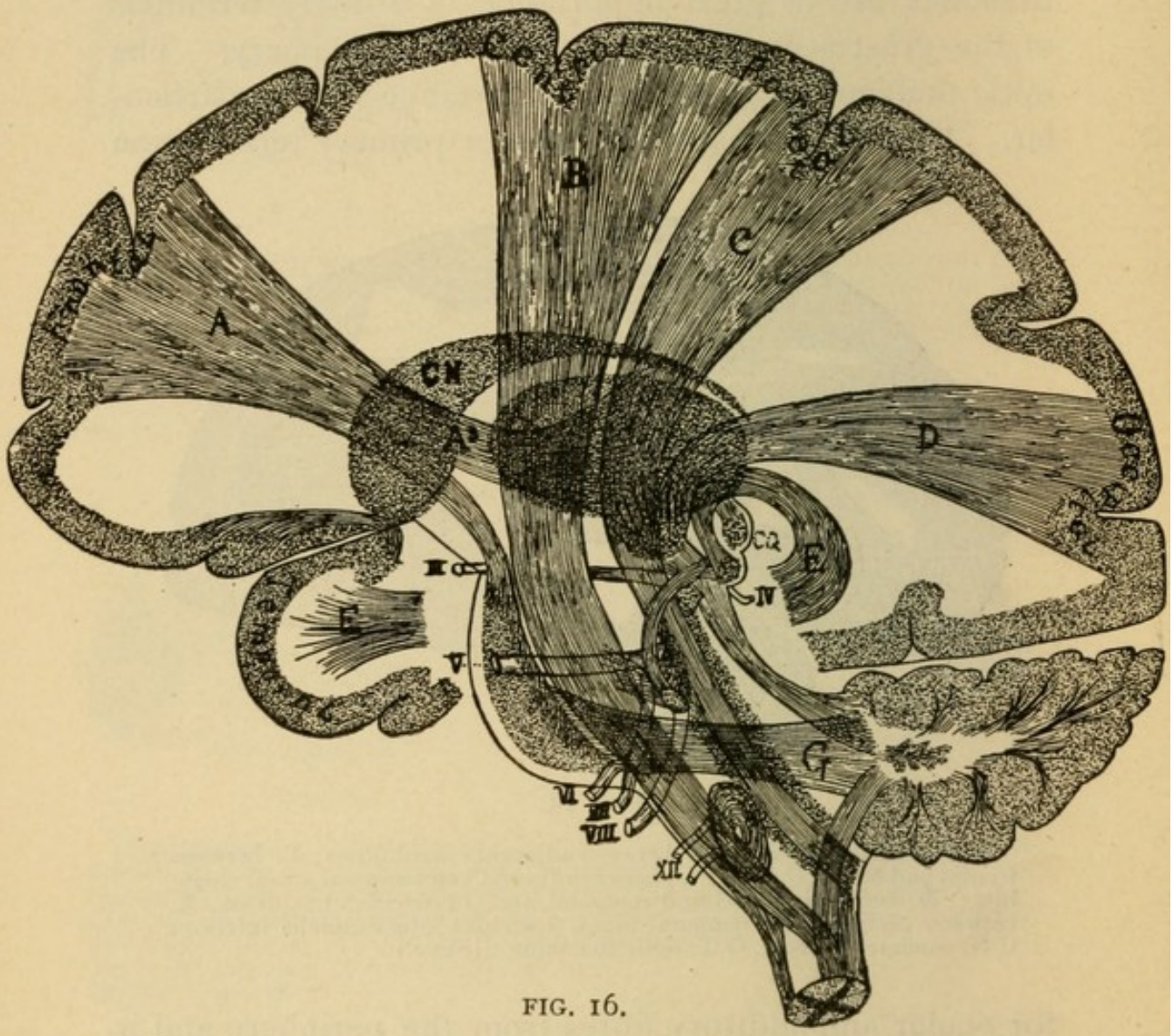


FIG. 16.

The projection tracts joining the cortex with lower nerve centres. Sagittal section showing the arrangements of tracts in the internal capsule. A, tract from the frontal lobe to the anterior half of the capsule, thence in part to the optic thalamus, A^2, and in part to the pons, and thus to the cerebellar hemisphere of the opposite side; B, motor tract from the central convolutions to the facial nucleus in the pons and to the spinal cord; C, sensory tract from posterior columns of the cord, through the posterior part of the medulla, pons, crus, and capsule to the parietal lobe; D, visual tract from the optic thalamus (O T) to the occipital lobe; E, auditory tract from the int. geniculate body (to which a tract passes from the VIII. n. nucleus) to the temporal lobe; F, superior cerebellar peduncle; G, middle cerebellar peduncle; H, inferior cerebellar peduncle; C N. caudate nucleus; C Q, corpora quadrigemina. The numerals refer to the cranial nerves. (Starr.)

lum, the fronto-, occipito-, and temporo-pontine tracts, which ultimately terminate in the cortex of the great

lobes of the cerebellum by means of ponto-cerebellar fibres which pass out through the middle cerebellar peduncles.

In a transverse section through the crura the various tracts and nuclei lie as shown in diagram 19. The third nerve nucleus lies in the floor of the aqueduct of Sylvius, and the fibres of the nerve pass out through the crus on their way to the orbit.

The practical and useful facts in the anatomy of the pons and medulla relate especially to the disposition of the cranial nerve-nuclei and certain bundles of fibres. The superficial origin of the cranial nerves is seen in Fig. 17. A knowledge of the relative positions of their nuclei is best gathered from the accompanying diagrams (18 and 19).

The relations of certain of the cranial nerves to the motor tract will be treated of in connection with the description of the latter.

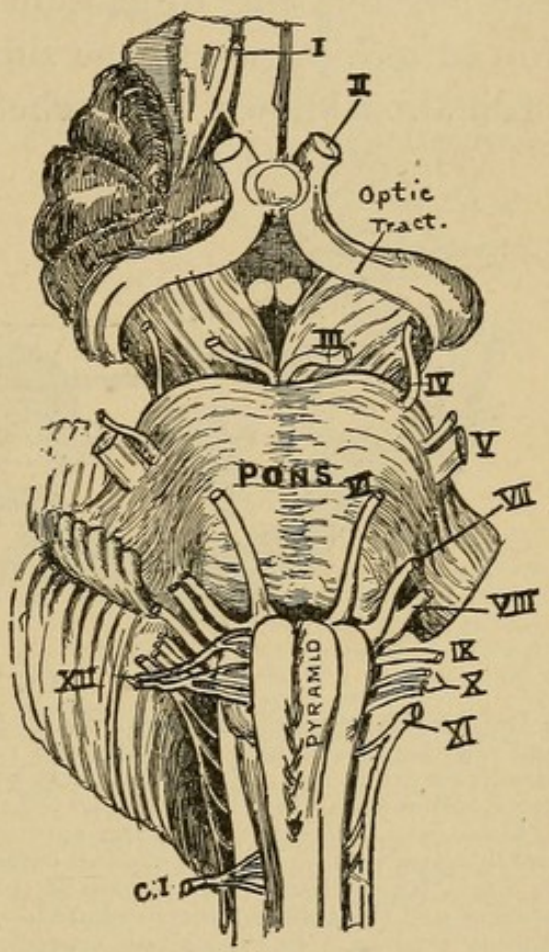


FIG. 17.

The base of the brain and the cranial nerves.

The superficial anatomy of the cerebellum is portrayed in the two accompanying illustrations (Figs. 20, 21) of its dorsal and ventral aspects. Each hemisphere of the cerebellum is joined to the mid-brain, to the pons, and to the medulla by three large diverging bundles of fibres, known respectively as the superior, middle, and inferior cerebellar peduncles. The peduncles are shown in Fig. *22*.

We are still greatly in the dark as regards the physiology of the cerebellum. Disease here so easily produces indirect effects upon the pons and medulla that the actual focal symptoms are often obscured. But there is undoubted evidence of the relation of the organ to bodily equilibrium, for disease in a peduncle may produce forced movements to one side or the other, and the incoordination known as cerebellar staggering or titubation

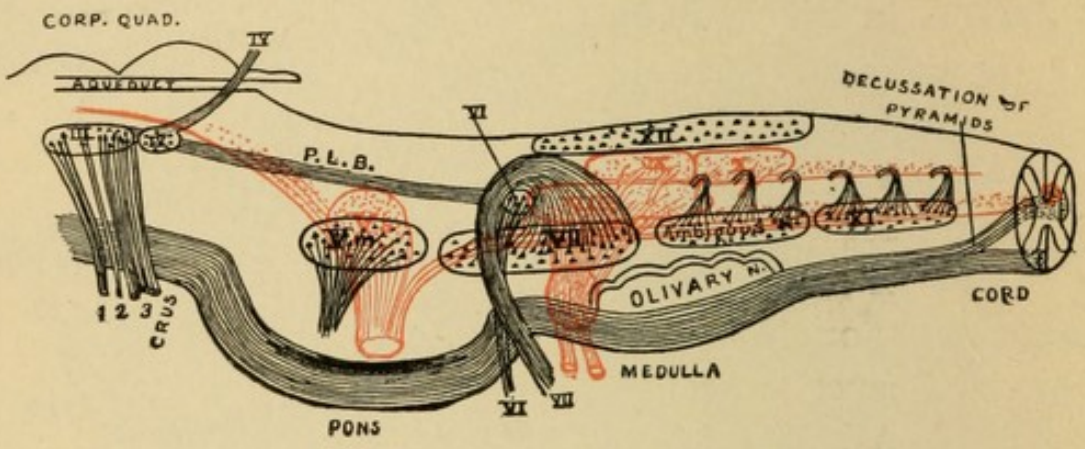


FIG. 18.

Diagram showing the nuclear origin of the cranial nerves; the medulla, pons, and crus being seen from the side, and represented as transparent. The sensory structures are in red. III, oculo-motor nucleus—1, fibres from the cells that subserve accommodation; 2, fibres from cells subserving the reflex action of the iris; 3, fibres from cells innervating the external muscles of the eye. P. L. B., posterior longitudinal bundle associating certain cells of the IIId nucleus with cells of the VIth nucleus. The portion of this bundle which lies between the VIth nucleus and the cord is not represented. Vm, motor nucleus of trigeminus. Vs, sensory nucleus. The ascending sensory root of the 5th is reprinted as coming from the substantia gelatinosa of the cord. The descending or trophic root arises in the quadrigeminal region. VII, facial nucleus. VIII, auditory nuclei. IX, glosso-pharyngeal nucleus. X, vagus nucleus. The roots of the vagus receive filaments from the nucleus ambiguus. XI, spinal accessory nucleus. XII, hypoglossal nucleus.

is due to disturbance of the middle portion of the cerebellum, the vermis, or worm. There is some evidence that it influences the power of muscular movements, and there is also some probability that this organ forms a substratum for some of the intellectual processes, through its intimate connection with the frontal lobes of the cerebrum by means of the fronto-cerebellar tract of fibres

(sometimes called "intellectual tract"). (*Vide* Fig. 22.)

For further information as to the disposition of the various parts of the brain, the reader is referred to works on the descriptive anatomy of the brain.

The Spinal Cord.—The spinal cord averages from seventeen to eighteen inches in length, and extends from the superior plane of the atlas to the second lumbar vertebra. The spinal canal is some nine or ten inches longer than the cord, reaching as it does through the remaining lumbar vertebræ and sacrum to the coccyx. The segments of the cord are short, and each pair of spinal nerves takes a downward direction from the segment before it reaches the intervertebral foramen. The intraspinal course of the nerves is longer the lower we descend the cord, so that the lumbar and sacral pairs form the large bundle of nerves known as the cauda equina (horse-tail) occupying the lower end of the spinal canal. The cord is not of uniform diameter, for there is an intumescence in the cervical cord, called the cervical

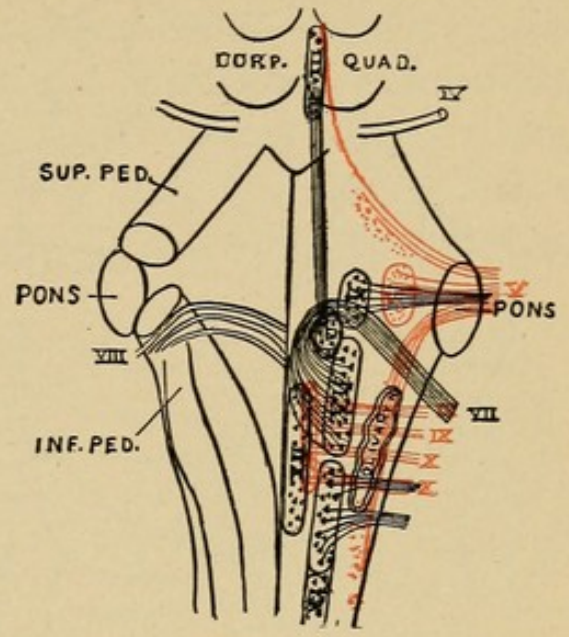


FIG. 19.

Diagram showing the nuclear origin of the cranial nerves (except the auditory). The sensory structures are in *red* and the motor in black. The floor of the 4th ventricle is seen from above, and on the right side the structures are represented as transparent. VII, the facial nucleus. The fibres from this nucleus wind round the nucleus of the abducens nerve (VI), and make their exit just below the pons IX and X, common nucleus of the glosso-pharyngeal and vagus nerves. The roots of the vagus receive filaments from the nucleus ambiguus. XI, spinal accessory nucleus. XII, hypoglossal nerve. V. the trigeminus nerve. Fibres pass to this nerve from above (descending or trophic root), from below (ascending sensory root), from the level of its exit from a sensory nucleus, and from a motor nucleus. P.L.B., posterior longitudinal bundle connecting the VIth nucleus below with the IIId nucleus above. VIII, the striæ acousticæ.

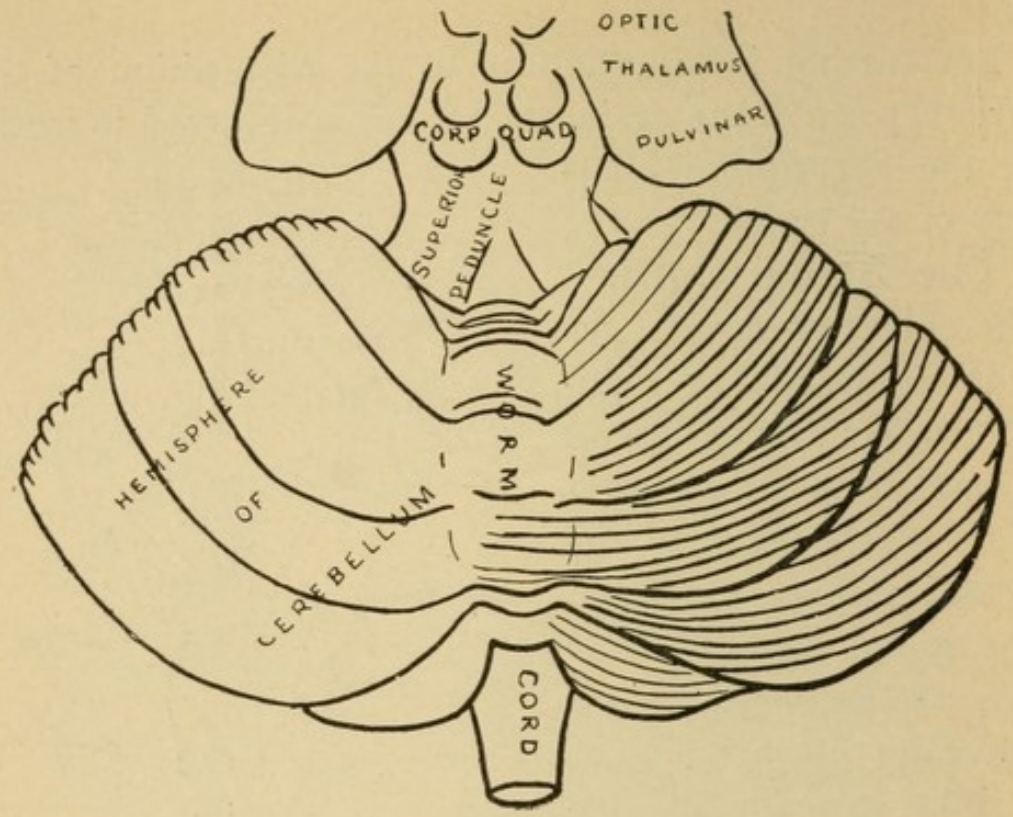


FIG. 20.

Diagram of dorsal surface of cerebellum, showing the position of the worm. (After Edinger.)

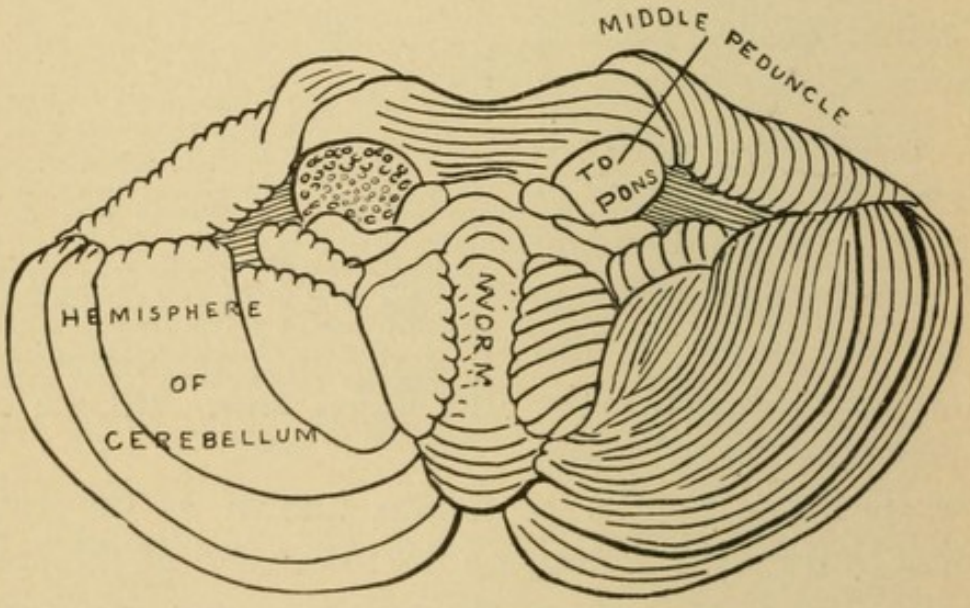


FIG. 21.

Diagram of ventral surface of cerebellum. (After Edinger.)

enlargement, due to the fact that it is the motor and sensory cord centre for the upper extremities; and the lower extremity of the cord also presents an intumescence known as the lumbar enlargement, containing the centres subserving motion and sensation in the lower extremities, together with the sexual, anal, and vesical centres. The term lumbar enlargement is a little misleading, and it must be remembered that though this enlargement gives rise to lumbar nerves, the greater portion of it is contained in the lowest part of the dorsal region of the spine.

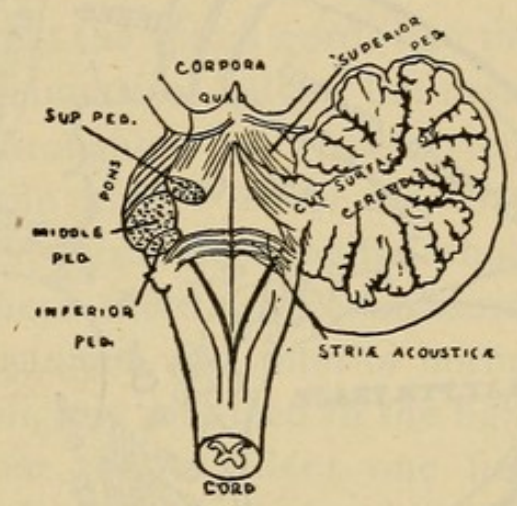


FIG. 22.

Diagram showing the three peduncles of the cerebellum—the superior peduncle going to the quadrigeminal region, the middle peduncle going to the pons, and the inferior peduncle coming from the cord below. (After Edinger.)

The spinal cord, taken as a whole, must be studied from two points of view; first, as a kind of cable through which nerve-impulses are transmitted to and fro between the peripheral portions of the body and the brain; secondly, as a series of segments superimposed like blocks one upon the other, each giving rise to a pair of spinal nerves, and each serving as a centre for various reflexes, for trophic influences, and for vascular control. The long fibres necessary for the first purpose are disposed in columns running the whole length of the cord and surrounding the central gray matter.

The H-shaped central gray matter consists of the anterior and posterior horns, which are connected with the anterior and posterior roots of the spinal nerves. The lateral and anterior columns contain the motor-fibres from the cortex, and the posterior columns certain

sensory tracts. The anterior columns are often called the columns of Türck; the posterior are composed of the columns of Goll and Burdach. Their exact positions are indicated in the accompanying diagram of a transverse section of the cord.

Reflex, trophic, and vaso-motor centres are situated

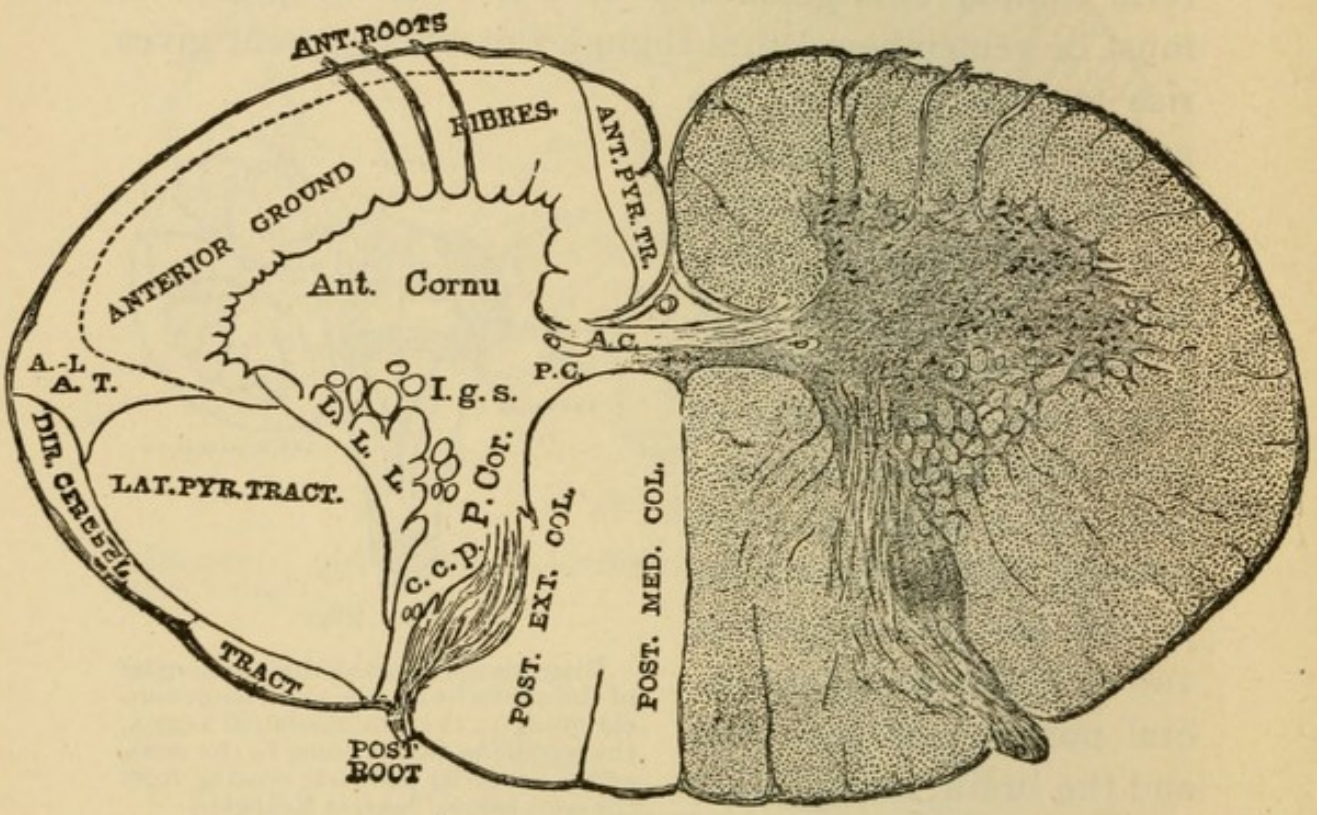


FIG. 23.

Diagram of a section of the spinal cord in the cervical region. A. C., anterior commissure; P. C., posterior commissure; I. g. s., intermediate gray substance; P. Cor., posterior cornu; c. c p., caput cornu posterioris; l. l. l., lateral limiting layer; a.-l. a. t., antero-lateral ascending tract, which extends along the periphery of the cord. (Gowers.)

in the gray matter. The various reflexes will be considered elsewhere. The trophic centres in the anterior horns regulate the nutrition of the motor nerves and muscles, and the destruction of them is followed by degeneration of the motor nerves and atrophy of the muscles innervated by them. The ganglia of the pos-

terior roots are trophic centres for most of the fibres of the sensory nerves, but a few of the posterior root-fibres have trophic centres in the posterior horns. There are trophic centres, also, for viscera, bones, joints, and for the skin and nails, all along the cord, and these are probably situated in the gray matter about the central canal. There are communicating nerve-filaments between them and the ganglia and fibres of the sympathetic nervous system.

It must be borne in mind that the spinal cord, like the brain, is composed of two symmetrical halves, but more closely bound together. Throughout its whole extent fibres pass from one side to the other. There are, first, the sensory nerve-fibres, which decussate along the whole of the cord, and besides these there are commissural fibres between the anterior columns and anterior horns.

The following is a table, slightly modified in the light of recent investigations from the excellent one first elaborated by Starr, which shows the relation between each segment of the spinal cord and the principal muscles or groups of muscles, sensory areas, and reflexes in connection with it.

Segment of cord	*Nerves*	*Muscles*	*Sensory areas*	*Reflexes*
Second and third cervical	Occipitalis major and minor Auricularis mag. Superficialis colli Supraclavicular	Sterno-mastoid Trapezius Scaleni and neck muscles *Diaphragm*	Neck and back of head	Hypochondrium (?)
Fourth cervical	Supraclavicular Circumflex Musculo-cutaneous Musculo-spiral	*Diaphragm* Supra- and infra-spinatus Deltoid Supinator longus Rhomboidei	Neck Superior surface of shoulder Outer surface of arm	Cilio-spinal (4th cervic'l to 2d dorsal)

Segment of cord	*Nerves*	*Muscles*	*Sensory areas*	*Reflexes*
Fifth cervical	Supraclavicular Circumflex External cutaneous Internal cutaneous Posterior spinal branches	Deltoid Biceps and coraco-brachialis Supinator longus and brevis Rhomboidei Deep muscles of shoulder-blade Pectoralis (clavicular part) Teres minor Serratus magnus Brachialis anticus	Back of shoulder and arm Outer side of arm and forearm	Scapular (5th cervic'l to 1st dorsal) Biceps jerk Supinator jerk
Sixth cervical	External and internal cutaneous Radial	Biceps Brachialis ant. Pectoralis (clavic. part) Subscapular Serratus mag. Triceps Extensors of wrist and fingers Pronators	Outer side and front of forearm. Back of hand (radial distribution)	Elbow jerk (triceps) Wrist jerk
Seventh cervical	External and internal cutaneous Radial Median Posterior spinal branches	Triceps (long head) Extensors of wrist and fingers Pronators Flexors of wrist Subscapular Pectoralis (costal part) Serratus mag. Latissimus dorsi Teres major	Radial and median distribution	Palmar (7th cervical to 1st dorsal)
Eighth cervical	Internal cutaneous Ulnar	Triceps (long head) Flexors of wrist and fingers Small muscles of hand	Inner side of arm and forearm Ulnar area of hand	
First dorsal	Int. cutaneous nerve of Wrisberg	Extensors of thumb Small muscles of hand Thenar and hypothenar muscles	Inner side of arm and forearm	

Segment of cord	*Nerves*	*Muscles*	*Sensory areas*	*Reflexes*
Second dorsal	Intercosto-humeral		Inner side of arm near axilla	
Second to twelfth dorsal	Intercostals and dorsal posterior nerves	Muscles of back and abdomen Erectores spinæ	Skin of back and upper gluteal region, and of breast and abdomen in bands running downward & forward	Epigastric (4th to 7th dorsal) Abdominal (7th to 11th dorsal)
First lumbar	Ilio-hypogastric Ilio-inguinal	Ilio-psoas Sartorius Rectus	Skin over groin and front of scrotum	Cremasteric (1st to 3d lumbar)
Second lumbar	Genito-crural External cutaneous	Ilio-psoas Sartorius Quadriceps fem'ris	Outer surface of thigh	
Third lumbar	Anterior Crural Internal cutaneous Long saphenous Obturator	Quadriceps fem'ris Anterior part of biceps Inner rotators of thigh Adductors of thigh	Anterior surface of thigh	Knee-jerk
Fourth lumbar	Internal cutaneous Long saphenous Obturator	Adductors of thigh Abductors of thigh Flexors of knee Tibialis anticus Peroneus longus	Inner side of thigh, leg, and foot	Gluteal (4th to 5th lumbar)
Fifth lumbar	External popliteal External saphenous [neous Musculo-cutaneous Plantar	Outward rotators of thigh Flexors of knee Flexors of ankle Extensors of toes Peronei	Outer and back side of leg and foot Sole of foot	Ankle clonus
First and second sacral	Same as 5th lumbar	Flexors and extensors of ankle Long flexor of toes Small foot muscles	Same as 5th lumbar	Plantar (5th lumbar to 2d sacral)
Third, fourth, and fifth sacral	Small sciatic Pudic Inferior hemorrhoidal Inferior pudendal	Perineal Muscles of bladder, rectum, and external genitals	Back of thigh, anus, perineum, genital organs	Vesical and anal centres
Fifth sacral & coccygeal	Coccygeal	Coccygeus	Skin about anus and coccyx	

In addition to the reflex centres indicated in the table, and which have great practical importance, there are others whose precise location has not yet been determined (such as the parturition centre and the erectile and ejaculatory centres), although we know they are in the lumbar enlargement.

The reflexes here given vary somewhat in importance. The knee jerk and the character of the plantar response are of greatest importance. The latter is known as the Babinski sign and will be further dealt with in the chapter on diagnosis.

It is always to be borne in mind that changes in the character of a given reflex mean primarily the involvement of the cord segment in which it is represented. For example, in spinal-cord tumor we shall have an absence of or a change in the character of all the reflexes, superficial and deep, up to the segment involved.

The sympathetic nervous system was originally so called because of its supposed relationship to emotional states. The name is obviously bad and should be dropped, more especially since this system subserves no such purpose. It is composed of the afferent and efferent nerve-fibres distributed to the various viscera and the vascular apparatus, the ultimate centres for which are within the cerebro-spinal axis. The so-called sympathetic system *per se* consists of a series of ganglia lying above, between the cranial and facial bones, and running down each side of the vertebral column from the skull to the coccyx, connected together by non-medullated nerve-fibres, sending branches to the smaller terminal ganglia of the tissues or viscera, and having communications with most of the cranial and spinal nerves, and through them with the central nervous system (see Fig. 24). Besides the pairs of lateral ganglia

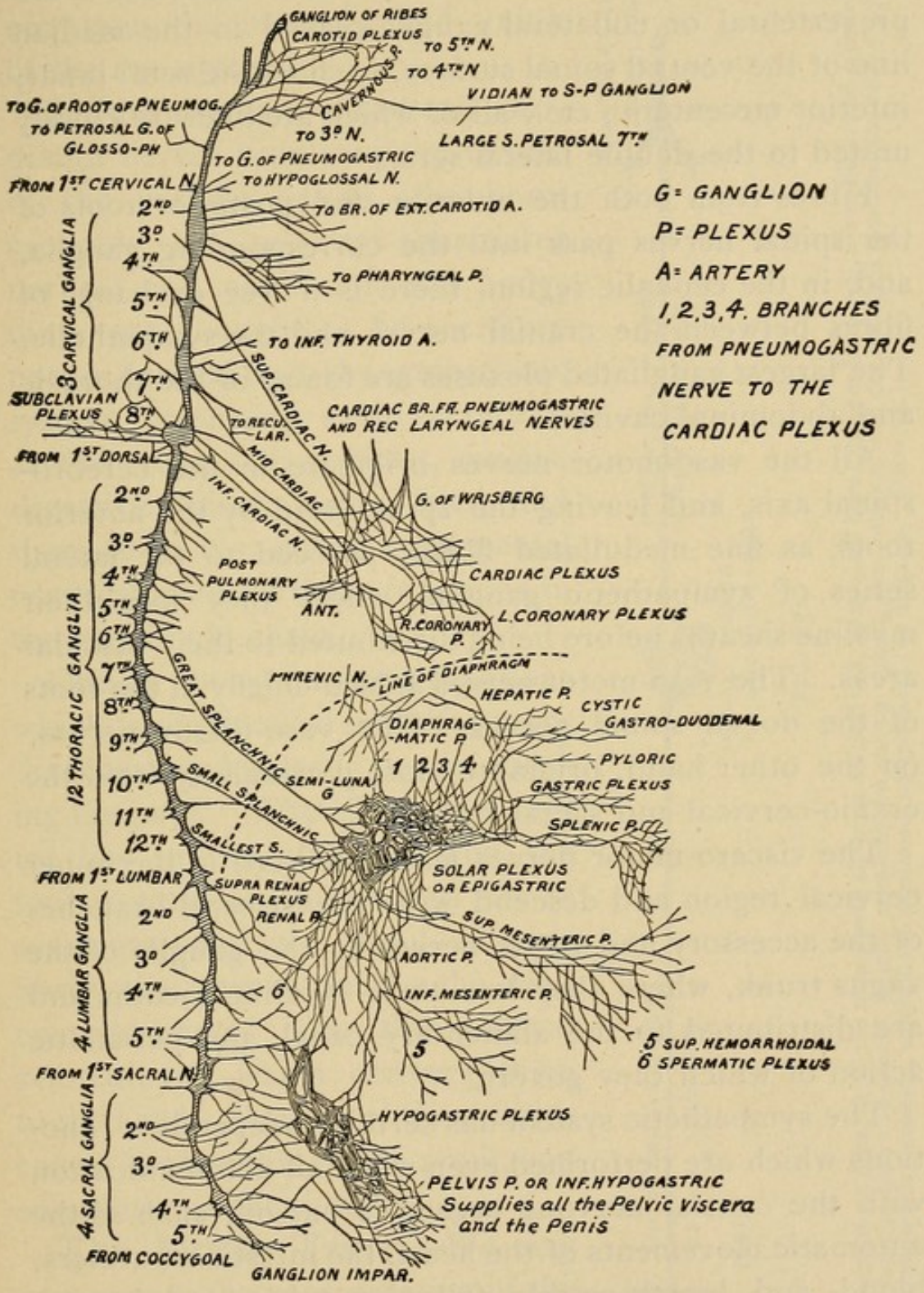


FIG. 24.

The sympathetic system of nerves.

lying upon the sides of the vertebral bodies, there are prevertebral or collateral ganglia placed in the median line of the ventral spinal surface, such as the semi-lunar, inferior mesenteric, etc., all of which are likewise closely united to the double lateral series.

Fibres from both the anterior and posterior roots of the spinal nerves pass into the corresponding ganglia, and, in the cephalic region, there is a free exchange of fibres between the cranial nerves and the sympathetic. The largest gangliated plexuses are found in the thoracic and abdominal cavities.

All the vaso-motor nerves originate in the cerebro-spinal axis, and leaving the spinal cord by the anterior roots, as fine medullated fibres, proceed to the lateral series of sympathetic ganglia, where they lose their myeline sheaths before being distributed to their vascular areas. The vaso-motor nerves appear chiefly in the roots of the dorsal spinal nerves. The vaso-dilator nerves, on the other hand, originate more particularly from the cranio-cervical and sacral regions.

The viscero-motor nerves leave the cord in the upper cervical region and descend with the visceral branches of the accessory and vagus nerves to the ganglia of the vagus trunk, where they lose their myeline sheaths and are distributed to the alimentary canal, the peristaltic action of which they govern.

The sympathetic system has certain independent functions which are performed even when all communication with the central nervous organs is cut off, such as the automatic movements of the heart, the intestines, ureters, blood- and lymph-vessels, fallopian tubes, and uterus; but these functions are more or less influenced in the way of inhibition and stimulation by fibres from the cerebro-spinal system. There are other functions, such

as the sensory function of the splanchnic nerve, which are dependent upon the integrity and union of the two systems.

Besides the vaso-motor and vaso-dilator nerves supplying the vessels of the various tissues (the brain probably has no vaso-constrictor nerves) and viscera of the body, there are thought to be secretory nerves derived from the sympathetic system, governing such secretions as the tears, saliva, sweat, mucus, digestive juices, urine, etc., and there is in a measure a segmental representation of these functions in the ganglionic chain.

One of the sympathetic functions which is of practical importance for diagnostic purposes is that of dilating the pupil. The cilio-spinal centre lies in the cord between the fourth cervical and second dorsal segments, and the pupil-dilating fibres pass thence out by the rami communicantes into the cervical sympathetic, which conveys them to the head. Section of these fibres causes contraction of the pupil, and stimulation dilatation.

The Motor Tract.—We may now pass to the consideration of the parts of the nervous system that are concerned in voluntary motion.

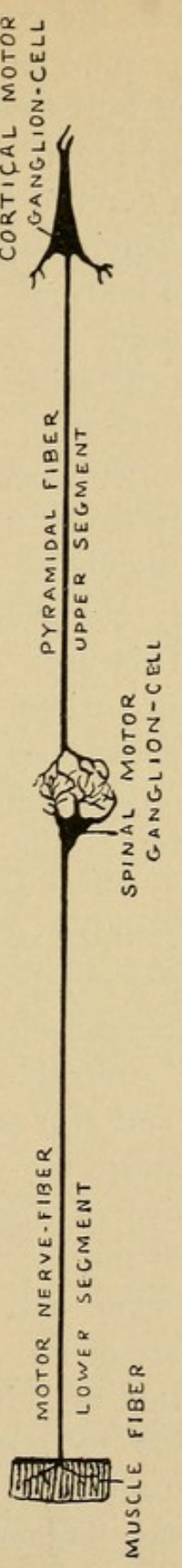


FIG. 25.

Diagram of an element of the motor path showing cortical cell, pyramidal fibre, anterior-horn cell, nerve-fibre, and muscle. More than one anterior-horn cell should, strictly speaking, be shown coming into relation with the terminal ramifications of one pyramidal fibre. To show this, however, would have needlessly complicated the diagram. (Gowers.)

Between the cortex of the brain of one side and the voluntary muscles of the opposite side of the body lies a system of fibres which we call the motor-tract. It is convenient to regard this tract as composed of two neurons: one, the cerebro-spinal; the other, the spino-muscular.

Each cerebro-spinal neuron consists of a cortical portion, the body of the neuron, and a spinal portion, its axis-cylinder process, which terminates by communicating with the spino-muscular neuron of the anterior horn of a spinal cord. The latter terminates by means of its axis-cylinder process in the muscle-fibre of the periphery.

Let us study the topography and relations of these neuron segments, beginning with the part of the cortex especially connected with voluntary motion, the motor area. This area comprises the ascending frontal and ascending parietal convolutions bounding the Rolandic fissure (sometimes called from their position the "central" convolutions), the superior parietal lobule, the paracentral lobule, and part of the precuneus (*vide* Figs. 7 and 8). The leg muscles are mainly represented in the upper third of the central convolutions and in the paracentral lobule.

The arm muscles are governed from the middle third of these central convolutions of the Rolandic region. The leg and arm areas probably overlap one another.

The centre for the movements of the face is in the lower third of the ascending frontal and possibly in a portion of the ascending parietal convolution.

The tongue and lips are represented together in the lowest part of the ascending frontal convolution and in the inferior or third frontal convolution, and it is not practicable to separate their regions, because of the habitual functional association of the orbicularis oris and transversus linguæ. The motor-area for speech occu-

pies about the same position as the centre for the tongue and lips; that is, the lower or third frontal convolution in its posterior part. These areas either immediately adjoin or overlap each other, but the motor-speech centre is usually located only in the left hemisphere.

While there can be no doubt as to the relation existing between the parts described and voluntary motion, it must be remembered that sensation, also, is to a certain extent localized in the same regions, the sensory fibres from the periphery terminating by communication with special neuron types, which ultimately carry the sensory impulse over to the motor system of neurons. The sensory areas and tracts will be discussed in a subsequent paragraph.

The Rolandic region of the cortex contains the largest neurons in the central nervous system, some of them being as much as 65 μ in diameter. They lie in what is known as the third of the five layers of the cortex, and their bodies have a pyramidal shape, with their apices toward the periphery and their bases directed inward. From the bases of these pyramids spring the axis-cylinder process, which continues uninterrupted into the spinal cord, where it communicates with the spinal-neuron segment of the anterior horn. The motor impulse is thus transmitted. As they descend toward the lower part of the brain these fibres converge to form in the internal capsule the large compact bundle of the motor tract.

The internal capsule is the name given to the knee-shaped mass of white substance seen upon making a horizontal section of the brain through the basal ganglia. It lies between the nucleus lenticularis on the outside and the thalamus and nucleus caudatus on the inside. The motor tract occupies but a portion of the internal capsule, viz., the anterior two thirds of the portion

behind its angle or knee lying between the optic thalamus and the nucleus lenticularis. Before reaching the internal capsule the fibres for the leg, arm, and face hold the same relative position in the centrum ovale that they

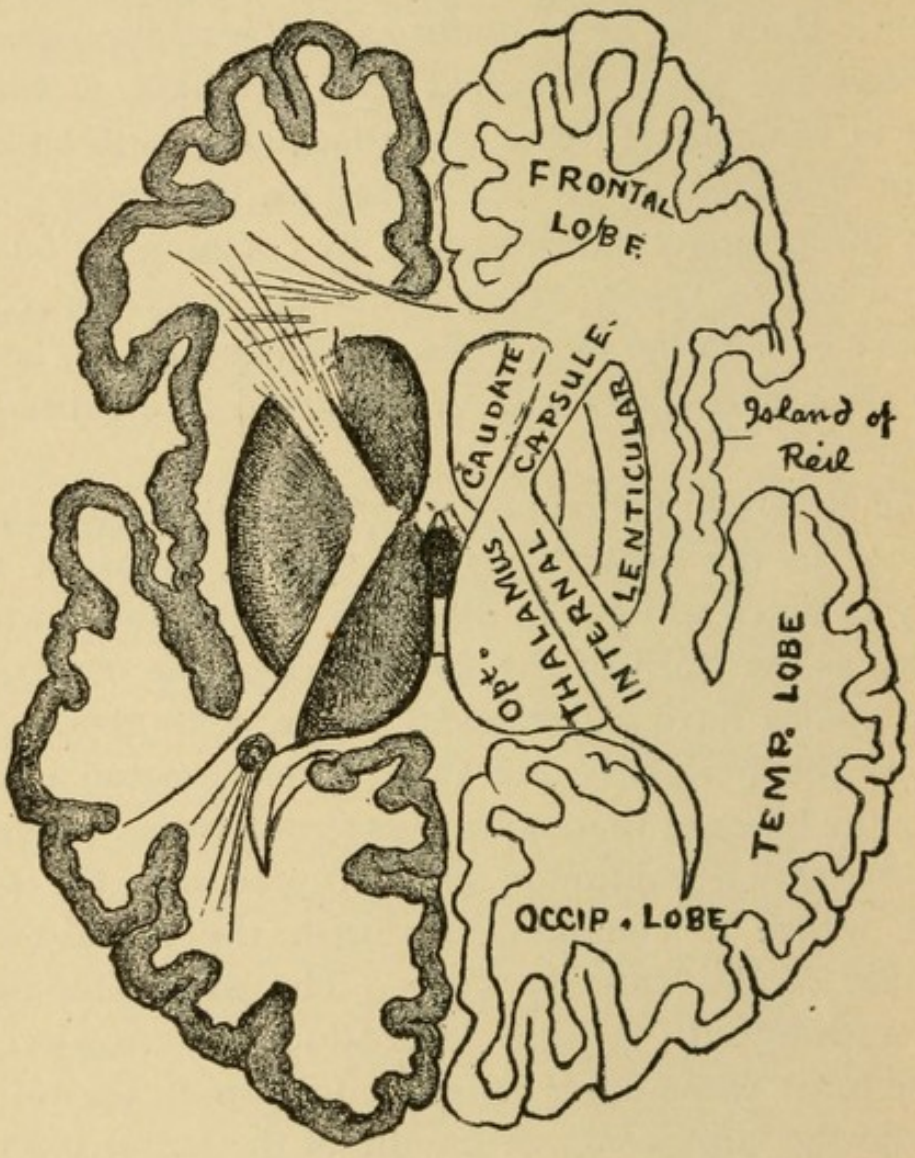


FIG. 26.

Horizontal section through the brain showing the position of the optic thalamus, the corpora striata (caudate and lenticular nuclei), and the internal capsule.

have upon the cortex—that is, from the median line outwards, leg, arm, face—L A F. As the bundles enter the internal capsule their relative positions change so that L A F is the formula to express their situation from

behind forwards. Their positions are altered again as they enter the crus cerebri, the formula L A F showing their relative location from without inwards, for the leg bundle is outermost, the arm bundle next in order, and the face bundle nearest the median line. Thus their situations with regard to the median line are completely reversed in their progress from the cortex to the pons. In the pons the same positions are maintained until the facial bundle crosses to its nucleus on the opposite side, and a little lower down the tongue-fibres cross to the hypoglossal nucleus. The leg and arm bundles continue down together to form the remainder of the pyramidal tract.

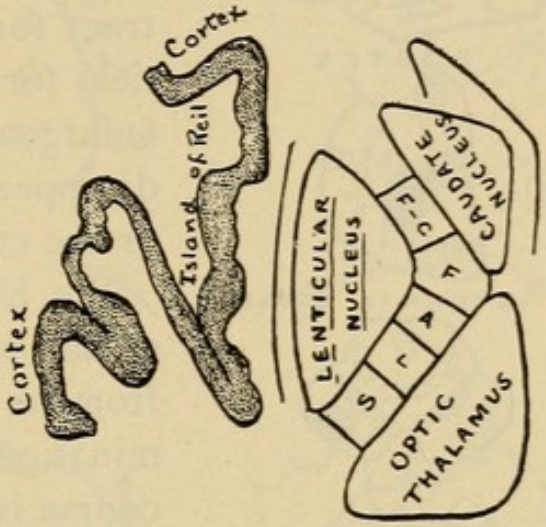


FIG. 27.

Diagram of horizontal section through the basal ganglia and internal capsule (left side), showing the position of the chief tracts in the internal capsule. The region of the capsule marked by the letters L A F is occupied by motor fibres: L corresponds to the leg fibres, A to the arm fibres, F to the face fibres (including fibres to face muscles, and tongue). The region F–C contains the fronto-cerebellar tract (intellectual tract). The region marked S contains the general sensory tract from the opposite side and the fibres from the optic and olfactory nerves of the opposite side, sometimes called the "sensory crossway."

The motor tracts occupy the anterior part of the pons and constitute the anterior pyramids of the medulla. Just as they pass from the medulla to the cord most of the fibres cross to the opposite side. The number of fibres which decussate to enter the opposite lateral column of the spinal cord is variously estimated by different authors at from seventy-five to ninety-seven per cent. It varies considerably in different individuals. The remainder of the fibres of the motor tract continue down into the anterior column of the same

side, crossing to the opposite anterior horns at various levels. The former bundle is designated the crossed pyramidal tract, and the latter the direct pyramidal tract.

There is good reason for believing that the crossed pyramidal tract is destined chiefly for the innervation of the trunk and lower extremities, and the direct pyramidal tract for that of the upper extremities; for one continues to the lumbar enlargement, and the other generally disappears in the mid-dorsal region or the cervical enlargement.

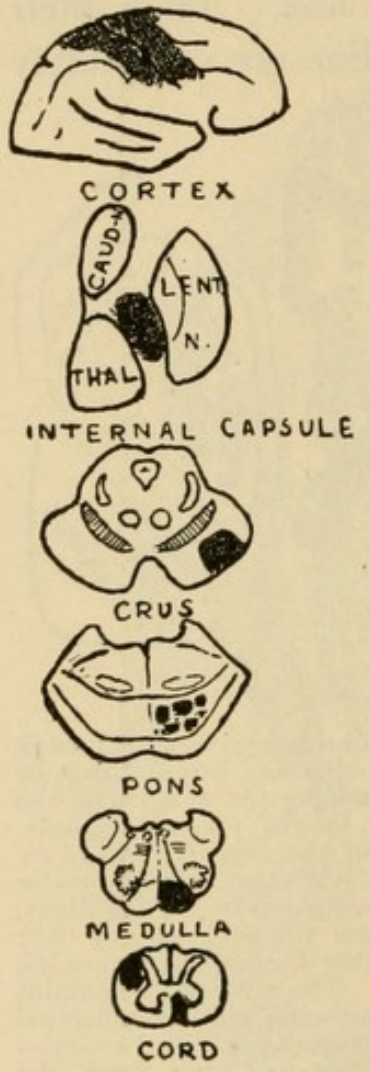


FIG. 28.

Diagram showing the position of the pyramidal tract of the right side at different levels of the brain and in the spinal cord. (After Gowers.)

We have now traced the cerebro-spinal segment of the motor tract from its origin in the cortex to its termination in the anterior horns. Its course is uninterrupted throughout.

Passing to the consideration of the spino-muscular portion of the motor tract we have to examine the disposition of the bodies of the neurons in the anterior horn. These are distributed in groups or clusters in the anterior horns, and their arrangement differs in the various levels of the spinal cord. We distinguish by their relative position in transverse sections at certain levels the following five groups: a median, an anterior, an antero-lateral, a postero-lateral, and a central. (Fig. 29).

Often the second and third of these are not distinguishable from one another by any well-marked line of separation.

The occurrence of the neurons in clusters is most marked in the cervical and lumbar enlargements. In the dorsal region, where the anterior horns are small, the distinction of isolated groups can usually be made out.

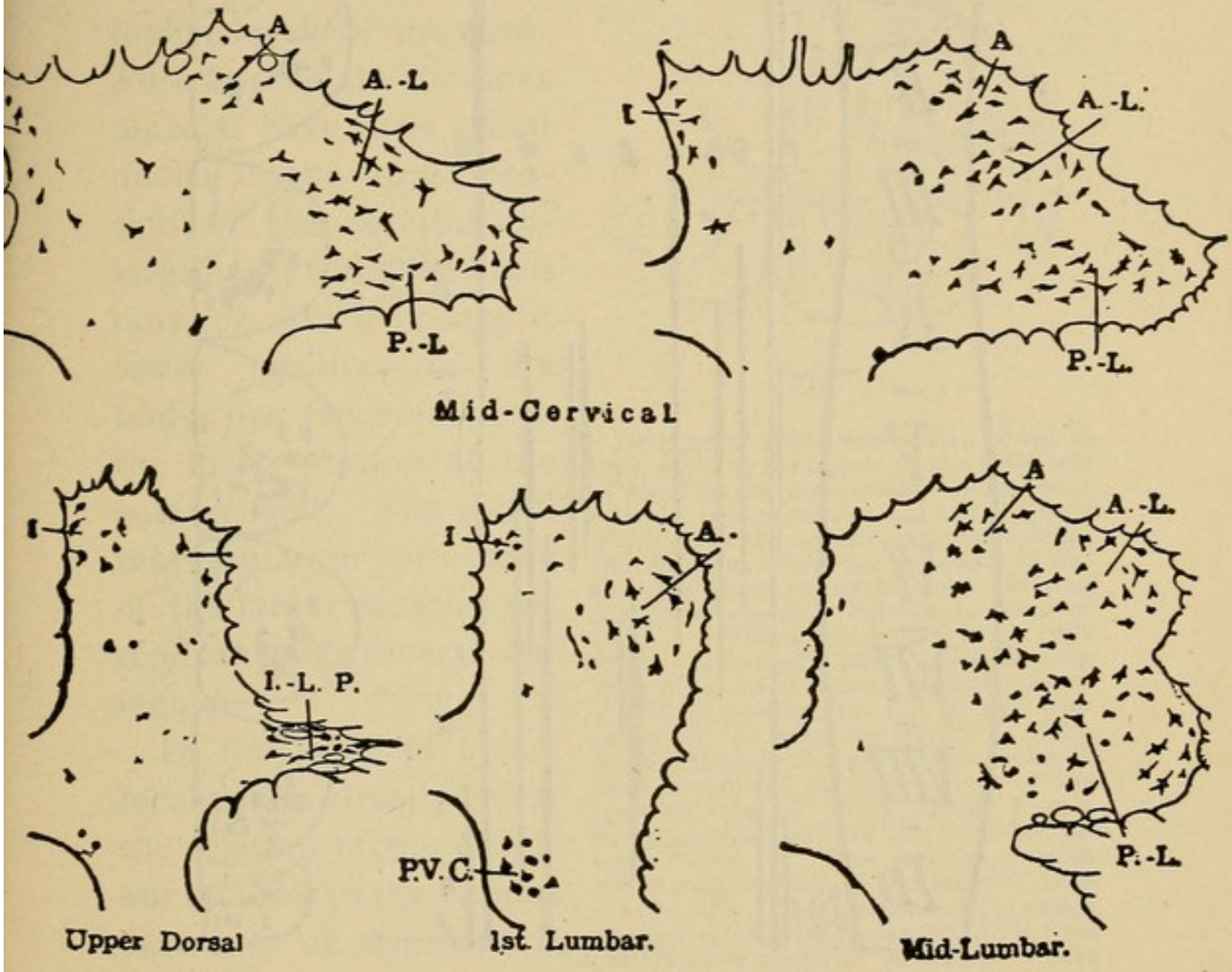


FIG. 29.

Diagrams of the groups of nerve-cells in the anterior cornu. Groups: I, inner or medial; A, anterior; A.-L., antero-lateral; P.-L., postero-lateral; I. L. P., intermediate lateral process; P. V. C., posterior vesicular column or tract. The two mid-cervical sections are only a few millimetres apart, and show how the anterior group, separate in the one, may be blended with the antero-lateral group in a neighboring part of the cord. (Gowers.)

A knowledge of this grouping is not of much importance in diagnosis, for we have not yet learned of the exact relationship existing between particular neuron aggregations and the muscles which they govern. We know

much more of the relations of the masses of neurons taken as a whole in the anterior horns of particular

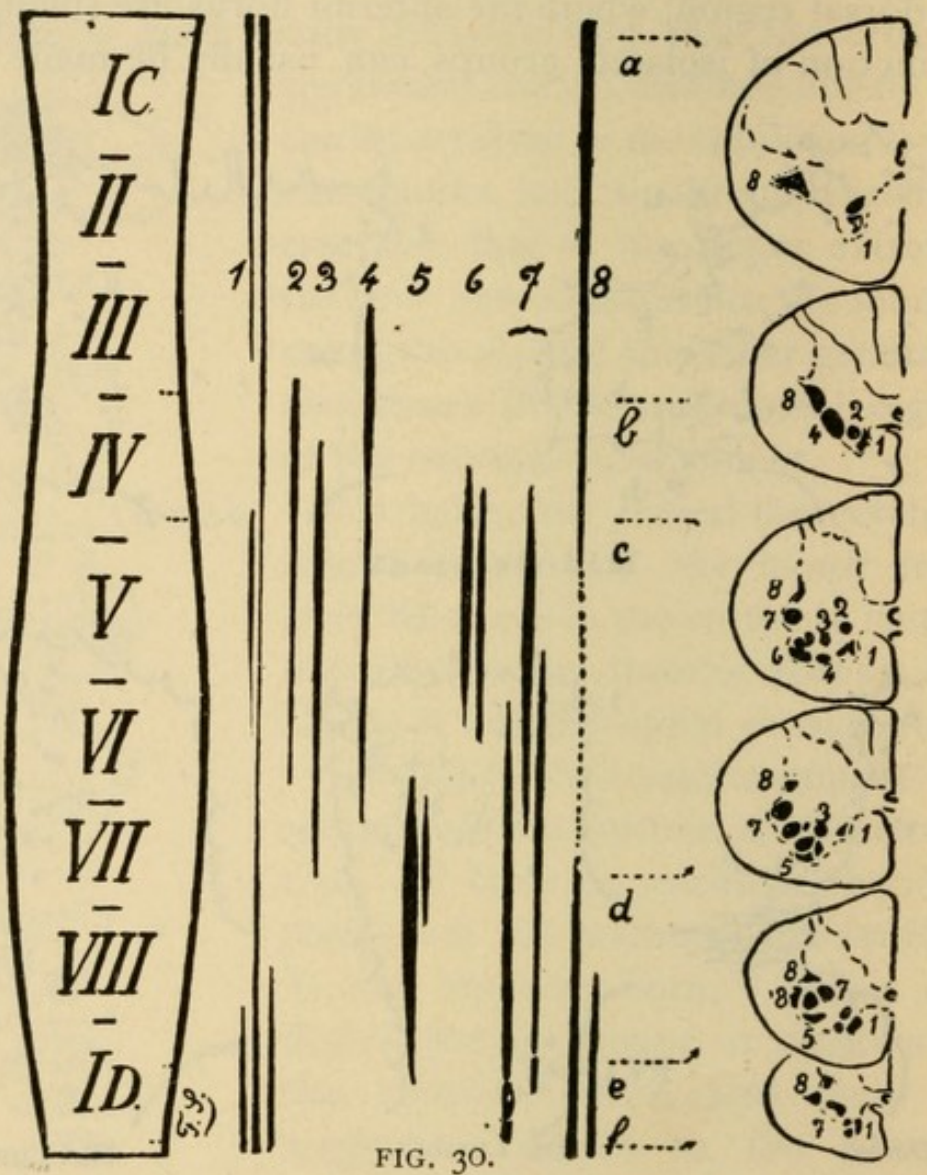


FIG. 30.

Columns of gray matter and motor nuclei of the cervical enlargement (After F. Sano). Columna medialis—1, *a*, short rotators of head; M. subhyoid muscles; *b*, *c*, *d*, *e*, *f*, extensors and rotators of the vertebral column. 2, nucleus diaphragmatis (the series of sympathetic nuclei composed of small cells have not been drawn in), they are situated behind the column medialis near the columna canalis centralis. Columna intermedio-lateralis—8, *a*, accessorius M. trapezius and M. sterno-cleido mastoideus; *b*, *c*, plexus cervicalis; Mm. trapezius sterno-cleido-mastoideus; *d*, *e*, middle portion of M. trapezius; *f*, inferior part of M. trapezius; *e*, beginning of the nucleus for the M. latissimus dorsi. Columna extremitatis superioris—3, Mm. pectorales; 4, *b*, M. levator scapulæ; *c*, M. serratus major; 6, muscles of the shoulder; 7, *c*, M. biceps; lower down, supinators and extensors of the fingers; between *d* and *e*, flexors and pronators; *e*, thenar and hypothenar muscles; *f*, hypothenar muscles; 5, *d*, M. triceps brachii; *e*, M. anconæus. (Barker.)

segments of the cord, and the muscles with which they are connected. There are probably, also, frequent

though inconsiderable variations in individuals, as regards the extent and complexity of muscle representation at different levels of the cord. It should be noted that muscle-groups which have a higher position in the body are localized, as a rule, in higher levels of the cord. For example, the shoulder muscles have their spinal nuclei in the upper portion of the cervical enlargement, while the hand muscles, which occupy a lower position in the body, are represented in the lower portion of the enlargement. The spinal representation of many of the larger muscles extends through several segments.

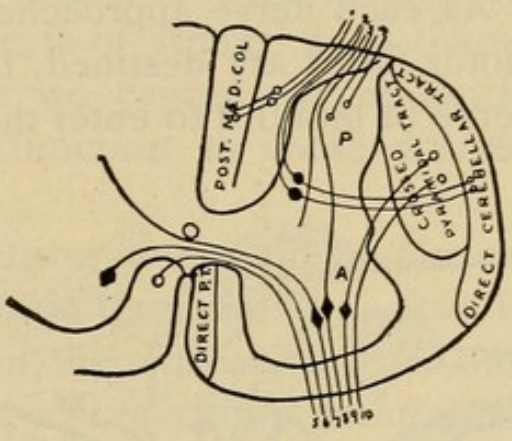


FIG. 31.

Diagram of transverse section of spinal cord, showing the course of the chief sets of fibres in the nerve-roots. (A. anterior horn; P. posterior horn.) 1 and 2. Fibres which enter the postero-external column (Burdach) and take an ascending or descending course, many of them entering the postero-median column (column of Goll). 3. Fibres which pass to the cells of the column of Clarke, which in turn send fibres to the direct cerebellar tract. 4. Fibres passing to the proper substance of the posterior horn. 5. Fibre coming through the anterior commissure from the direct pyramidal tract of the opposite side. 6. Fibre coming from a motor ganglion-cell of the opposite anterior horn. 7. Fibre connected with motor ganglion-cell of the anterior horn of same side. This ganglion-ceil is probably connected with a sensory fibre from the opposite posterior horn. 8. Fibres from motor ganglion-cell of anterior horn of same side. This ganglion-cell is probably connected with a sensory fibre from the posterior horn of the same side. 9 and 10. Fibres from the crossed pyramidal tract of the same side.

In the table of spinal localization already given, the relation of the different muscles to the various segments of the cord is presented.

The neurons of the anterior horn are of large size, the axis-cylinder processes of which collect in bundles and pass through the white substance to emerge as the anterior roots of spinal nerves from the cord. When joined by the posterior or sensory root at the intervertebral foramen it contributes to form a mixed nerve—that is, one having

both motor and sensory fibres. There is no way of distinguishing the motor from the sensory fibres, since they have no characteristic arrangement in the spinal nerves and no known structural differences.

As each nerve approaches the muscles for which its motor fibres are destined, these latter leave the mixed nerve in bundles to enter the muscles and be distributed

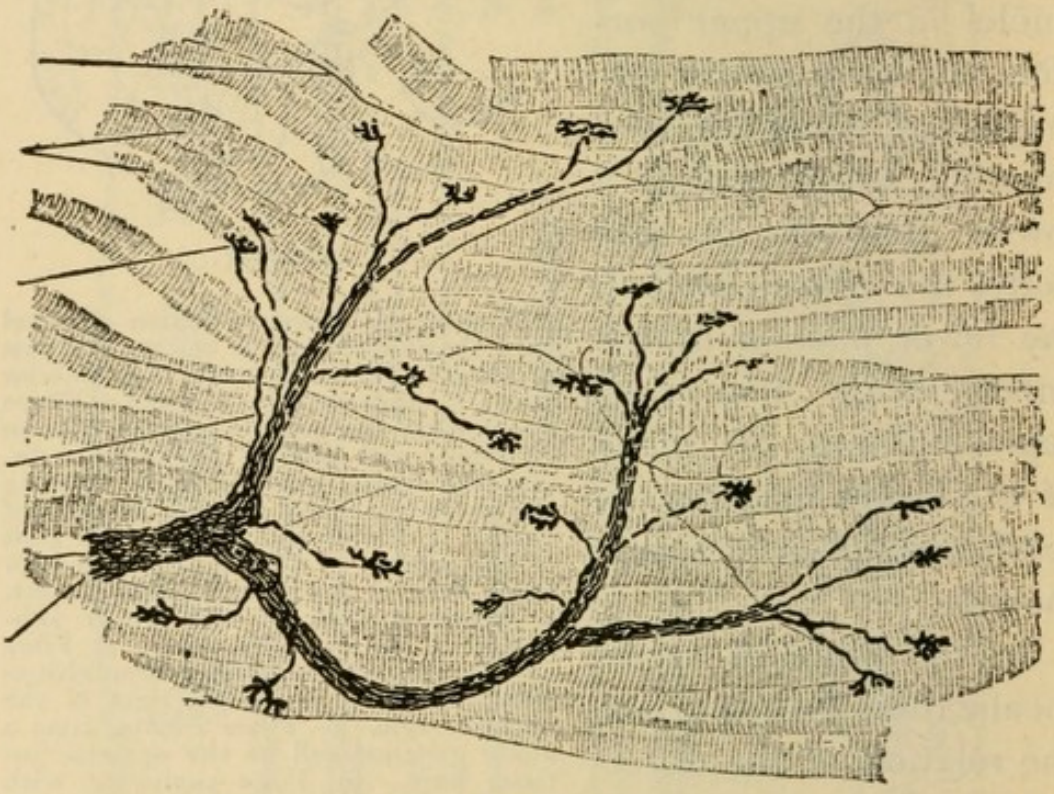


FIG. 32.

Motor nerve-ending of intercostal muscle-fibres of a rabbit. (Stohr.)

to their fibrillæ. Each nerve fibril terminates in a muscular fibre as a motorial end-plate.

The Sensory Tract.—We know far less of the nervous structures that subserve ordinary sensibility than of those which underlie voluntary motion, and the difficulties of tracing the sensory paths are increased by the fact that, in certain parts at least, different forms of sensation are conducted by different structures. The

skin is the seat of a variety of bodies related to sensory perception. Of these some, like corpuscles of Pacini and Meissner, and the end-bulbs of Krause, are differentiated tactile organs whose office it is to modify and possibly to multiply the effects of stimuli upon the terminal nerve filaments, but we cannot as yet ascribe specific forms of sensory function to these different bodies.

The simpler nervous structures in the skin are plexuses of non-medullated nerve-fibres terminating in free extremities between the cells of the mucosa, and are more widely distributed than the end-organs just mentioned.

There is some reason for believing that these various terminal nerve-structures respond only to particular kinds of sensory stimuli.

Sensory fibres also enter the muscles, and the impressions conveyed by them upon contraction of the muscle-fibres have much to do with the sense of posture and the nice control and proper co-ordination of muscular movements.

There are good reasons for believing that the various sensations of touch, pain, temperature, and the muscular sense, have special terminal filaments for their apprehension, and possibly for their conveyance from the periphery to the central nervous system.

In the intervertebral foramen all of the sensory fibres separate from the motor bundle to pass into the spinal cord as the posterior root.

The fibres composing a sensory nerve are to be looked upon as the dendrites of the neuron, the body of which is in the ganglion of the sensory root with its axis-cylinder process in the spinal cord. The first neuron in the sensory system is, therefore, composed of a single dendrite which extends into the periphery and

appreciates the sensation, a body which is located in the posterior-root ganglion and an axis-cylinder process which transmits the sensation for a longer or shorter distance in the spinal cord. This anatomical arrangement applies to all sensory nerves. The destination of the various axis-cylinder processes entering the cord varies considerably. The following are generally distinguished (see Fig. 33): A. Fibres ending by communication with the various types of cells found in the posterior horn. B. Fibres that enter the posterior columns and terminate by communication with the cells of the nucleus gracilis and nucleus cuneatus. C. Fibres which pass to the anterior-horn cells of the same side constituting the direct reflex path and fibres which pass to the opposite side forming a bundle which transmits the sensations of temperature and pain.

Among the first group of fibres we recognize, besides those terminating in the cells composing the gelatinous substance, others which terminate by communication with neurons situated at the base of the posterior horn. These bodies constitute Clarke's vesicular column and are found especially well marked in the dorsal and upper lumbar regions of the spinal cord. They exist less pronounced in the cervical and lower lumbar regions, where they are known as Stilling's column. These groups of cells are of great importance because of their relationship to the direct cerebellar tract which lies just to the outer side of the crossed pyramidal tract. A further tract to the cerebellum is derived from neurons lying at the base and at the junction of the anterior and posterior horns (Gowers' bundle).

The second (B) group of fibres mentioned above do not terminate till they reach the nucleus gracilis and nucleus cuneatus of the medulla.

From these nuclei the sensory system is continued to the brain cortex, on the one hand, by the formation of the lemniscus, which is made up of crossed fibres from these nuclei, and to the cerebellum, on the other, by fibres passing by way of the restiform body to the cortex of the vermis. Some of these fibres pass direct, without interruption in these nuclei.

Among the third (C) group of fibres are those which pass to the antero-lateral cord region of the opposite side and which transmit pain and temperature sensations. These fibres form a distinct bundle in that region known as the fasciculus-spino-tectalis et thalamicum (Horsley) fibres, which ultimately find their way into the lemniscus and end in the thalamus opticus.

We see that the path and distribution of a sensory nerve is extremely complex; fibres being distributed to and ultimate terminations being found in the posterior horns of the same and the opposite side and the anterior horns of the same side in the cord; fibres to the cerebellum which are interrupted in the cord (Clarke's column) and fibres to the cerebellum which are interrupted in the medulla (nuclei gracilis and cuneatus), fibres which reach the basal ganglia, and other fibres which are ultimately distributed to the cerebral cortex. All sensory nerves are thus complex and in addition contain also elements belonging to the so-called sympathetic system. Like the motor tract the decussation of the sensory tract is an incomplete one. Much of this decussation takes place in the spinal cord, but rather more occurs in the medulla.

As regards the position of the sensory tract in the medulla, pons, and brain only general statements can be made.

In the upper portion of the medulla the cerebellar

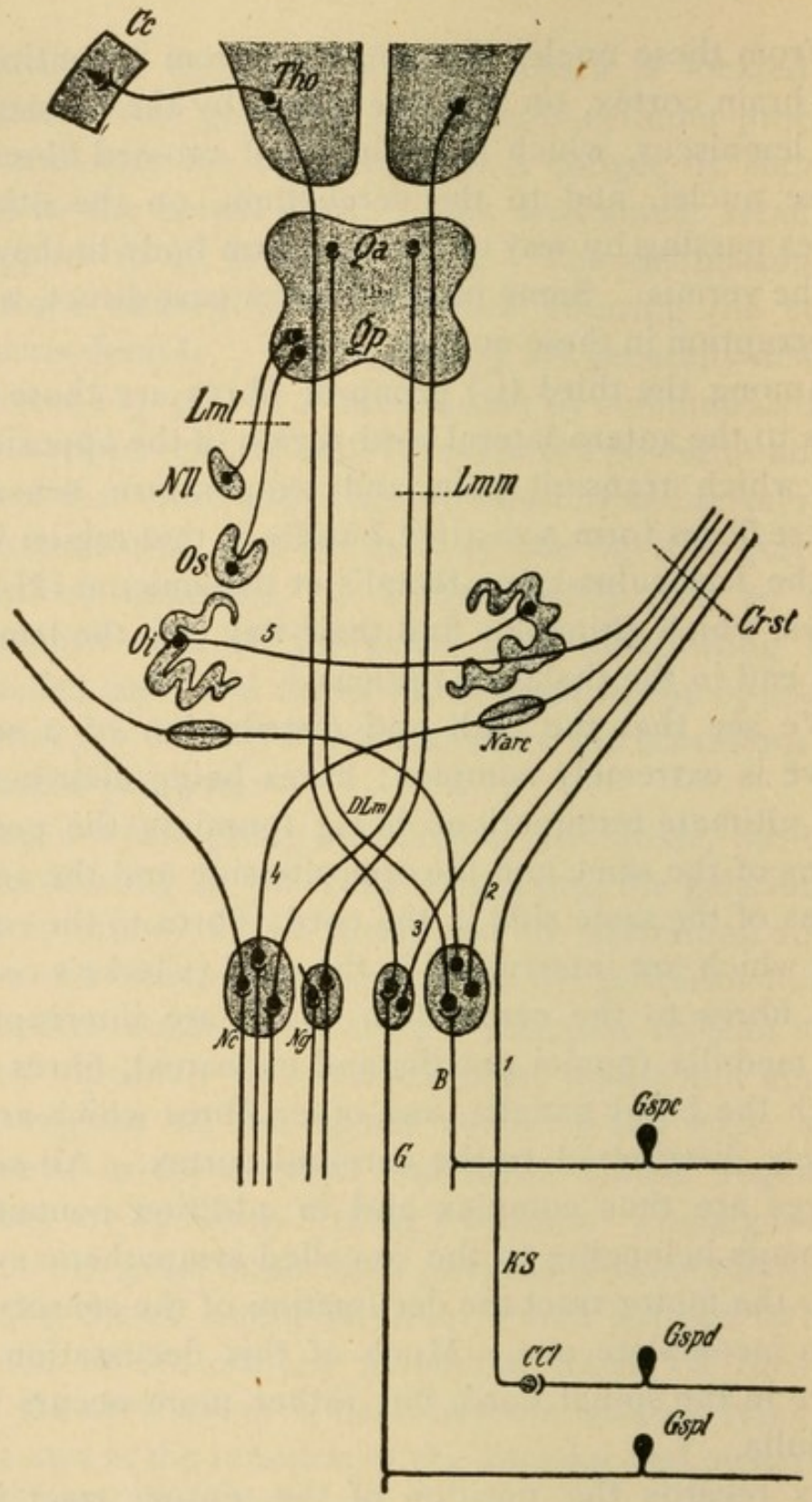


FIG. 33.

Schematic diagram of the more important tracts of the posterior columns of the cord. Gsp, spinal ganglion (l, lumbar, d, dorsal, c, cervical); CCl, Clarke's column; Ks, cerebellar tract; G, Goll's column; B, Burdach's column; Ng, nucleus gracilis; Nc, nucleus cuneatus; DLm, decussation of the lemniscus; Crst, restiform body; Narc, nucleus arcuatus; Oi, inferior olive; Os, superior olive; Lmm, medial lemniscus; Lml, lateral lemniscus; Nll, nucleus of the lateral lemniscus; Qp, Qa, anterior and posterior corpora quadrigemina; Tho, optic thalamus; Cc, cerebral cortex. (Obersteiner.)

fibres have already passed to their destination, and the crossed sensory fibres which here constitute the lemniscus or fillet are only to be considered. They end in nuclei belonging to the optic thalamus and ultimately reach the cortex in all probability through various groups of fibres known as the inferior, anterior, posterior, and external radiators of the thalamus. Another group of sensory fibres occupies the posterior third of the posterior limb of the internal capsule, a position immediately behind and contiguous to the motor tract. The cortical termination is probably in the ascending frontal and ascending parietal convolutions and contiguous areas of the parietal lobe.

From what has gone before it will be seen that certain of the sensory fibres cross to the opposite side in the cord (temperature and pain), while certain others (tactile and muscle sense) remain uncrossed till they reach the medulla. From this circumstance is made possible the symptom-complex of Brown-Séquard's paralysis.

It is probable that the fibres making up the posterior columns transmit certain muscular impulses, as well as tactile sense and muscle pain sense.

Reflex Paths.—A reflex movement of the simplest kind may be imperfectly defined as one that results from the conversion or "reflexion" of a sensory stimulus into a motor excitation. A sensory or afferent impulse is conveyed centrally by a sensory neuron to a motor neuron, whence it is reflected outward along a motor nerve to a muscle or group of muscles.

Thus the integrity of two neurons is necessary in order to make possible reflex action, a sensory neuron on the one hand and a motor neuron on the other, and such a combination constitutes a "reflex arc." The motor element in this "arc" is known as the "reflex centre," and these are the same neurons as those constituting the

final link in the cerebro-spinal motor system, under which circumstances they act as transmitters of voluntary motor impulses descending from the cortex.

The upper neuron elements in the motor system do more than conduct voluntary impulses from the brain to the lower neuron group, in that they exercise a restraining or inhibitory influence upon the elements of the reflex arc, more particularly its motor element. If, therefore, the upper neuron is injured or destroyed, as occurs, for example, in lesions of the internal capsule or disease of the motor cortex or the crossed pyramidal tracts, this inhibitory influence is removed, the activity of the reflex centre is uncontrolled, and the motor phenomenon becomes exaggerated. This applies particularly to the deep tendon reflexes.

The gray substance of the spinal cord contains a series of reflex centres, cutaneous, muscular, and visceral. Some are complex and of considerable vertical extent. The relations of the more important reflexes to particular segments are given in the table of localization of the functions of the spinal cord. The technique of their examination will be dwelt upon in another chapter.

The Spinal Nerves.—There are thirty-two pairs of spinal nerves. They arise at nearly equal intervals between the conus medullaris and the medulla. Each nerve is formed by the junction of an anterior (motor) and a posterior (sensory) root, and, leaving the spinal canal through the intervertebral foramen as a mixed nerve, soon divides into two branches, an anterior and a posterior. These branches either join with others in the formation of plexuses from which arise new nerve-trunks, or pass directly to their cutaneous, muscular, or visceral distribution. The nerve-roots differ in size at various levels of the cord, those of the dorsal region, for instance, being smaller than those of the lower cervi-

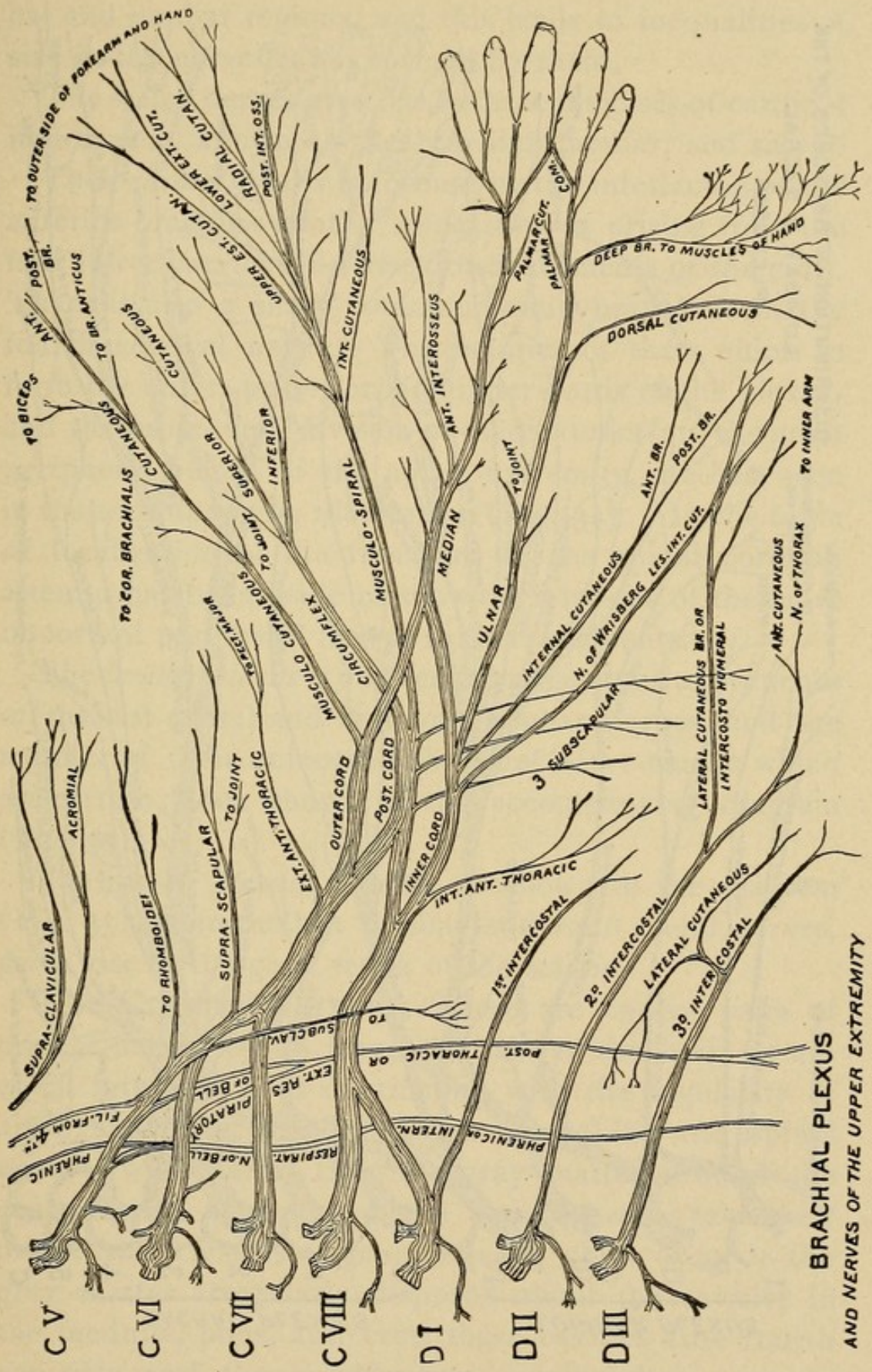

BRACHIAL PLEXUS
AND NERVES OF THE UPPER EXTREMITY
C V
C VI
C VII
C VIII
D I
D II
D III
SUPRA-CLAVICULAR
ACROMIAL
TO RHOMBOIDEI
SUPRA-SCAPULAR
TO JOINT
EXT. ANT. THORACIC
OUTER CORD
POST. CORD
INNER CORD
INT. ANT. THORACIC
1ST INTERCOSTAL
2D. INTERCOSTAL
3D. INTERCOSTAL
LATERAL CUTANEOUS
INTERCOSTO HUMERAL
LATERAL CUTANEOUS BR. OR
ANT. CUTANEOUS
N. OF THORAX
TO INNER ARM
3 SUBSCAPULAR
INTERNAL CUTANEOUS
N. OF WRISBERG
LES. INT. CUT.
ANT. BR.
POST. BR.
ULNAR
TO JOINT
MEDIAN
ANT. INTEROSSEUS
PALMAR CUT.
PALMAR COM.
DEEP BR. TO MUSCLES OF HAND
DORSAL CUTANEOUS
MUSCULO-SPIRAL
CIRCUMFLEX
MUSCULO CUTANEOUS
TO JOINT
SUPERIOR
INFERIOR
INT. CUTANEOUS
UPPER EXT. CUTAN.
LOWER EXT. CUT.
RADIAL CUTAN.
POST. INT. OSS.
TO OUTER SIDE OF FOREARM AND HAND
ANT. POST. BR.
TO BR. ANTICUS
TO BICEPS
TO COR. BRACHIALIS
CUTANEOUS
CUTANEOUS
PHRENIC
FIL. FROM 4TH
RESPIRAT. N. OF BELL
EXT. RES. OF BELL
TO SUBCLAV.
POST. THORACIC OR
PHRENIC OR INTERN.

FIG. 34.

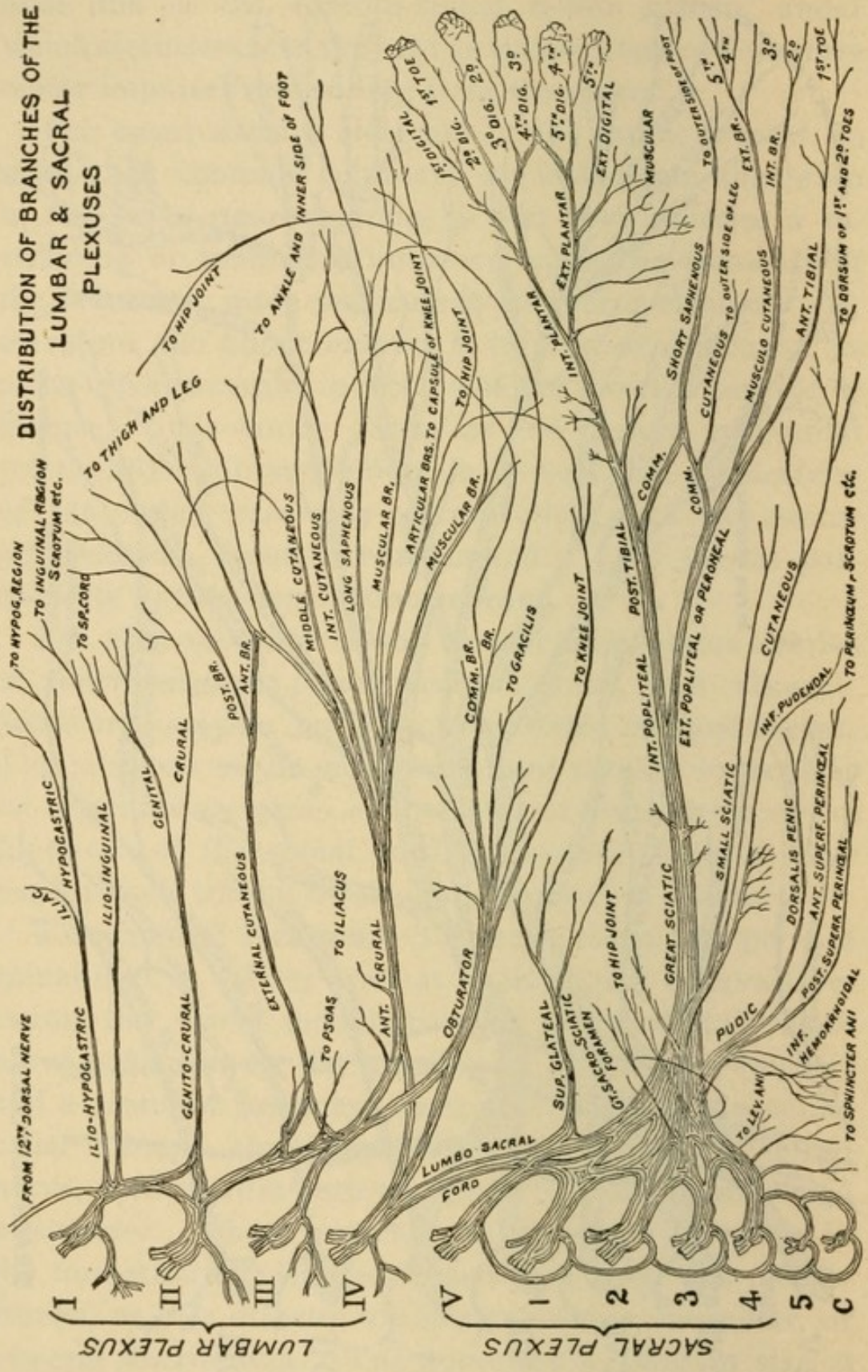

DISTRIBUTION OF BRANCHES OF THE LUMBAR & SACRAL PLEXUSES
FROM 12TH DORSAL NERVE
ILIO-HYPOGASTRIC
ILIO-INGUINAL
HYPOGASTRIC
TO HYPOG. REGION
TO INGUINAL REGION SCROTUM etc.
TO SP. CORD
GENITO-CRURAL
GENITAL
CRURAL
POST. BR.
ANT. BR.
EXTERNAL CUTANEOUS
TO THIGH AND LEG
TO HIP JOINT
TO ANKLE AND INNER SIDE OF FOOT
MIDDLE CUTANEOUS
INT. CUTANEOUS
LONG SAPHENOUS
MUSCULAR BR.
TO PSOAS
TO ILIACUS
CRURAL
ANT.
OBTURATOR
ARTICULAR BRS. TO CAPSULE OF KNEE JOINT
MUSCULAR BR.
TO HIP JOINT
COMM. BR.
TO GRACILIS
TO KNEE JOINT
LUMBO SACRAL CORD
SUP. GLUTEAL
GT. SACRO SCIATIC FORAMEN
TO HIP JOINT
GREAT SCIATIC
INT. POPLITEAL
POST. TIBIAL
COMM.
EXT. POPLITEAL OR PERONEAL
SMALL SCIATIC
PUDIC
INF. HEMORRHOIDAL
TO LEV. ANI
TO SPHINCTER ANI
1ST DIGITAL 1ST TOE
2D DIG. 2D
3D DIG. 3D
4TH DIG. 4TH
5TH DIG. 5TH
EXT DIGITAL
INT. PLANTAR
EXT. PLANTAR
MUSCULAR
SHORT SAPHENOUS
TO OUTER SIDE OF FOOT
CUTANEOUS TO OUTER SIDE OF LEG
MUSCULO CUTANEOUS
EXT. BR.
INT. BR.
ANT. TIBIAL
1ST TOE
2D
3D
TO DORSUM OF 1ST AND 2D TOES
CUTANEOUS
INF. PUDENDAL
TO PERINÆUM - SCROTUM etc.
DORSALIS PENIC
ANT. SUPERF. PERINEAL
POST. SUPERF. PERINEAL
I
II
III
IV
V
1
2
3
4
5
C
LUMBAR PLEXUS
SACRAL PLEXUS

FIG. 35.

cal and lumbar regions, and this leads to inequalities of size in the nerve-trunks formed by them.

The spinal nerves give rise to three plexuses of cardinal importance, known as the brachial, lumbar, and sacral.

The *brachial plexus* is formed by the interlacing of the anterior branches of the spinal nerves arising from the four lower cervical and first dorsal segments of the cord. There is also a small communicating branch from the fourth cervical nerve. The manner of their union to form the outer, posterior, and inner cords of the plexus, and the subsequent division of these trunks into the great peripheral nerves of the upper extremity, are best seen in the accompanying illustration (Fig. 34). In the table of localization of the functions of the spinal cord an attempt has been made to show the relations of the more important peripheral nerves to their segments.

The *lumbar plexus* is derived from the anterior branches of the last dorsal and first four lumbar nerves, and the manner of their union and the chief nerve-trunks which arise from it are shown in the accompanying diagram (Fig. 35).

The *sacral plexus*, formed, as shown in the diagram (Fig. 35), from the last lumbar and four sacral nerves, gives rise to the great trunk of the sciatic.

The Cranial Nerves.—There are twelve pairs of cranial nerves which leave the base of the brain at unequal intervals, thus contrasting with the regularity of arrangement of the spinal nerves. Like the spinal nerves, which arise from the gray matter around the central canal of the cord, the cranial nerves take their origin from clusters of ganglion-cells which occur in the gray matter irregularly disposed about the cavities in the medulla, pons, and crus, these cavities (the fourth ventricle and the aqueduct of Sylvius) being merely continuations upward of the central canal of the cord.

Leaving their nuclei of origin, the fibres constituting the various cranial nerves pass, by more or less direct paths, to the base of the brain, to emerge at points known as their superficial origins. From here to their foramina of exit the nerve-trunks are of different lengths and their relative disposition is such that pathological processes at the base of the brain produce very different and valuable localizing symptoms. Indeed, a clear picture of the position of the nuclei and of the arrangement of the intracranial courses of these nerves is of the greatest importance for diagnosis. For instance, in the diagram of the origins of the cranial nerves (Fig. 17) it will be seen that a lesion at the side and upper part of the medulla might involve six of the most important trunks; that the long course of the sixth pair over the pons renders them very susceptible to injury; and, again, that some pairs, like the optic nerves at the chiasm, the third nerves, and the sixth pair, may be very readily affected together because of their nearness, or, on the other hand, that others may be seldom affected because of their wide separation at their superficial origin, as in the case both of the fourth and fifth pairs.

We now pass to the discussion of the entire course of each of these nerves.

The First or Olfactory Nerve.—Our knowledge of this nerve and its central connections is meagre. The peripheral filaments from the Schneiderian membrane pass through the numerous foramina of the ethmoid bone into the olfactory bulb lying along the under surface of the frontal bone. From this bulb the olfactory nerve passes backward, dividing into three roots. The fibres of the external root pass into the temporo-sphenoidal lobe of the same side and disappear in the anterior part of the uncinate gyrus, which is probably the cortical area for the sense of smell. The fibres of the middle root dis-

appear in the anterior perforated space, while those of the inner root pass to the anterior cerebral commissure. There is good reason for believing that each olfactory nerve is connected with the cortical areas for smell of

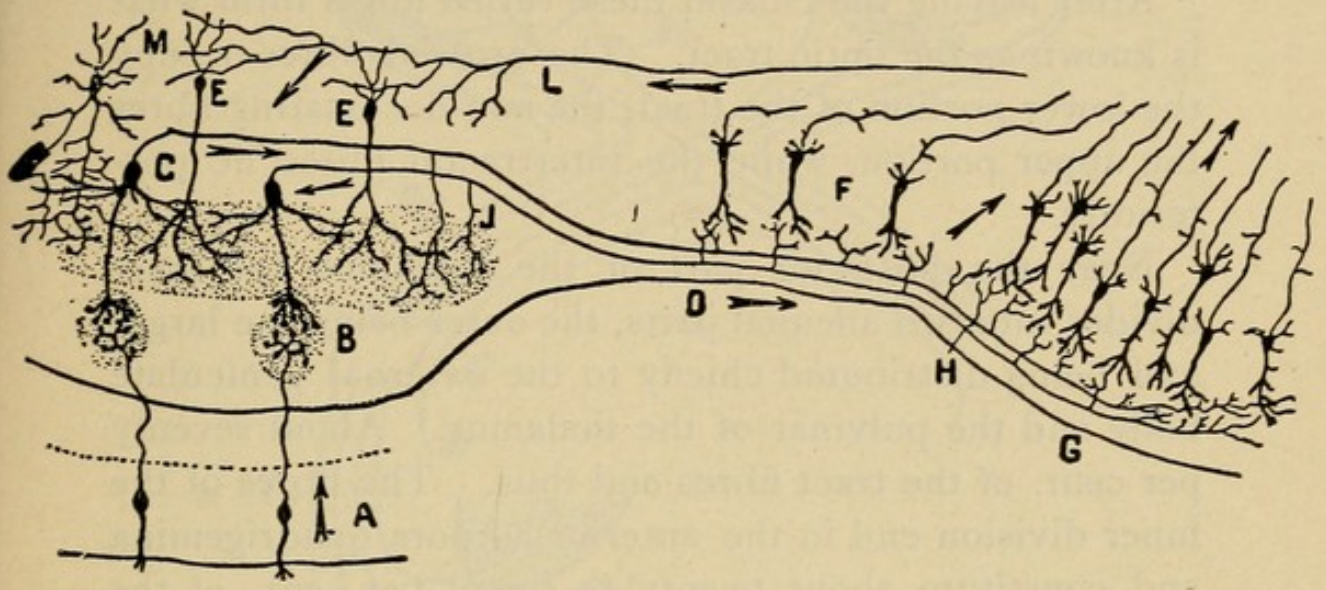


FIG. 36.

The olfactory bulb and tract. A, Schneiderian membrane in nose in which lies peripheral olfactory neuron; B, glomerulus of olfactory bulb; C, mitral cells with dendrites in B, and axones in D, olfactory lobe; E, granule cells; F, cells in olfactory lobe; H, G, fibres of olfactory tract. (Ramon y Cajal.)

both hemispheres, but the exact paths of these connecting fibres are not yet known.

The Second or Optic Nerve.—The nerve of sight is far more important in its diagnostic relations. Passing backward from the retina, the optic nerve undergoes partial decussation, the fibres from the nasal half of the fundus crossing to the opposite side. The nasal half of the retina corresponds to the temporal half of the field of vision, and as it is larger than the temporal half of the retina, owing to the limitation of the nasal visual field by the projecting nose, a greater number of fibres decussate than follow the direct path.

The amount of decussation varies somewhat in different animals and in individuals. In the chiasm we recognize three groups of fibres: (1) The decussating fibres

mentioned above (from the nasal side of the retina); (2) non-decussating fibres from the temporal side of the retina; (3) fibres passing between the two optic tracts and which have no connection with the retina. (See Fig. 37)

After leaving the chiasm these varied fibres form what is known as the optic tract. The crossed fibres occupy the lower portion of the tract, the non-decussating fibres the upper portion, while the intertractal fibres lie posteriorly.

Near the posterior part of the thalamus the tract divides into two unequal parts, the outer being the larger and being distributed chiefly to the external geniculate body and the pulvinar of the thalamus. About seventy per cent. of the tract fibres end thus. The fibres of the inner division end in the anterior corpora quadrigemina and constitute about twenty to thirty per cent. of the tract fibres. These endings are to be looked upon as the points of termination of the second neuron in this particular sensory system. The first neuron lies entirely within the retina.

From these various points the second neuron completes the system to the cortex, the fibres being gathered into a considerable bundle, the anterior arm of the corpora quadrigemina. These fibres pass to the most posterior portion of the internal capsule and finally reach the cortex through the optic radiation.

A few fibres probably reach the cortex without interruption in the basal ganglia.

The cortical areas in which the optic radiation terminates are certain portions of the occipital lobe, outer aspect, the cuneus, and certain areas about the calcarine fissure.

If it is the right optic path that is affected, the right retinal halves become blind. Hemianopsia has been

caused by diseases of the apex, external surface, and internal surface of the occipital lobe, but is most com-

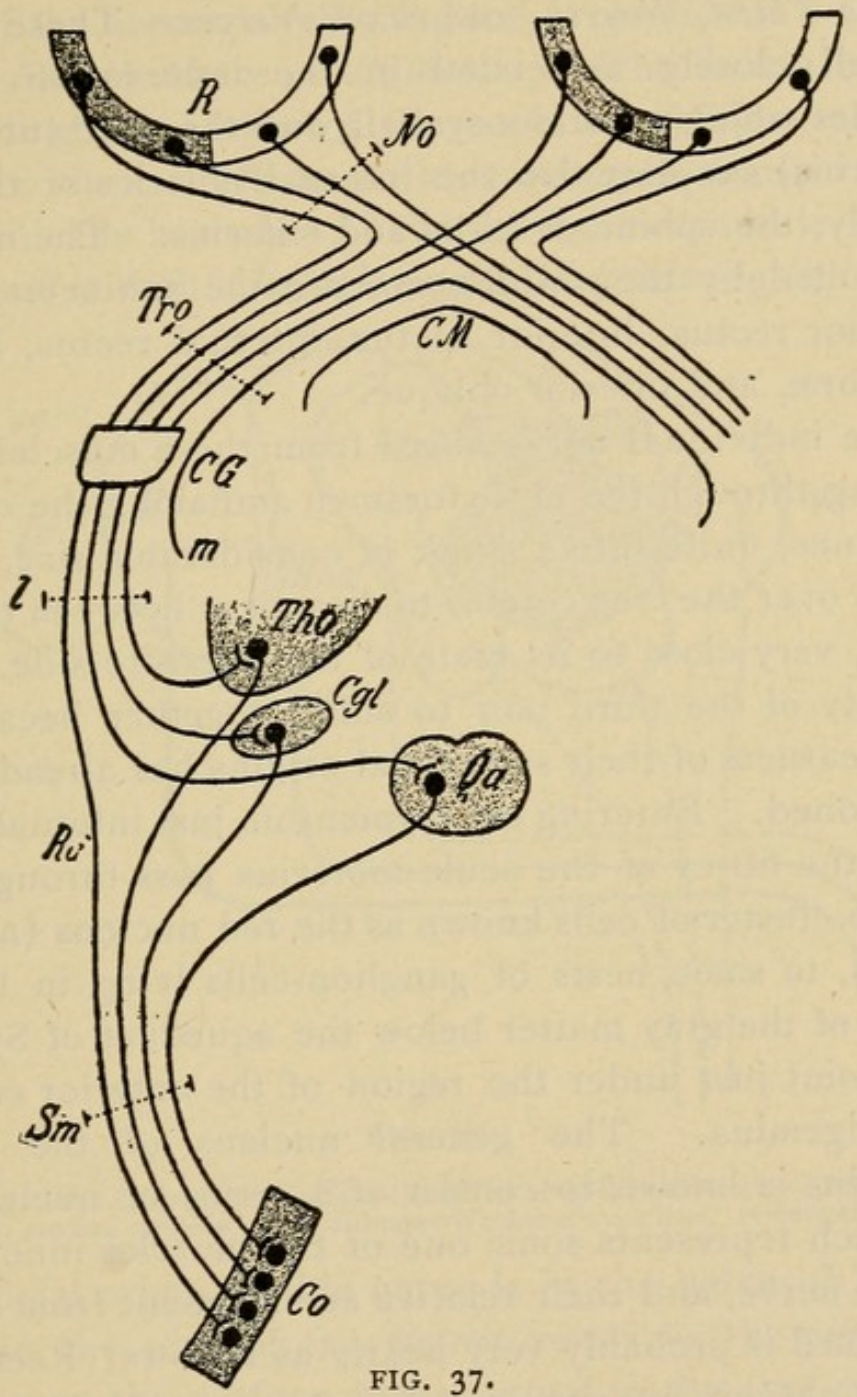


FIG. 37.

Schematic diagram of the central origin of the optic nerve. R, retina—dark portion derives its nerve supply from the left, while the light portion is supplied from the right hemisphere; No, optic nerve; Tro, optic tract; CM, Meynent's commissure; Cg, Gudden's commissure; l, lateral root tract; m, medial root tract; Tho, optic thalamus; Cgl, lateral geniculate body; Qa, anterior corpora quadrigemina; Rd, direct root tract to the cortex; Sm, optic radiation to the occipital lobes; Co, posterior occipital cortex. (Obersteiner.)

mon when the cuneus is involved; and it may be, when other occipital areas than the cuneus are invaded with

resulting hemianopsia, that the underlying fibres of the optic radiations are included in the lesion.

The Third, Fourth, and Sixth Nerves.—These nerves are all closely associated in the innervation of the muscles which move the eyeball, and the third (or oculo-motorius) supplies also the internal muscles of the eye, namely, the sphincter iridis and ciliarius. The muscles controlled by the third nerve are the sphincter iridis, superior rectus, inferior rectus, internal rectus, levator palpebræ, and inferior oblique.

The individual nerve-fibres from these muscles, after passing through the optic foramen and along the cavernous sinus, unite into a trunk of considerable size, which winds over the crus cerebri to disappear between the two crura, very close to its mate of the opposite side. The liability of the third pair to suffer together because of this nearness of their superficial origins has already been mentioned. Entering the tegmentum just internal to the crus, the fibres of the oculo-motorius pass through and by the cluster of cells known as the red nucleus (nucleus ruber), to some nests of ganglion-cells lying in the interior of the gray matter below the aqueduct of Sylvius, at a point just under the region of the anterior corpora quadrigemina. The general nucleus of the oculo-motorius is known to consist of a series of nuclei each of which represents some one of the muscles innervated by the nerve, and their relative arrangement from before backward is probably very nearly as follows: Rectus inferior, obliqus inferior, rectus internus, rectus superior, levator palpebræ. (See Fig. 38.) The cell group bearing relationship to the ciliary muscles of the iris is probably separate from the others and is constituted in a group known as the Edinger-Westphal nucleus, the two halves of which are situated near the median line.

The arrangement of the muscle representation in the nucleus from without inward is also shown in the diagram of the accompanying sagittal projection.

The Fourth Nerve or Patheticus.—The fourth nerve, controlling the action of the superior oblique muscle, lies upon the outer side of the cavernous sinus in company with the branches of the third nerve, and is hence apt to suffer with it in lesions affecting this region. The

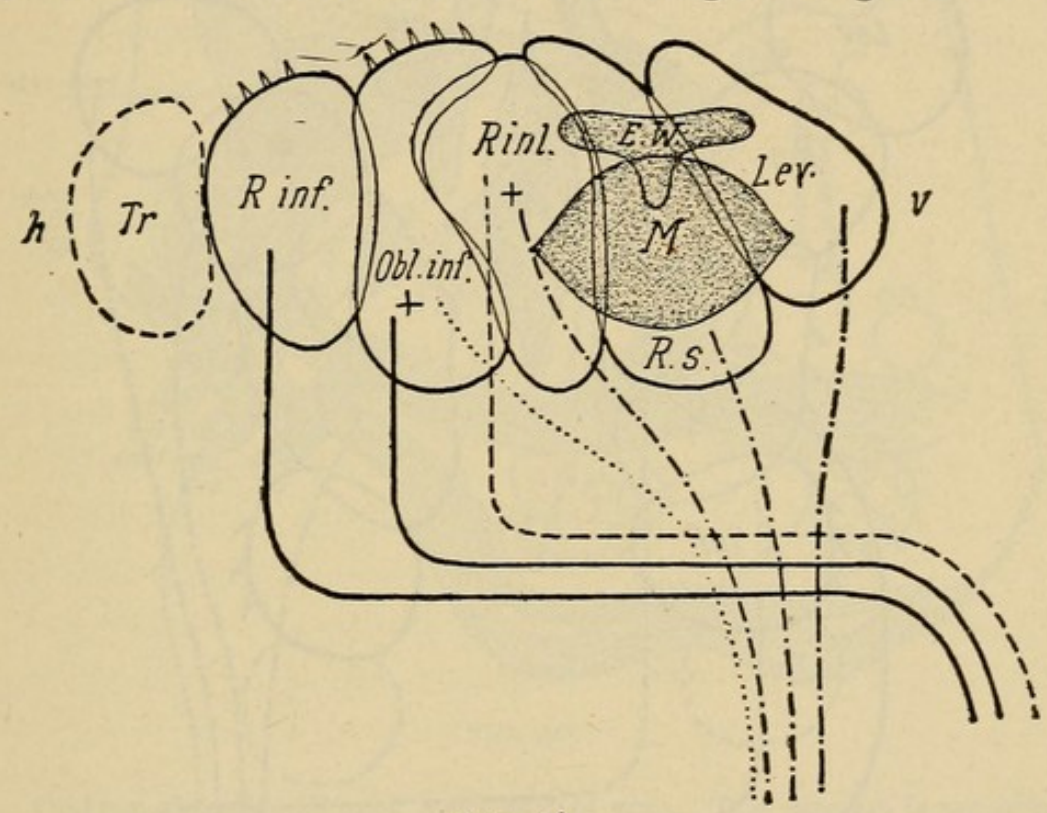


FIG. 38.

Scheme of the oculo-motor nucleus (modified after Bernheimer), sagittal projection. M, median nucleus ; EW, Edinger-Westphal's nucleus. (Obersteiner.)

superficial origin of this nerve is in the valve of Vieussens, in the roof of the fourth ventricle, the nerve appearing on the surface just external to the crus cerebri, and on a level with the upper border of the pons.

Within the substance of the valve there is a decussation of the fourth pair of nerves. It is probable that all of the fibres take part in this decussation. Then the fibre-bundles pass downwards and forwards around the

Sylvian aqueduct to the gray matter in its floor, just posterior to the nuclei of the third nerve.

The Sixth Nerve or Abducens.—This nerve supplies the external rectus and probably gives some fibres also to the internal rectus. In the cavernous sinus it lies beside the fibre-bundles of the third and fourth nerves.

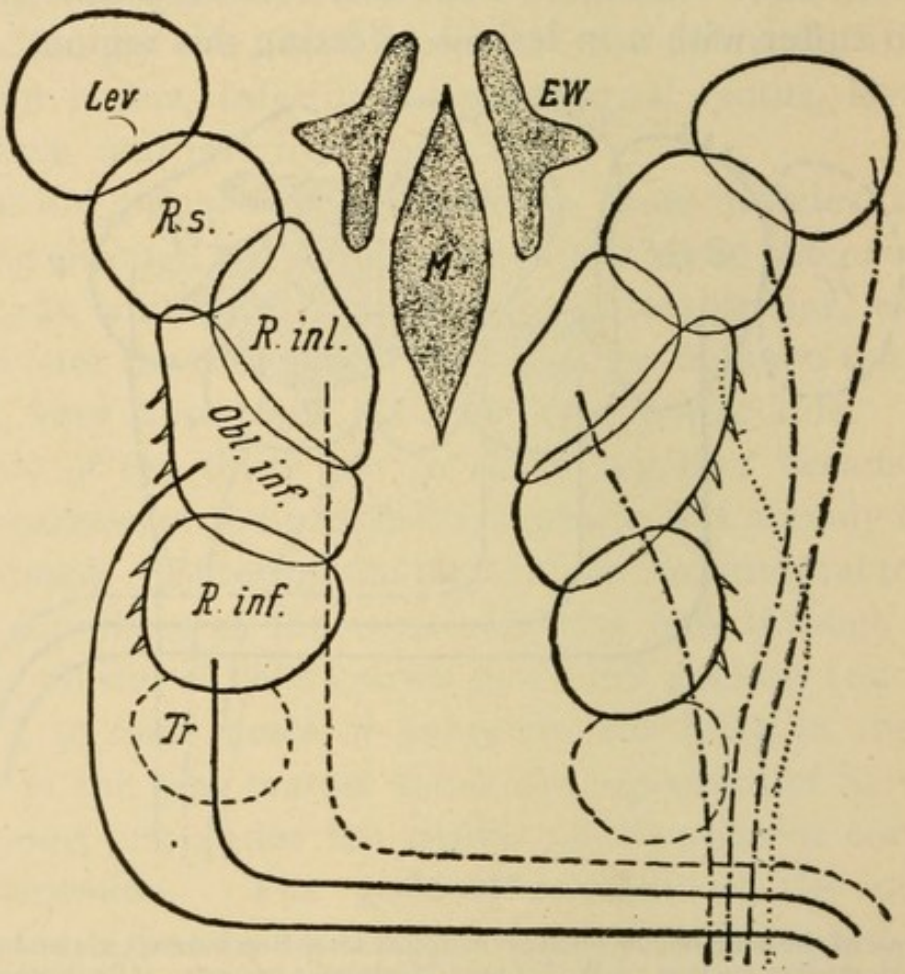


FIG. 39.

Scheme of the oculo-motor nucleus (modified after Bernheimer), basal projection. M, median nucleus; EW, Edinger-Westphal's nucleus. (Obersteiner.)

From here it takes a long course over the prominent convex surface of the pons (thus rendering it very liable to suffer compression, especially from sub-tentorial growths) to its superficial origin between the lower pontine border and the pyramid of the medulla, not far from the median line. Both nerves often suffer together

because of their nearness to each other in their origin and course, and lesions at the base of the brain sometimes involve one of the sixth nerves together with the fifth, for the abducens passes very close to the superficial origin of the latter in its course over the pons.

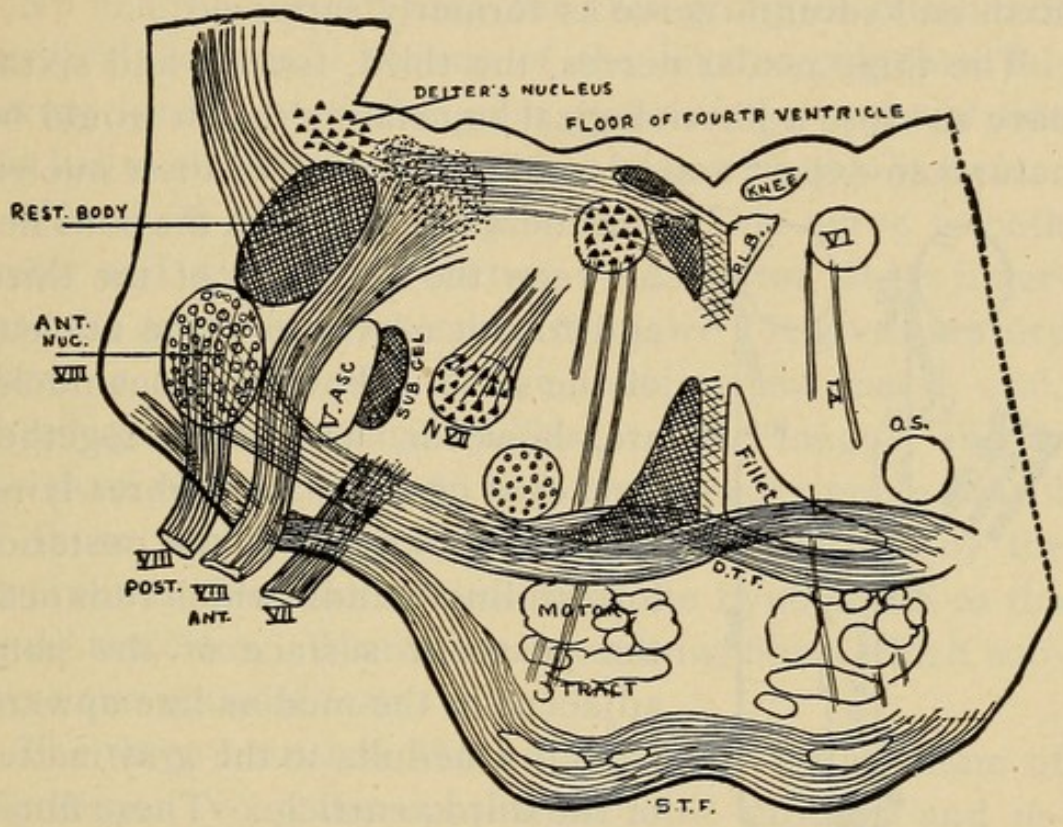


FIG. 40.

Diagram of section through lower part of pons. This diagram is not quite accurate, as it represents, for convenience, certain structures which are not actually observed at exactly the same level. (Thus it shows the origin, course, and exit of the 7th nerve.) VI, abducens nerve and nucleus; N VII, nucleus of facial nerve; VII, facial nerve; VIII ant. and VIII post., anterior and posterior or deep and superficial roots of the auditory nerve; Corp. Rest., restiform body: Knee, knee of the facial nerve; O. S., superior olive; P.L.B., posterior longitudinal bundle; S.T.F., superficial transverse fibres of the pons; D.T.F., deep transverse fibres of pons; V. Asc., ascending root of trigeminus; Sub. Gel., substantia gelatinosa. The internal or "chief" auditory nucleus lies just internal to the nucleus of Deiter's.

The central origin of the sixth nerve is from a small nucleus immediately below the floor of the fourth ventricle, beneath the eminentia teres and in front of the striæ acusticæ. A transverse section of the pons at this level shows the seventh nerve curving about this

nucleus of the sixth nerve, and these two nerves are sometimes affected together by a lesion. When they are thus involved, and without including the eighth nerve, disease of the pons is indicated. There is no direct anatomical connection between the nuclei of the sixth and seventh nerve as formerly supposed.

The three ocular nerves, the third, fourth, and sixth, have so close a physiological association that it would be natural to expect considerable proximity of their nuclei. But the whole length of the pons lies between the cell-nests of the third and fourth nerves and the nucleus of the sixth. These various nuclei are, however, associated together by means of association-fibres lying in the tract known as the posterior longitudinal bundle which runs near the posterior surface of the pons adjacent to the median line upward from the medulla to the gray matter of the third ventricle. These fibres subserve the conjugate movement of the eyes from side to side, for in such movements the external rectus of one eye (supplied by the sixth nerve) and the internal rectus of the other eye (supplied by the third nerve) must act simultaneously, as shown in Fig. 43.

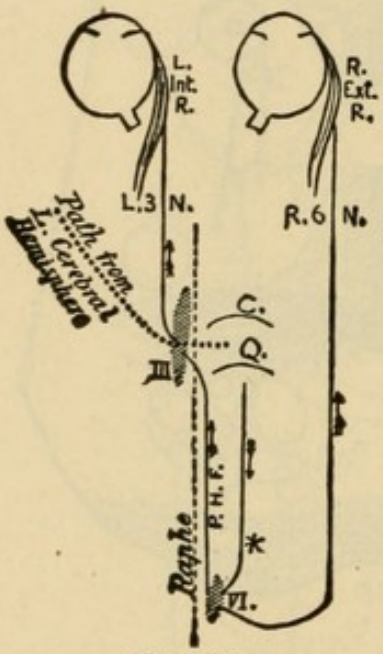


FIG. 41.

Diagram of the mechanism for the associated lateral movement of the eye. (Gowers.)

It is highly probable that these muscle groups have a bilateral cortical representation. That is, each muscle is represented on both sides in the cerebral cortex. This is true of some other muscle groups as well, such as the upper facial and the back muscles. In fact, the law of bilateral representation seems to apply to certain muscle

groups capable of being used voluntarily, but under constant usage having passed from under the immediate control of the will. We are thus enabled to understand why complete paralysis of these muscles of cortical origin is never seen.

No definite cortical area for this group of muscles is positively known. Paresis of the levator palpebræ following lesions of the angular gyrus of the opposite side has led to the association of this area with this muscle.

The Fifth Nerve or Trigeminus.—This nerve is both motor and sensory in function. Its motor fibres innervate the muscles which move the jaw. It gives sensory fibres to all parts of the face, conjunctiva, nasal, oral, pharyngeal, and buccal cavities, to the tongue, and to the dura mater. In fact, it is the sensory nerve for all of those parts whose movements are governed by the several motor-cranial nerves from the hypoglossal to the oculo-motorius. Moreover, it contains fibres which subserve the sense of taste.

The three branches which have given it the name of trigeminus, viz., the ophthalmic and superior and inferior maxillary nerves, unite into one great trunk, about an inch from the superficial origin of the nerve, upon meeting in the Gasserian ganglion. The inferior maxillary branch contains all the motor fibres (see Fig. 44). The sensory root enters the pons in close company with the motor root about the middle of the pons, but at some distance from the median line. The origins of this pair of nerves are the most widely separated of any of the cranial nerves, and to this is due the rarity of their involvement together in any one lesion. They are also quite remote from the roots of other nerves, the nearest being those of the fourth and sixth.

The nuclear origin of the fifth nerve is very com-

plex as shown in Fig. 43, particularly in its sensory portion. The sensory root passes backward and inward through the outer part of the pons to some small groups of cells lying beneath the floor of the fourth

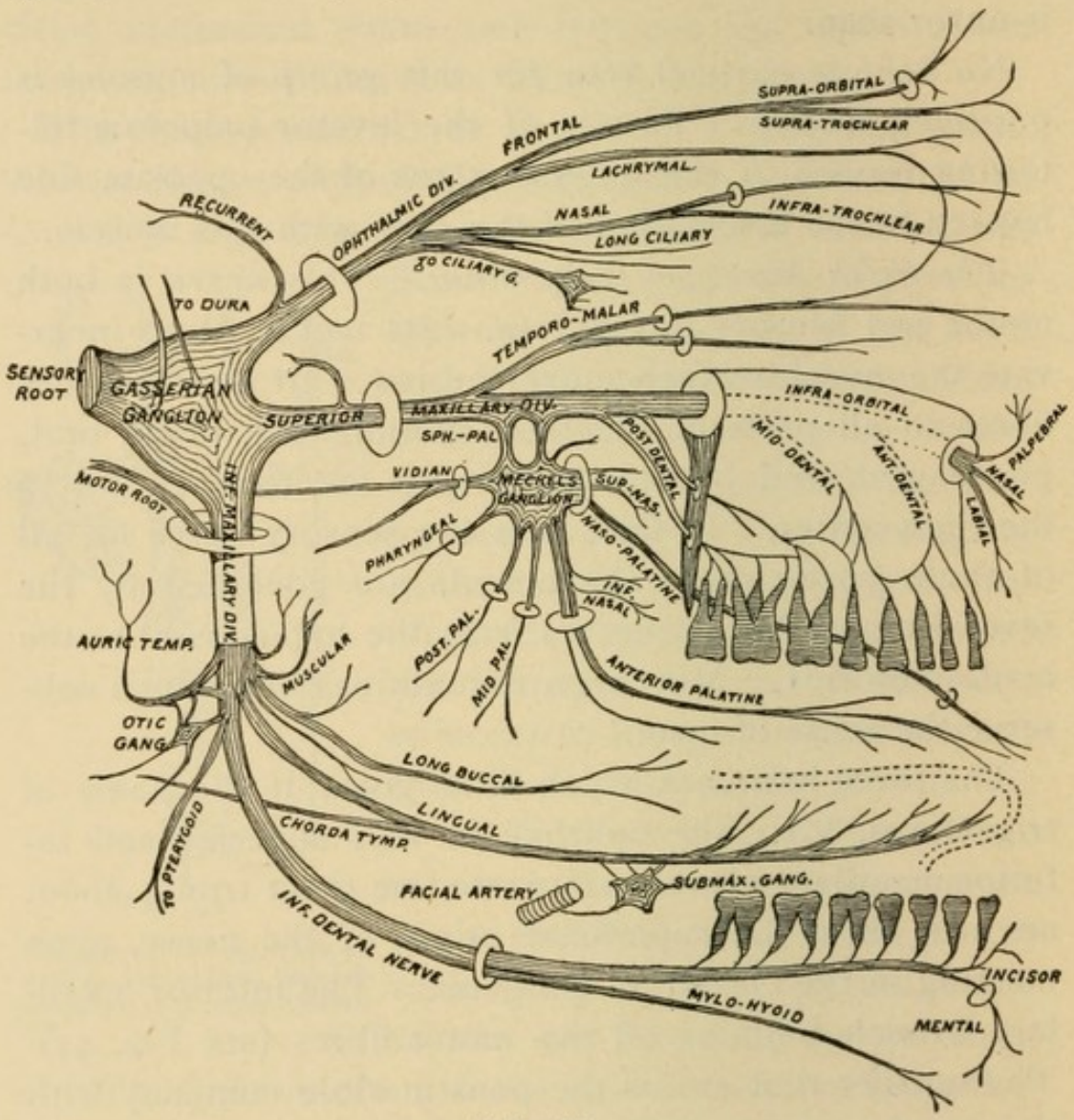


FIG. 42.

Fifth nerve or trigeminus.

ventricle known as the middle nucleus. The majority of the fibres, however, turn downwards through the pons and medulla under the name of the descending root. This root lies just internally to the restiform body, and, growing smaller as it passes downwards, term-

inates in the upper part of the spinal cord in close connection with the upper expansion of the gelatinous

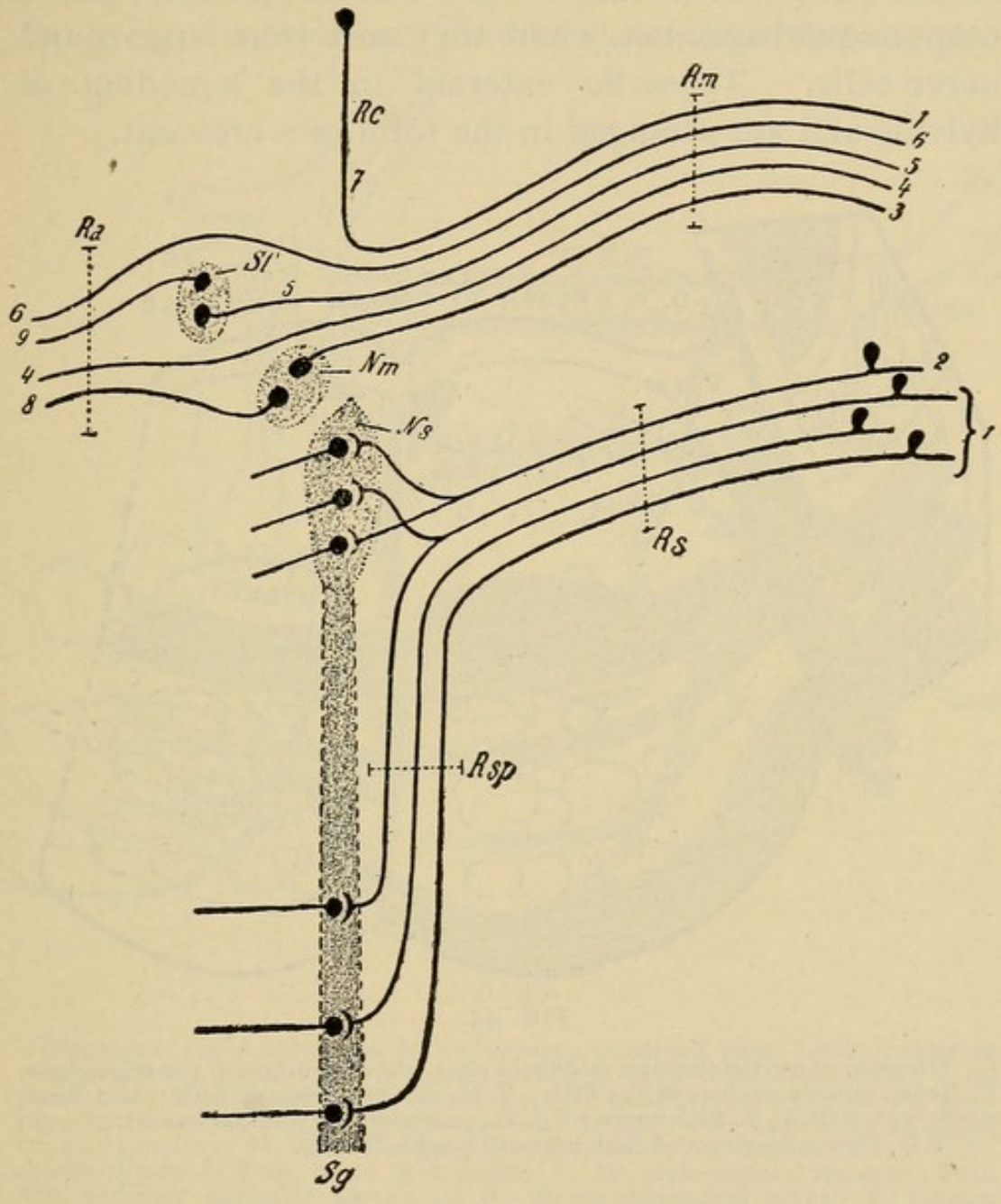


FIG. 43.

Scheme of the central course of the trigeminal nerve. Rs, sensory root; Rm, motor root; Rsp, spinal root; Rc, cerebral root; Ns, sensory nucleus; Sg, substantia gelatinosa; Nm, motor nucleus; Sf, substantia ferruginea; Ra, raphe. (Obersteiner.)

substance of the posterior horn (the gray tubercle of Rolando).

The descending root consists of several small bundles which may be traced from the level of the middle nucleus in an upward direction as high as the upper part of the corpora quadrigemina, where they arise from large round nerve-cells. These lie external to the aqueduct of Sylvius and are arranged in the form of a crescent.

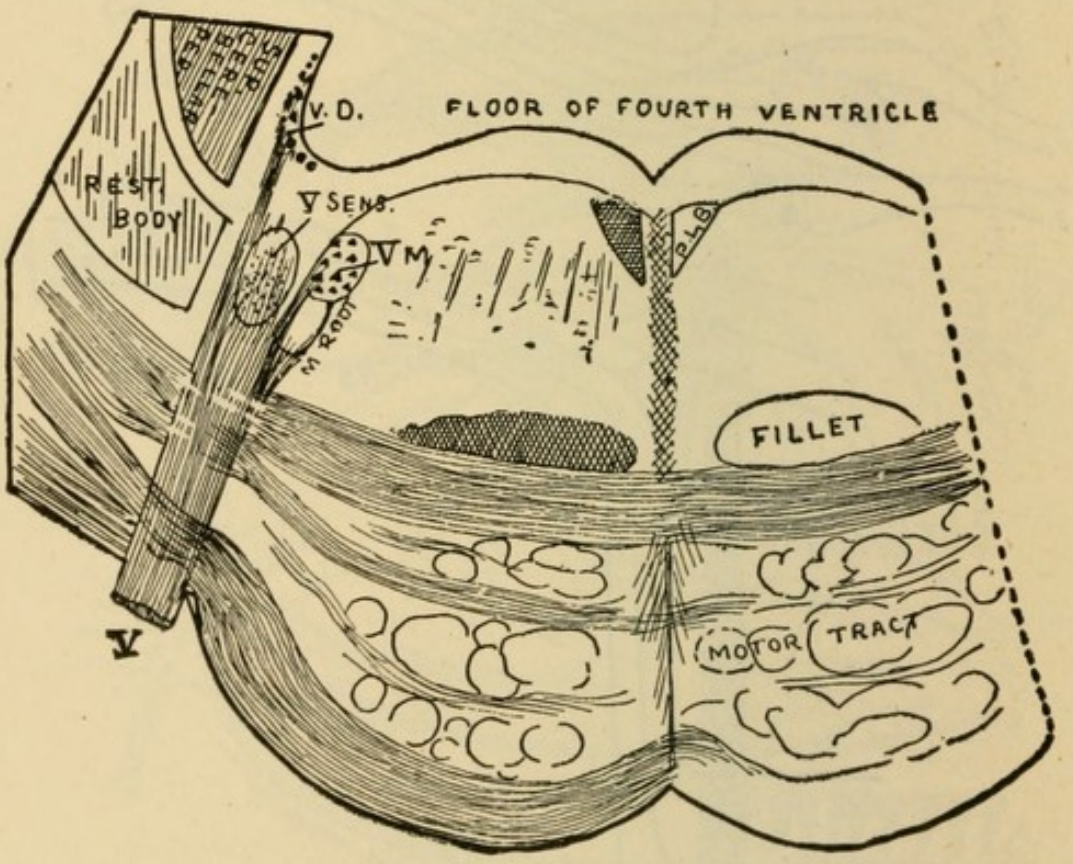


FIG. 44.

Diagram of section through middle of pons, showing origin of the trigeminus. V Sens., sensory nucleus of the fifth; V M, motor nucleus of fifth; M Root, motor root of fifth; V, fifth nerve; P.L.B., posterior longitudinal bundle. V D, Descending root of fifth nerve ("trophic" root).

The majority of the fibres of the motor root arise from a collection of large ganglion-cells of limited vertical extent which lies just internal to the sensory root of the fifth nerve in the lateral part of the tegmentum of the pons.

There are many intricate arrangements of the sensory portion of this nerve. Its so-called descending root

corresponds to the Rolandic substance of the posterior horns of the spinal cord and really consists of small cells from which the sensory fibres destined ultimately to join the great sensory tract, the lemniscus, take their origin.

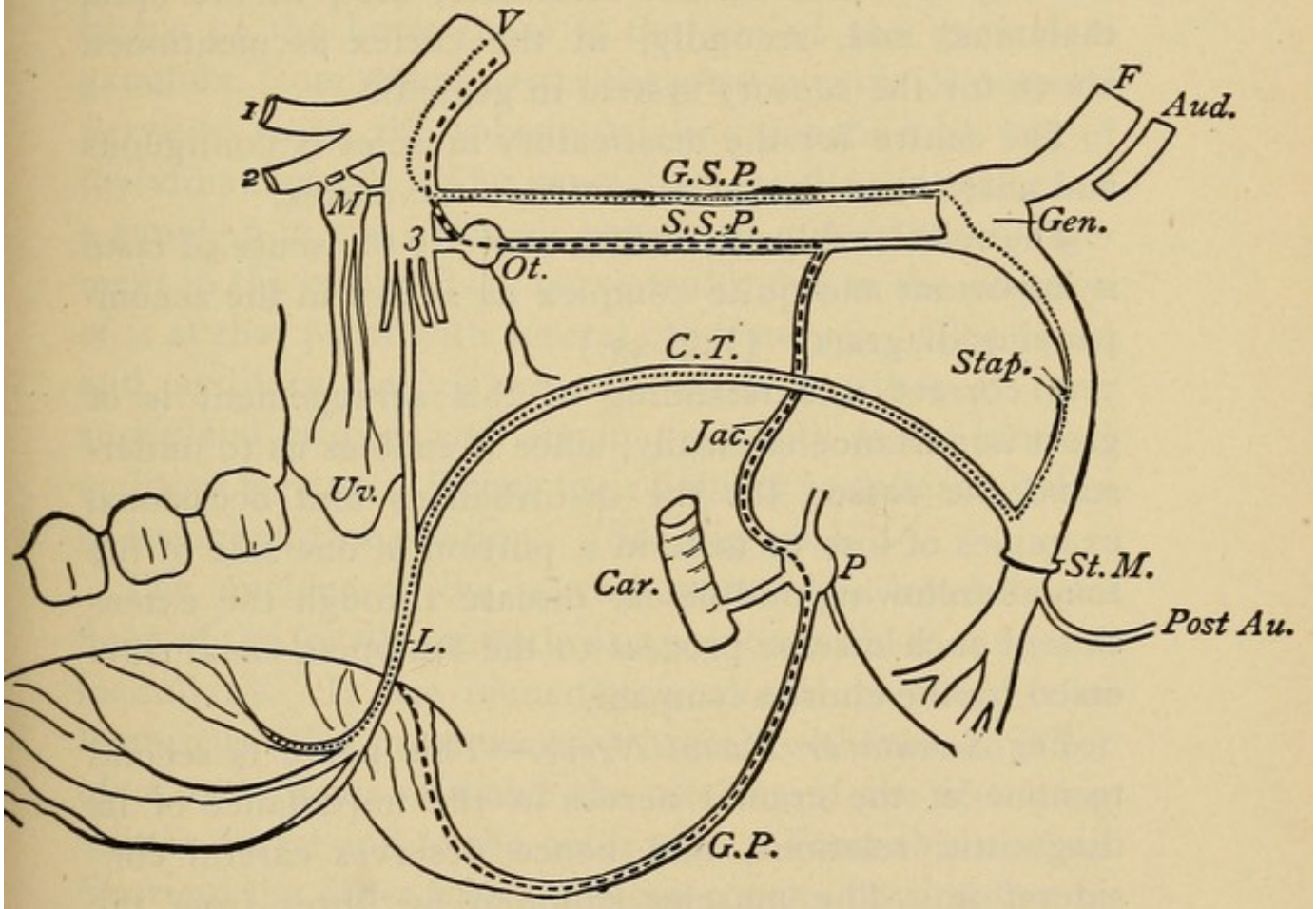


FIG. 45.

Diagram of facial nerve and its connections (modified from Leube), showing course of taste fibres. V, trigeminal nerve; 1, 2, 3, first, second, and third divisions of trigeminus; F, facial nerve; Aud, auditory nerve; G. S. P., great superficial petrosal; S. S. P., small superficial petrosal; Gen, geniculate ganglion; Ot, otic ganglion; M, Meckel's ganglion; Uv, uvula; L, lingual nerve; C. T., chorda tympani; Stap, nerve to stapedius; St. M., stylo-mastoid foramen; Post. Au., posterior auricular nerve; G. P., glosso-pharyngeal nerve; P, petrous ganglion of glosso-pharyngeal; Jac, Jacobson's nerve; Car, tympanic plexus near carotid artery.

N. B.—Dotted line = taste fibres from anterior two thirds of tongue; dash line = taste fibres from posterior third of tongue. (Allchin.)

The distribution of the sensory fibres so originating forms no exception to the disposition of the sensory system generally, as already described. There is a partial de-

cussation of the tract so formed, a considerable portion passing to the opposite side, the fibres ultimately joining the lemniscus in the region of the red nucleus. Fibres from the same side also pass to the cerebellum. Those which join the lemniscus terminate, first, in the optic thalamus, and, secondly, in the cortex as mentioned above for the sensory system in general.

The centre for the masticatory muscles is contiguous and anterior to the facial centre in the cortex.

The relationship of the fifth nerve to the sense of taste is important and quite complex as shown in the accompanying diagram. (Fig. 45.)

A correct understanding of this arrangement is of great importance clinically, since it enables us to understand the reason for the disturbances and occasional examples of loss of taste in a portion of one side of the tongue following middle-ear disease through the extension of such disease process to the Fallopian canal traversed by the chorda tympani.

The Seventh or Facial Nerve.—This nerve is second to none of the cranial nerves in the importance of its diagnostic relations, and hence deserves careful consideration. The muscles supplied by fibres from the facial nerve are as follows: Occipito-frontalis, muscles of the external ear, stylo-hyoid, posterior belly of digastric, all of the facial muscles, platysma, stapedius, tensor of palate and azygos uvulæ (possibly).

The nerve takes a winding course from its superficial origin at the lower margin of the pons, between the olivary and restiform bodies, to its point of exit from the stylo-mastoid foramen, passing through the Fallopian canal of the temporal bone. Within the Fallopian canal the nerve is separated from the middle ear by only a thin lamina of bone pierced by foramina for a small artery,

the chorda tympani nerve, and the nerve to the stapedius. This fact is of importance, because it explains how inflammatory processes in the middle ear may extend by contiguity to the facial nerve, with resulting paralysis.

The angle or knee made by the seventh nerve at the hiatus of the bony canal is the site of the geniculate ganglion, from which passes the great superficial petrosal nerve to reach the spheno-palatine ganglion by way of the vidian canal. The geniculate ganglion is really not a ganglion in the ordinary sense, but merely an enlargement in the curve of the nerve-trunk due to the junction of it at that point with several other nerves. The facial and auditory nerves are very close together at their superficial origins, and are in proximity at the internal auditory meatus. Hence their liability to suffer together in lesions in this position. (See Fig. 46.)

The nucleus of the seventh nerve lies beneath the floor of the fourth ventricle at a point ventral to the striæ medullaris. It lies immediately dorsal to the medial lemniscus (the great sensory tract) with the superior olive to its inner side and the nucleus of the fifth lying a little dorsal and to the outer side. From this nucleus of origin the fibres take a devious course, passing first to a point dorsal to the nucleus of the sixth nerve, where they ascend for a short distance, finally making their exit from the medulla at the point above mentioned. (See 50.)

There is good reason for believing that some of the muscles supplied by fibres from the facial are not represented in the facial nucleus, but in the nuclei of other nerves. This applies especially to the fibres composing the superior branch of the facial and which are distributed to the occipito-frontalis, the corrugator supercilii, and the orbicularis palpebrarum. This group of muscles

is not paralyzed in palsies of cerebral origin, but only in palsies involving the trunk. This can also be explained on the theory of the bilateral cortical representation of these muscles, in favor of which there is some evidence.

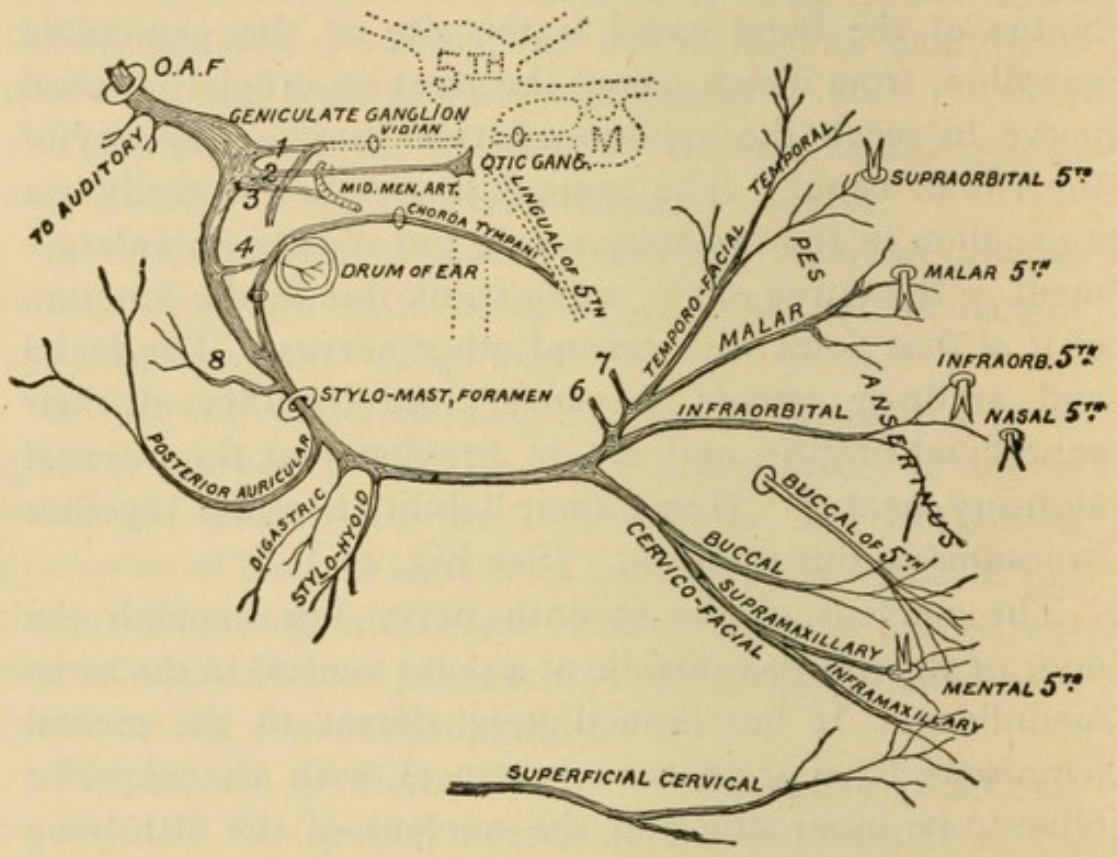


FIG. 46.

Seventh nerve, facial, diagrammatic.

1. Great petrosal, to form vidian with No. 5.
2. Small petrosal, to otic ganglion.
3. External petrosal, to plexus on mid. meningeal artery.
4. Tympanic br. to stapedius, etc.
5. Br. from carotid plexus, making vidian with No. 1.
6, 7. Brs. to auriculo-temporal of 5th.
8. Br. to auricular of vagus.
M. The ganglion of Meckel.
O. A. F. Orifice of aqueductus Fallopii.

The fibres to the orbicularis palpebrarum originate in the nucleus of the third nerve, but course in the seventh.

The orbicularis oris probably has a certain representa-

tion in the hypoglossal nucleus, because of the intimate association of the movements of the lips and the tongue.

The fibres from the facial nucleus to the cortex decussate in the pons just above the nucleus. Hence, in facial paralysis from a one-sided lesion above the middle of the pons the paralysis of the face is on the side opposite to the lesion, whereas in lesions below this level the paralysis is on the same side. The course of the fibres through the crus and internal capsule, and the facial cortical area have already been described.

The Eighth or Auditory Nerve.—The auditory nerve is formed by the union of two sets of fibres derived respectively from the cochlea and vestibule and from the semicircular canals. (See Figs. 47 and 48.) These sets of fibres differ in function, the former conducting sound-waves, and the latter serving to carry from the semicircular canals impressions relative to the movements and position of the body. Changes in bodily position probably cause irritation of the mucosa of the semicircular canals by means of solid bodies contained in the endolymph.

Thus we are to conceive of the vestibular portion of this nerve as conducting impressions relative to the position of the body in space and are to consider the semicircular canals of the vestibule as the point from which these special sensations depart. This sensory nerve follows the same arrangement as obtains for sensory nerves in general. The fibres are first interrupted in the vestibular ganglion and from this point pass to enter the medulla together with the cochlear fibres at a point ventral to the corpus restiformis, and to end finally in the vestibular nucleus lying immediately beneath the striæ medullaris of the floor of the fourth ventricle. There is also a group of larger cells lying to the outer side of this nucleus, which is commonly known as Deiter's

nucleus, and these two cell groups form the chief endings in the medulla of the vestibular fibres. There are two other nuclei which belong properly to the cerebellum which are a part of the vestibular system and in which

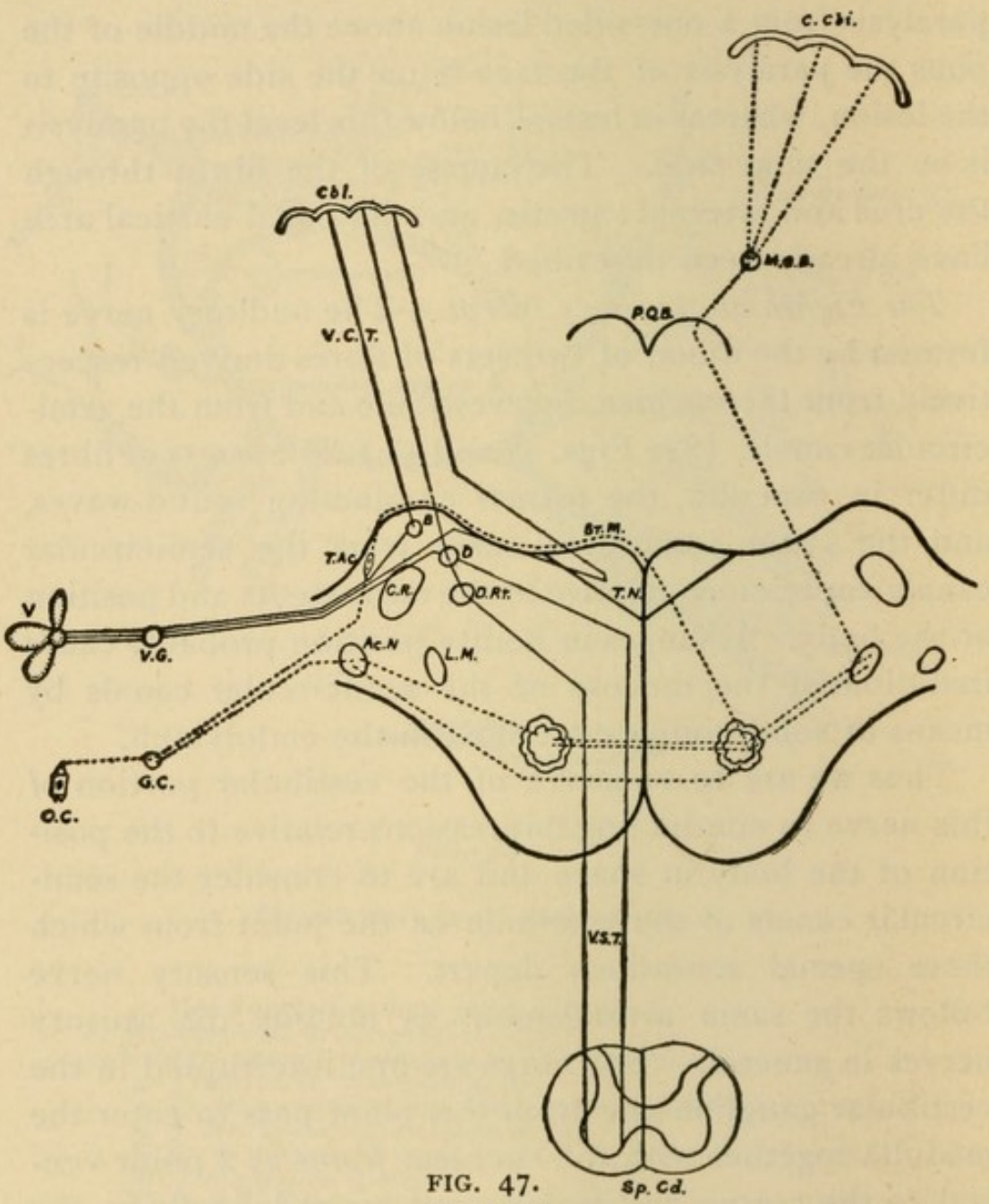


FIG. 47.

Eighth or auditory nerve, diagrammatic. Thin black lines, vestibular division of eighth nerve; dotted lines, cochlear division of eighth nerve; V., vestibule; V.G., vestibular ganglion; cbl., superior vermis of cerebellum; B., nucleus of Becterew; D., nucleus of Deiter; D.Rt., descending root of eighth nerve; T.N., triangular nucleus; V.S.T., vestibulo-spinal tract; V.C.T., vestibulo-cerebellar tract; O.C., organ of Corti; G C., ganglion cochleare; T.Ac., tubercular acusticum; St.M., striæ medullares; Ac.N., accessory nucleus; O S., superior olive; L.M., lateral lemniscus; P.Q B., posterior quadrigeminal body; M.G.B., median geniculate body; C.cbi., cortex cerebri, third temporal convolution; C.R., corpus restiforme.

vestibular fibres terminate, viz., the nucleus angularis and the nucleus tecti. The association of these various nuclei with the spinal cord is brought about by means of two systems of fibres which extend the vestibular apparatus to the lower levels of the cord. A portion of these fibres course in the fasciculus longitudinalis posterior and another in the so-called descending acoustic root. It is to be noted that the latter really contains no acoustic fibres whatever. The cortical termination of this system is in the vermis of the cerebellum. We are thus enabled to understand the occurrence of motor disturbance in diseased conditions involving the middle lobe of the cerebellum on the one hand or the semicircular canals on the other (Ménière's disease).

The cochlear fibres of the eighth nerve are those immediately concerned with the sense of hearing. Beginning in the mucous membrane of the cochlea the fibres are first interrupted in the cochlear ganglion lying in the whorl of the cochlea. The fibres proceed, together with those from vestibular portion above described, to a point below the pons and ventral to the corpus restiformis to enter the medulla. The primary distribution of these fibres is twofold; one portion passing to the ventral acoustic nucleus which lies immediately ventral to the corpus restiformis and the other portion passing to the tuberculum acusticum (accessory nucleus) lying dorsal to the corpus restiformis. These two nuclei are the primary medullary nuclei of the cochlearis. From these two nuclei the acoustic tract is continued by means of fibres from the accessory nucleus which form the striæ acusticæ on the floor of the fourth ventricle and cross the middle line to the opposite side, and fibres from the chief nucleus (ventral), some of which pass by means of the corpus trapezoides to cross the median line and join

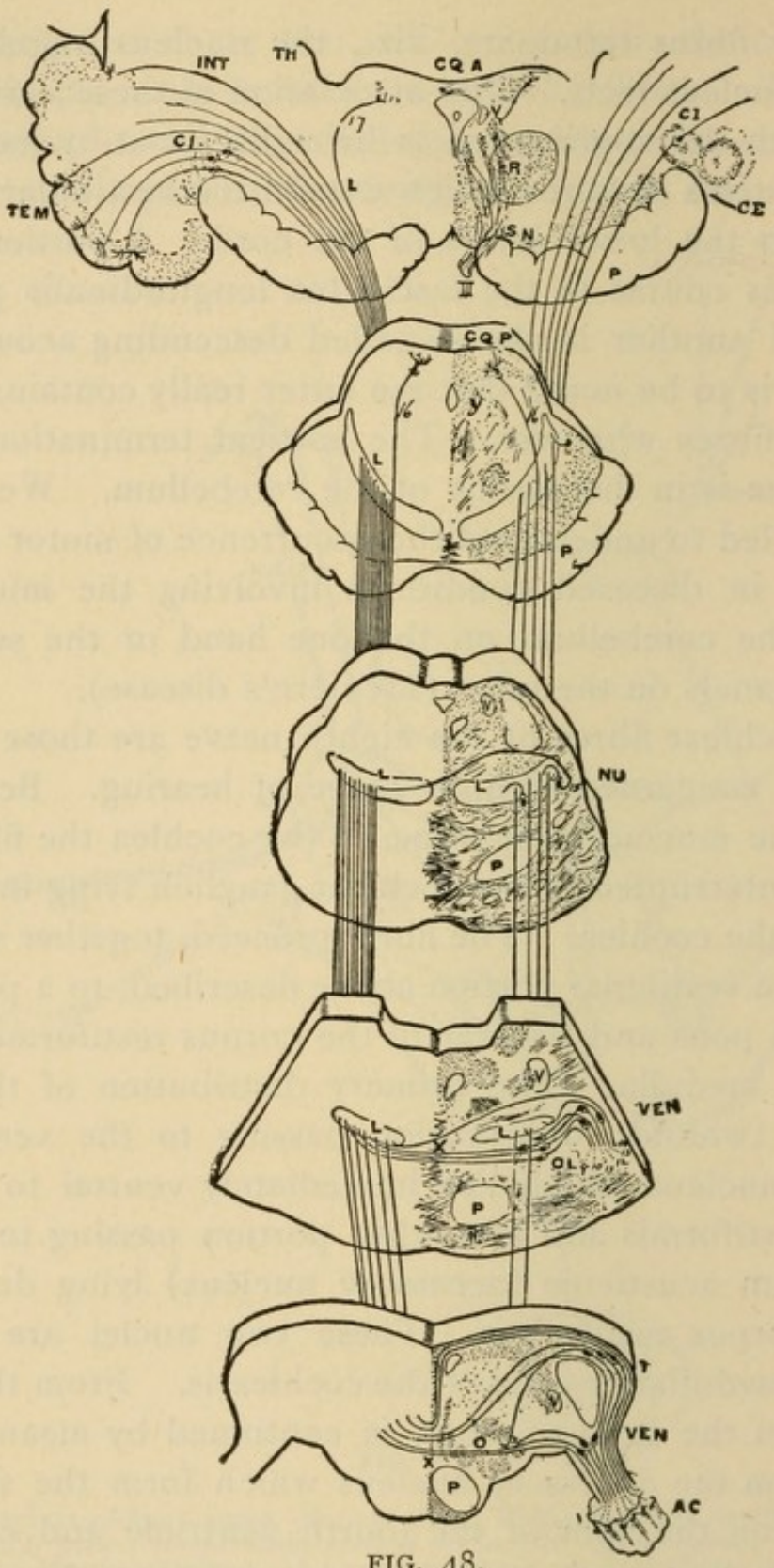


FIG. 48.

The auditory tract. Th, optic thalamus; Int, int. capsule; Ci, corp. geniculatum int.; Tem, temporal lobe; L, lemniscus; Cqa, corp. quadrigeminum ant.; Cqp, corp. quad. post.; R, red nucleus of tegmentum; Sn, substantia nigra; Ce, corp. geniculatum ext.; P, pes. pedunculi and pyramidal tract; Nu, nucleus of lemniscus; Ven, ventral nucleus of auditory nerve; V, fifth nerve; Ol, olivary nucleus; O, olive; X, decussation of auditory fibres; Ac, auditory nerve from cochlea; T, dorsal nucleus of auditory nerve. (Starr.)

the fibres from the striæ acusticæ and end in the superior olive of the opposite side. From the superior olive the acoustic tract is continued cerebral-wards by means of the lateral lemniscus which forms to the outer side of the superior olive to end in the posterior quadrigeminal body and to be further continued cerebral-wards as the brachium of the posterior quadrigeminal body. These fibres terminate in the middle geniculate body of the thalamus opticus and are finally distributed to the cortex of the first temporal convolution. The course of the auditory fibres is thus seen to be quite complex.

The wide difference in function between the cochlear and the vestibular fibres of the eighth nerve should always be borne in mind.

The left cortical auditory area is especially related to the memory of words, its destruction producing the form of aphasia known as word deafness.

The fact that the eighth nerve at the base of the brain and at the internal auditory meatus lies side by side with the facial is an anatomical point of some importance.

The Ninth or Glosso-Pharyngeal Nerve.—The ninth nerve is distributed to the back part of the tongue, the soft palate, the upper part of the pharynx, and the Eustachian tube. Besides supplying taste-fibres to the posterior part of the tongue and motor fibres to the other parts mentioned, it probably also furnishes fibres of common sensation to these parts and to the tympanic cavity.

It has already been stated that although taste-fibres are found in the peripheral portion of this nerve, they doubtless ultimately enter the brain with the fifth nerve.

Considerable uncertainty also exists as to the motor function of the ninth nerve. The motor fibres to the upper part of the pharynx come through the pharyngeal

plexus, but whether from the ninth or tenth nerve cannot be positively stated. The digastric branch of the seventh communicates with the ninth, and it is not improbable that the stylo-pharyngeus derives its motor supply from the former. Whether the ninth sends any motor fibres to the palate has not been determined.

This uncertainty regarding the exact function of the ninth nerve is due mainly to the fact that in man the nerve is seldom damaged by disease or injury apart from other adjacent cranial nerves, and experiments on animals have not been altogether satisfactory. Thus it happens that the functions of the nerve are inferred from a study of its anatomical distribution—a method which is apt to be misleading.

The deep and superficial origins of the glosso-pharyngeal will be considered in conjunction with those of the tenth and eleventh nerves.

The Tenth, Vagus, or Pneumogastric Nerve.—This nerve has the widest distribution of any cranial nerve, supplying as it does the vocal and respiratory organs, the heart, most of the alimentary canal, and some of the abdominal viscera. It has both sensory and motor fibres. It sends sensory branches to the meninges along the middle meningeal artery, to the posterior part of the external auditory meatus, to the pharynx, larynx, trachea, bronchi, heart, œsophagus, stomach, and intestines. Its motor fibres go to the pharynx and larynx. The vagus is the inhibitory and accelerator nerve of respiration and the inhibitory nerve of the heart. It contains also motor fibres for the œsophagus and in part for the stomach and intestines. After receiving many fibres from the spinal accessory nerve, the pneumogastric trunk passes down the neck in the same sheath with the carotid artery. Its pharyngeal branches unite with those of the ninth to form

the pharyngeal plexus. It gives off the superior and inferior (recurrent) laryngeal nerves, which supply all the muscles of the larynx. In the abdomen it enters into numerous conjugations with the splanchnic nerve.

The tenth nerve emerges from the side of the medulla just anterior to the restiform body, as a series of rootlets, the filaments of the ninth lying immediately above, and the root of the eleventh immediately below. The line of separation between these three nerves at their superficial origin is not sharply defined, and hence it is rare for any lesion at this point, even though small, to affect one of these nerves alone.

A part of the *Eleventh or Spinal Accessory Nerve* joins the pneumogastric, and receives its title because of being accessory to the vagus. The filaments which arise in this close relation to the tenth nerve constitute the accessory portion, the spinal portion arising at a wide interval from a lower level, in the anterior horn of the spinal cord.

The central origin of the ninth, tenth, and eleventh nerves is from a continuous series of cell-nests forming a longitudinal, narrow, gray column, which lies just lateral to the eminentia teres of the fourth ventricle, with its long axis running downward from the level of the auditory nucleus above.

Some of the upper cells of this column give origin to the ninth, the middle cells to the tenth, and the lowest cells to the accessory portion of the eleventh nerve. Motor fibres are derived from a special group of cells known as the nucleus ambiguous. From these groups of cells the fibres run outward through the formatio reticularis of the medulla to their superficial origins as described. In passing through the medulla these bundles of fibres traverse the ascending root of the fifth nerve,

and this fact furnishes an anatomical explanation for some of the reflex relations of the fifth nerve.

The spinal portion of the eleventh nerve rises from the upper cervical portion of the spinal cord, by a series of filaments from the anterior horn, and then takes a unique course upwards into the cranial cavity to join temporarily the accessory portion before again passing out of the cranial cavity to supply the trapezius and sterno-cleido-mastoid with motor fibres. It supplies these two muscles in conjunction with branches from some of the cervical nerves proper.

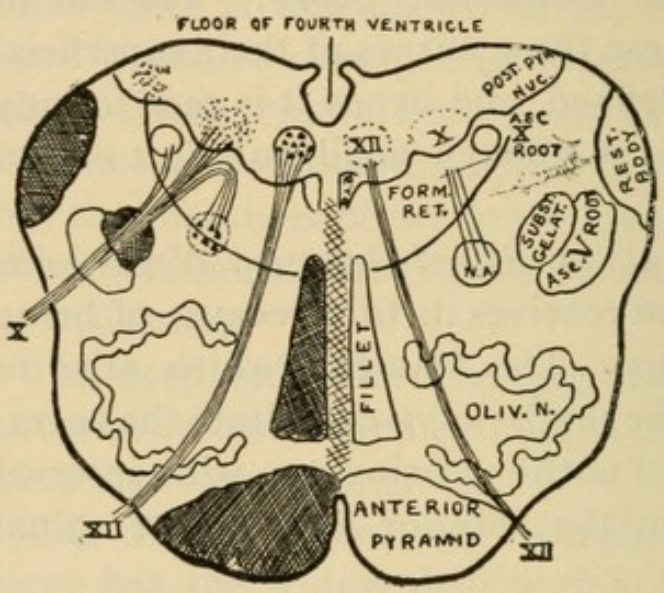


FIG. 49.

Diagram of a transverse section through the medulla at the lower level of the fourth ventricle. Especially to be noted are the tenth and twelfth nerves and their nuclei of origin, the position of the olivary nucleus, the fillet, the anterior pyramids (the motor path), the restiform body, and the ascending root of the fifth nerve. Between the olive in front and the tenth and twelfth nuclei behind lies the substantia reticularis (the path of common sensibility). The posterior longitudinal bundles lie on either side of the median line between the twelfth nerves.

The Twelfth or Hypoglossal Nerve.—This is a purely motor nerve destined for the muscles of the tongue and for most of the muscles connected with the hyoid bone. It arises from the medulla between the anterior pyramid and olivary body, by a series of filaments which are in line with the motor roots of the spinal nerves and immediately adjacent to the rootlets of the eleventh. The twelfth pair are separated at their superficial origin by the prominent anterior pyramids, and are rarely involved together by disease external to the medulla. The eleventh and

twelfth are so closely associated at their origin that they are frequently involved together, the result being paralysis of the tongue and vocal cord of the same side.

The nucleus of the hypoglossal nerve is a long column

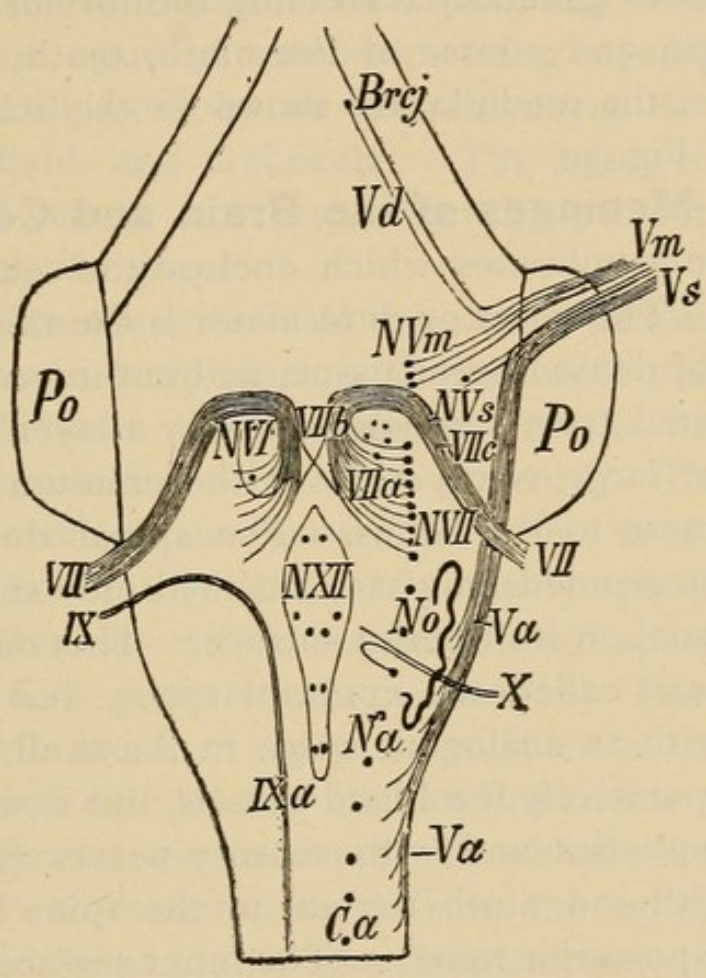


FIG. 50.

Schematic basal section of the medulla oblongata. Po, pons; Brcj, Brachium conjunctivum; Va, spinal, Vd, cerebral, Vm, motor, Vs, sensory root of the trigeminal nerve; NVm, motor, NVs, sensory root of the trigeminal nucleus; NVII, nucleus of the facial nerve; VII, a, b, c, root of the facial nerve; VII, exit of the facial nerve; NVI, abducens nucleus; NXII, hypoglossal nucleus; IXa, spinal root of the glosso-pharyngeal nerve (X); IX, its exit; No, olivary nucleus; X, vagus nerve (or glosso-pharyngeal) showing its part origin from the nucleus ambiguous; Na, nucleus ambiguous; Ca, anterior horn of the spinal cord; Ca, Na, NVII, NVm, column of origin of motor nuclei.

of motor nerve-cells extending from the level of the lower angle of the fourth ventricle (calamus scriptorius) downward to the decussation of the pyramids. It lies close to the median line, and in part of its course just medial

to the spinal-accessory nucleus. To reach the surface of the medulla, the fibres pass from the nucleus forward and outwards, through the formatio reticularis and between the olivary nucleus and the anterior pyramid, some of the fibres generally traversing the former. The relative origin and course of the ninth, tenth, and twelfth nerves in the medulla are shown in the schematic diagram of Fig. 50.

The Meninges of the Brain and Cord.—There are three membranes which enclose the central nervous system. The outer or dura mater is the thickest. It is formed of dense fibrous tissue, and in the vertebral canal is separated from the bony walls by a layer of fat and a plexus of large veins, while in the cranium it is closely approximate to the bones. The spinal dura is thus a loosely suspended long sac, attached only slightly to the spinal canal on its anterior surface. This outer space in the spine is called the epidural space, and is not connected with an analogous space in the skull. The dura has comparatively few blood-vessels, but is well supplied with lymphatics and with sensory nerves (in the skull by the fifth and ninth nerves, in the spine by filaments from the posterior roots). The inner surface is lined by endothelium and encloses the lymphatic subdural space, which contains a variable amount of serum. The middle membrane is the arachnoid, delicate and with few blood-vessels and no nerves, enclosing what is known as the lymphatic subarachnoid space. It is lined with endothelium on both surfaces. On the brain the arachnoid is separated from the innermost membrane or pia mater, between the convolutions, where the latter dips down into the sulci; but the separation between the two is complete in the spinal canal. There is no direct communication between the subdural and subarachnoid

lymph-spaces, but there is a free communication between the ventricles of the brain and the latter space by means of the foramen of Magendie and two smaller foramina in a part of the membrane projecting dorsally into the fourth ventricle. The base of the brain, therefore, lies upon a sort of water-bed, being separated from the base of the skull by an interval occupied by subarachnoid fluid and trabeculæ. The nerve-roots and basal blood-vessels are thus carefully protected from certain forms of injury.

The pia mater is a delicate membrane extremely rich in blood-vessels, lymphatics, and vasomotor nerves. It dips down between the convolutions of the brain, and sends connective-tissue septa into the cord. The blood-vessels and nerves pass inward from this membrane to the central nervous system, while the lymphatics communicate externally with the subarachnoid space.

The whitish granulations, known as Pacchionian bodies, which are seen upon the surface of the membranes on each side of the median line of the vertex of the brain, are connective-tissue villi which subserve the outflow of cerebro-spinal fluid from the subdural and subarachnoid spaces into the longitudinal and other dural sinuses.

The Blood-Supply of the Spinal Cord and Brain.—Branches from the vertebral, intercostal, and other arteries enter the spinal canal with the spinal nerves. Anterior branches pass along the anterior nerve-roots to the anterior median fissure, where they join to form a continuous vertical vessel—the anterior spinal artery. Branches pass to the bottom of the fissure and enter the cord, dividing into lateral twigs at the anterior commissure, which are destined for the anterior horn and partly for the lateral column. Ramifications

are made by both the anterior and posterior arteries in the pia mater, and twigs are given off everywhere to the white substance of the cord along the connective-tissue septa. The posterior arteries send branches into the caput cornu posterioris where they take vertical courses.

The veins have a very similar arrangement. Combining to form anterior and posterior spinal veins, these empty their blood into the large venous plexuses of the epidural space. These plexuses receive blood also from the vertebræ and even from the skin and subcutaneous structures of the back, finally delivering it to the vertebral and intercostal veins. The tortuous course of the arteries destined for the spinal cord and the large size of the venous plexuses undoubtedly protect those portions of the central nervous system from the dangers of high pressure, over-distension, and rupture.

The blood-supply of the brain is of far greater practical importance to the physician than that of the spinal cord. It is derived from the carotid and vertebral arteries. The courses of the left carotid and of the left vertebral are much nearer a direct line from the heart than those of their companions of the opposite side, a fact which renders them more liable to receive embolic plugs washed away from the endocardium.

On each side the internal carotid divides into the anterior and middle cerebral arteries, while the basilar artery, formed by the two vertebrals, redivides into the two posterior cerebral arteries. These three arteries, the anterior, middle, and posterior cerebral, supply each hemisphere in the region designated by their name. Practically the middle cerebral or Sylvian artery is of especial importance, for, coursing through the fissure of Sylvius, it is distributed over the motor area of the cortex. Hence the hemiplegia so often pro-

duced by embolism and by thrombosis of this artery. Although it has anastomoses with branches from the

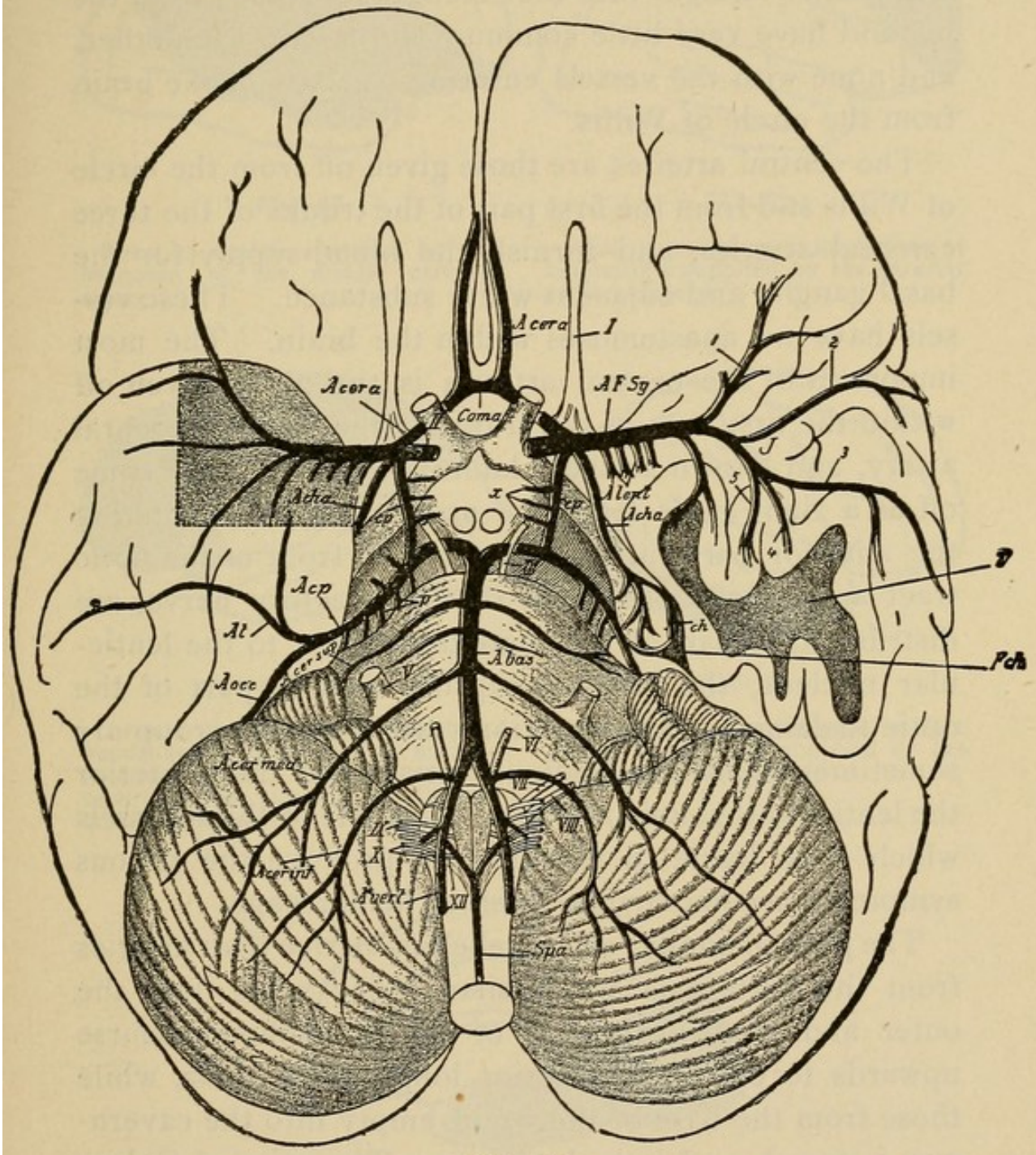


FIG. 51.

Arteries at the base of the brain. (Oppenheim.)

other arteries on the surface of the brain, they are rarely sufficient for the establishment of a collateral circulation.

The branches sent by these superficial vessels are short ones for the cortex, and long ones for the white matter. They enter straight into the cerebral substance from the pia and have very little communication with each other, and none with the vessels entering the base of the brain from the circle of Willis.

The central arteries are those given off from the circle of Willis and from the first part of the trunks of the three cerebral arteries, and furnish the blood-supply for the basal ganglia and adjacent white substance. These vessels have no anastomoses within the brain. The most important of the central arteries is the group given off within the first inch of the trunk of the middle cerebral artery, and known as the "lateral group." They come off at a right angle from the main artery, and entering the anterior perforated space, just in front of the optic tract and behind the roots of the olfactory nerve, are distributed to part of the caudate nucleus, to the lenticular nucleus, the internal capsule, and to part of the optic thalamus. The anterior vessels of this group are sometimes called the lenticulo-striate and the posterior the lenticulo-thalamic arteries. It is one of these vessels which is so liable to rupture and produce the serious symptoms accompanying cerebral hemorrhage.

The pons, medulla, and cerebellum derive their arteries from the vertebrals and basilar. The veins from the outer and median surfaces of the hemispheres course upwards to enter the superior longitudinal sinus, while those from the base of the brain empty into the cavernous, petrosal, and lateral sinuses. The veins of Galen, carrying blood from the choroid plexuses and walls of the ventricles and from the upper surface of the cerebellum, terminate in the straight sinus. There are free anastomoses between all of the sinuses, and there are

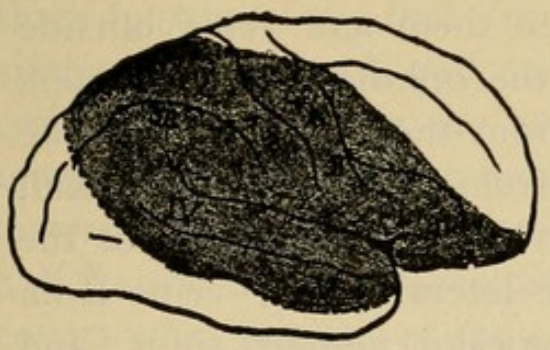


FIG. 52.

Area of cortex supplied by the branches of the middle cerebral artery.

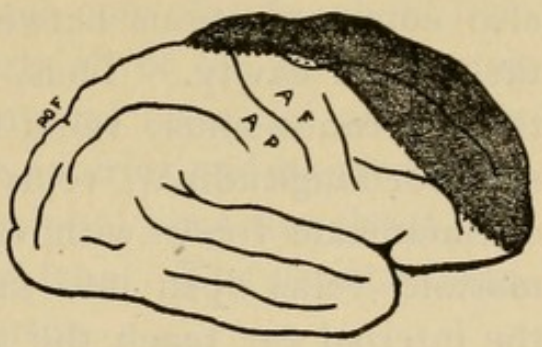


FIG. 53.

Area of cortex on outer surface of hemisphere supplied by the anterior cerebral artery.

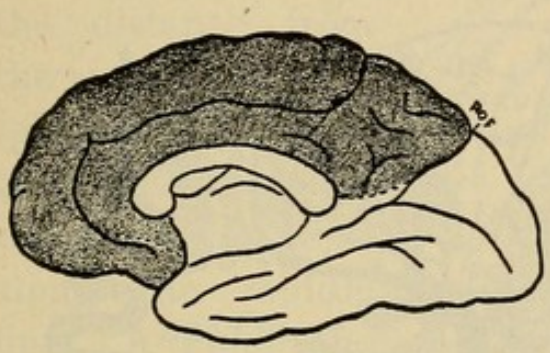


FIG. 54.

Area of cortex on inner surface of hemisphere supplied by the anterior cerebral artery. The dotted lines separate areas supplied by different branches of the artery.

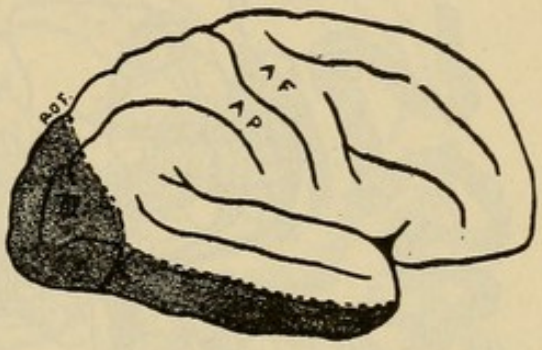


FIG. 55.

Area of cortex on the outer surface of the hemisphere supplied by the posterior cerebral artery.

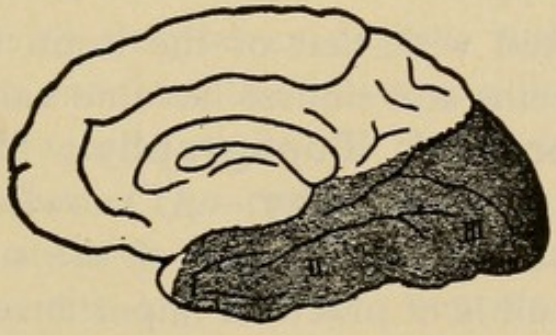


FIG. 56.

Area of cortex on the inner surface of the hemisphere supplied by the posterior cerebral artery. The dotted lines separate areas supplied by different branches of the artery

also communications between them and veins outside the cranial cavity. Thus, the ophthalmic vein enters the cavernous sinus; veins from the nose pass into the superior longitudinal; veins from the diploë of the skull communicate freely with various sinuses; some of the mastoid veins open into the lateral sinus; veins from the internal ear reach the superior petrosal sinus; and there are even numerous communications, some direct, others indirect, between veins of the face, scalp, and neck and the cerebral sinuses. The spinal venous sys-

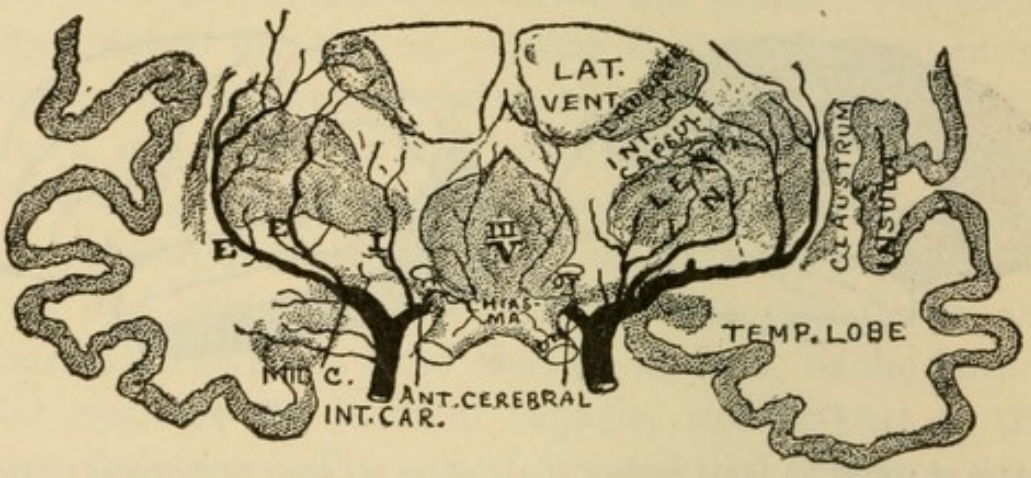


FIG. 57.

Diagram of the blood supply of the central ganglia and internal capsule. Mid. C., middle cerebral artery; E E, external, I, internal, lenticulo-striate arteries: III V, third ventricle. (After Duret.)

tem is connected with that of the brain by six veins. The cerebral veins and sinuses have no valves.

Cranio-Cerebral Topography. Spinal and Spinal-Cord Topography.—A knowledge of some of the relations of certain parts of the surface of the brain to the skull is of practical importance in diagnosis and for surgical purposes. The diagram on page 83 illustrates sufficiently well the general correspondence between the chief convolutions and fissures and the cranial bones. It will be noted that under the frontal bone lie

three fourths of the first frontal convolution, about five sixths of the second, and nearly all of the third frontal convolution. Under the temporal bone lies all of the temporal lobe, except its most anterior and its most posterior portion. Under the occipital bone lies about one half (posterior) of the occipital lobe. The parietal bone covers what remains of the cortex.

The fissure of Rolando is the part of the brain which it is the most often necessary to locate. The upper end of the fissure is best found by measuring the distance from the root of the nose (glabella) to the external occipital protuberance (inion) along the middle line. A point one half inch posterior to the middle point of this distance marks the upper extremity of the fissure. The fissure of Rolando in its upper two thirds makes an angle of sixty-seven degrees with the median line, in its lower one third the fissure is a little more vertical. The average length of the fissure is about three and a half inches. The central convolutions occupy about an inch anterior and posterior to the fissure, and the positions of the leg and face centres may thus be approximately located.

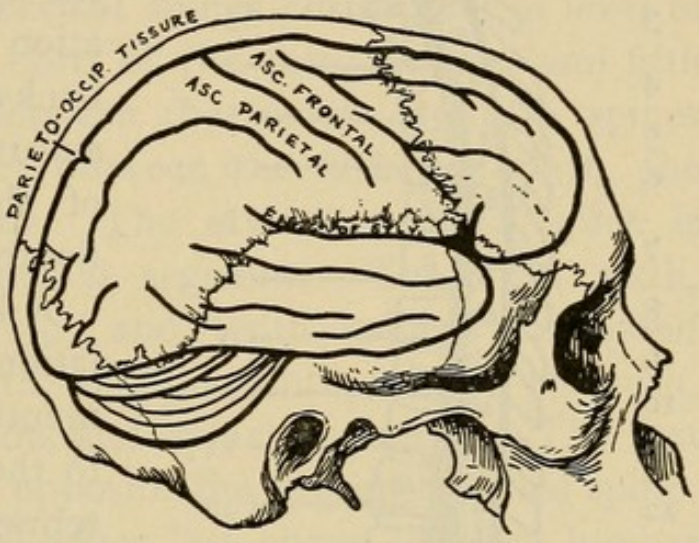


FIG. 58.

Diagram showing the relation of the convolutions to the skull.

What is known as the Sylvian line extends from the external angular process of the frontal bone to a point three quarters of an inch below the most prominent part

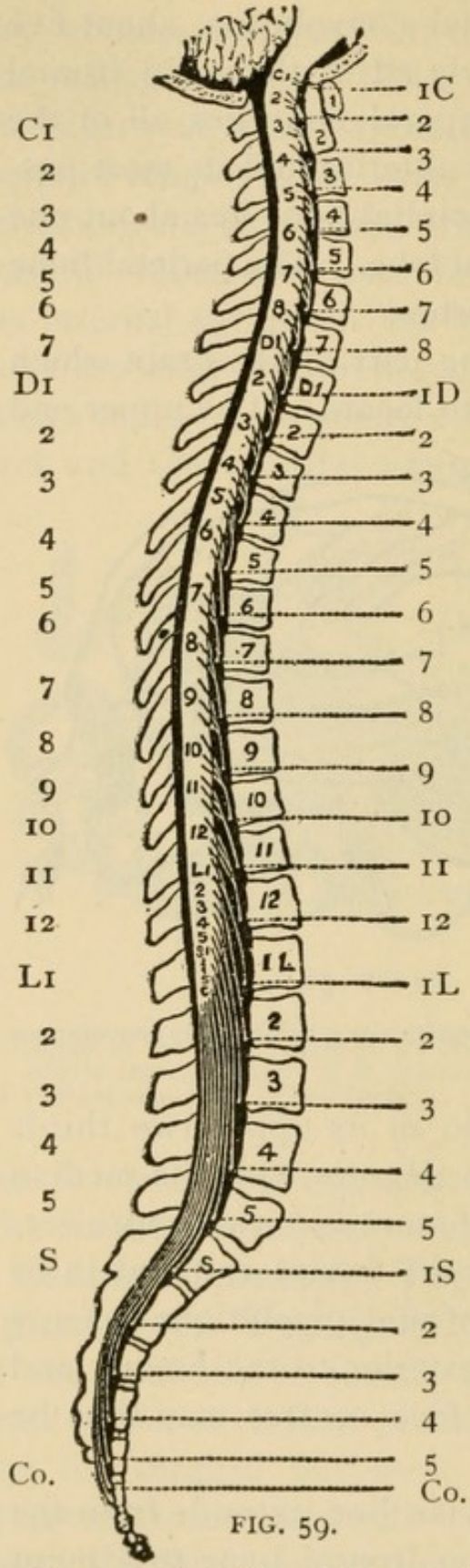


FIG. 59.

The mutual relations of the vertebral bodies and spines to the segments of the cord and to the exit of the nerves (Gowers).

of the parietal boss. The posterior limb of the fissure of Sylvius lies under the posterior three fifths of this line. The anterior limb of the fissure of Sylvius ascends almost vertically from the sylvian line at the middle of the zygoma. A continuation of the Sylvian line backwards to the sagittal suture marks the position of the parieto-occipital fissure.

A knowledge of the relations of the various segments of the spinal cord to the spines of the vertebræ is essential both for the diagnosis and successful treatment of certain spinal-cord affections. These relations vary somewhat in different individuals, but one description is sufficiently accurate for practical purposes. The chief facts may be deduced from the accompanying drawing. These facts are as follows:

(1) The spinal cord in the adult terminates at

about the lower border of the first lumbar vertebra (in children one to two years of age the cord extends as low as the third or fourth lumbar vertebra).

(2) The different pairs of nerves, excepting the upper cervical nerves, leave the vertebræ from which they are named at a lower level than that at which they are given off from the corresponding segments of the cord. This difference in the level of exit and origin of the nerve-roots increases from above downwards.[1] The first, second, and third cervical spines correspond in level to the origins from the cord of the third, fourth, and fifth cervical nerves. Between the fourth and fifth spines arises the sixth pair; between the fifth and sixth, the seventh cervical pair. The seventh cervical spine is opposite the first dorsal segment. The fifth, sixth, seventh, eighth, ninth, and tenth spines correspond nearly to the seventh, eighth, ninth, tenth, eleventh, and twelfth nerve-roots and spinal segments. The first lumbar nerve arises opposite the eleventh dorsal spine. The first sacral nerve arises just above the first lumbar spine.

(3) The cervical enlargement of the cord corresponds very closely to the cervical spines. The lumbar enlargement corresponds to the tenth, eleventh, and twelfth dorsal and to the first lumbar spines.

[1] It is convenient at times to know the exact lengths of certain of these nerves (especially the lower ones) between their origin and their exit from the spinal canal. It is easy to remember that the course of the third lumbar is about three inches, and that of the fifth lumbar about five inches.

CHAPTER II.

THE SYMPTOMATOLOGY OF NERVOUS DISEASES.

THE symptoms of organic disease of the nervous system, though exceedingly varied, can for the most part be referred to the operation of a small number of fundamental mechanisms. In the first place an important class of symptoms result from the destruction, complete or incomplete, of nervous structures.

Complete destruction of a portion of the nervous system always results in complete loss of function of the part involved. This loss of function is permanent except in cases where neighboring parts of the nervous system make up the loss, to some extent, by compensation. In adults extensive compensation can occur only in the brain, some functions of which are diffuse—that is, dependent on the activity of a large number of cells scattered over a wide area. Thus a destructive lesion in the frontal lobe of the brain, which is chiefly concerned with higher intellectual processes, does not cause any special loss of function, but simply a general lowering of its functional activity as a whole. The restoration of speech which occurs in children in whom the motor speech centre of the left side has been destroyed, is a good example of compensation, which, in this instance, occurs in corresponding structures of the opposite hemisphere. Compensation is limited, where, as in the cord and the medulla, and some parts of the brain, a particular function is per-

formed only by certain sharply limited structures, and these are destroyed.

Partial damage of nerve elements also gives rise to loss of function, which may be of any degree of severity. The following are the chief causes of structural damage to nerve elements :

1. Mechanical Injury.—Mechanical damage may cause destruction of cells and fibres, or interruptions in their continuity. Hemorrhage or trauma are the chief causes of such mechanical lesions. There is absolute loss of function of the parts thus injured.

An important form of mechanical injury to nervous structures is pressure. The severity of the effects of pressure depend largely on the rapidity of its production. A considerable degree of pressure, if of very gradual development, may give rise to little functional disturbance. Thus the entire motor area of the brain may be gently compressed by slow subdural or subarachnoid hemorrhage without giving rise to motor paralysis. Again, the spinal cord may be slowly compressed by Pott's disease to a surprising degree with little or no interference with the functions of the cord. A slight grade of pressure, on the contrary, if rapidly brought about, does far more damage to the nerve elements, and may cause immediate loss of function.

2. Loss of Blood Supply, Partial or Complete.—The loss of function is complete, if the blood supply is arrested ; partial, if there is merely diminution in blood supply. In both cases the effect comes on very rapidly if the interference with the circulation be sudden. Where the interference is gradual, the effects are of more gradual onset and of less severity, probably because there is time for the establishment of a collateral circulation.

Both cells and fibres suffer from diminished blood sup-

ply, but not equally. The nerve-cells are in general far more sensitive to the effects of deficient blood supply. For example, if the blood supply to the spinal cord of the rabbit be cut off by compression of the abdominal aorta, the motor ganglion-cells cease to functionate at once, but the white matter shows no reaction at first. If the circulation is restored after half an hour, the cells recover function : if not, necrotic changes begin, first in the cells, later in the white matter.

3. **Inflammation,** which involves the nerve and fibres (parenchyma), the blood-vessels, and usually the interstitial tissue. The process may be acute or chronic ; in the latter case the changes begin in interstitial structures, and secondarily damage the nervous elements.

4. **Wasting of Nerve Elements.**—A very frequent cause of the destruction of nervous tissues is the degeneration which affects cells and fibres. Often degenerative changes occur in these structures as a consequence of inflammation of interstitial tissues, but in a large and important class of cases the changes in the cells and fibres are primary. Many symptoms however of nervous disease, result not from destruction of nerve elements but from their irritation. This irritation usually expresses itself by an increase of action, or functional overaction, as, for example, in convulsive movements, or hyperæsthesia.

But irritation does not always result in overaction ; it may lessen function by the process termed inhibition. A good example of this effect of irritative inhibition is the loss of the knee-jerk which follows immediately on a severe mechanical injury to the spinal cord above the lumbar enlargement, say in the cervical region. The important difference between the diminution or loss of function which results from irritative inhibition and that which results from a destructive lesion, is that the former

is temporary, the latter permanent. It is important to remember that the effects of destruction and those of irritation are often met with in combination, as for example when a tumor causes both paralysis and spasm.

The mechanisms which have been mentioned as productive of symptoms relate mainly to organic diseases. There is a large and important class of symptoms which cannot be referred to structural changes in the nervous elements but which probably depend upon changes in their nutrition, changes of whose nature we at present know little.

Motor Paralysis. — Loss of voluntary muscular power (paralysis) may result from disease involving any part of the motor path—its upper segment or its lower segment (p. 24). Thus, paralysis may arise from disease of the motor cortex, of the motor path in the centrum semiovale, internal capsule, crus, pons, medulla, or cord (upper segment), or from disease of the anterior cornual cells, anterior nerve-roots, motor nerves, or muscle plates (lower segment).

Disease in other parts of the nervous system than those enumerated does not cause motor paralysis, unless it does so by giving rise to pressure on these structures.

Paralysis may or may not be associated with wasting or atrophy of the muscles paralyzed, according to the position of the lesion.

In general it may be said that paralysis and marked atrophy are rarely associated in disease involving the cerebro-spinal or upper segment of the motor path (p. 24), and that they are nearly always associated in disease of any part of the lower segment (ganglion-cells, nerve-roots, or nerves).

The varieties of paralysis according to distribution are without number, but certain typical forms are of

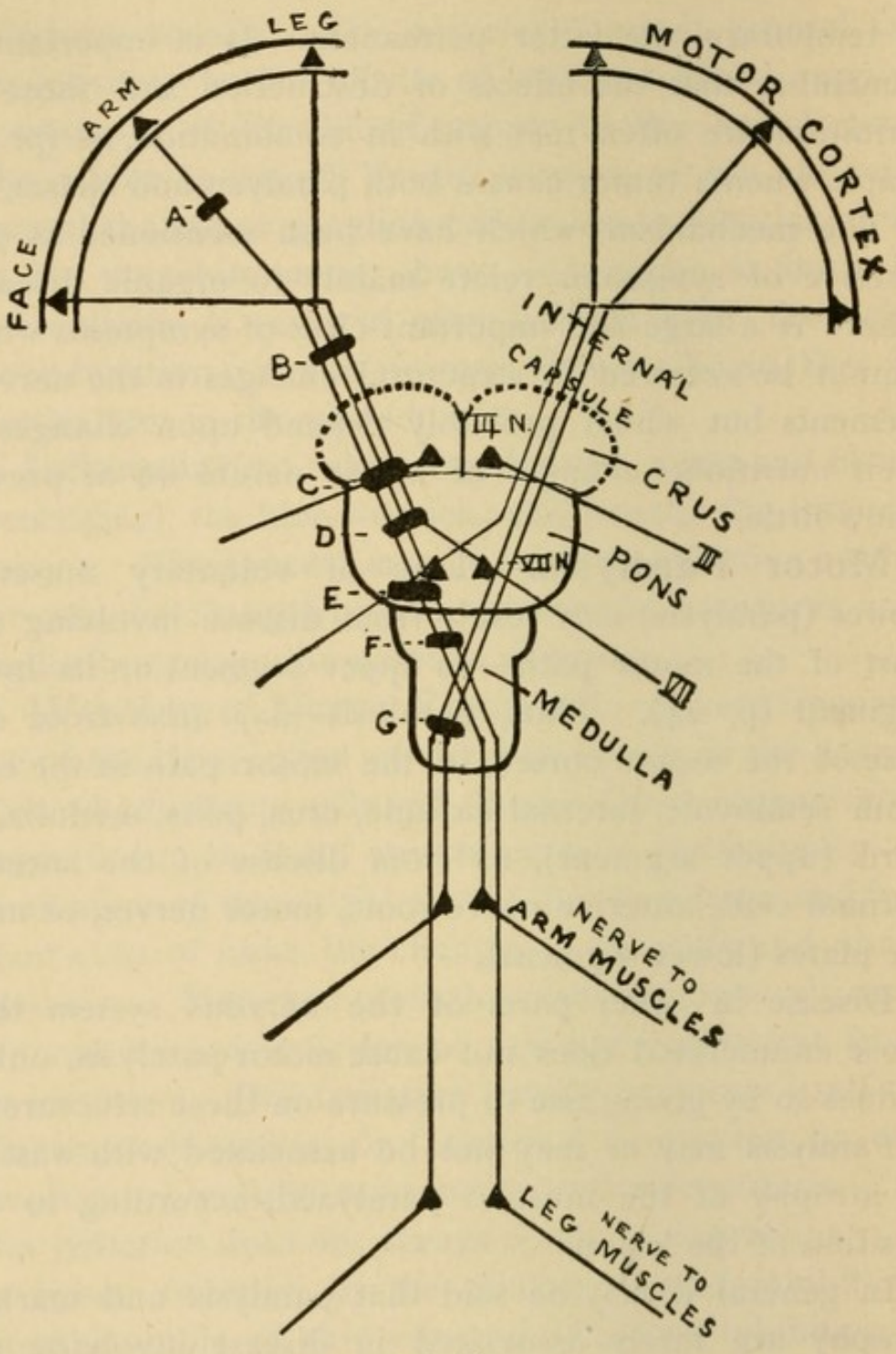


FIG. 60.

Diagram showing the effect of lesions at different levels of the motor path. A small lesion at some point, A, between the cortex aud the internal capsule, will cause a local paralysis—a lesion at A would cause a brachial monoplegia; a lesion at B will produce hemiplegia (complete) on the side opposite the lesion; a lesion at C would cause paralysis of the IIId nerve on the *same* side, and of the face, arm. and leg on the *opposite* side; a lesion at D would cause hemiplegia on the opposite side; a lesion at E would cause paralysis of the face on the *same* side, of the arm and leg on the opposite side; a lesion at F, paralysis of arm and leg on the opposite side (and perhaps of the XIIth nerve on the same side—XIIth nerve not represented); a lesion at G would cause paralysis of the arm and leg on the same side.

practical importance from their diagnostic significance and frequent occurrence.

Hemiplegia.—Hemiplegia is the paralysis of several groups of muscles of one lateral half of the body. In its more common form there is a more or less marked loss of voluntary motion involving face, tongue, arm, and leg on the same side—complete hemiplegia. Such hemiplegia may result from a lesion *anywhere in the motor tract between the cortex and the pons.* In the pons and crus the fibres of the motor tract are so close together that a lesion practically always involves all the fibres of the tract, and complete hemiplegia results. In the internal capsule the fibres begin to spread out, and a lesion here, if very small, may fail to involve all its fibres, thus giving rise to an incomplete hemiplegia. While this is theoretically possible it is practically of rare occurrence, and a more or less complete hemiplegia in range is the rule. The motor fibres are more and more widely separated in their course through the corona radiata of the cortex. Hence, in these situations even a lesion of considerable size may fail to affect all the parts of one side, face, tongue, arm, and leg. Thus it is not rare for the face and leg to escape more or less completely, the arm being paralyzed alone. Such a paralysis is called brachial monoplegia. Other monoplegias, as face and arm, arm and leg, but not face and leg, may occur from single lesions.

The expression "complete hemiplegia" implies that the paralysis affects all of one side, but this is not strictly true. Thus the upper facial muscles (orbicularis palpebrarum and occipito-frontalis) move almost or quite as well as on the non-paralyzed side, and the muscles of the eyeball and the muscles of mastication are unaffected. The tongue as a whole deviates towards the paralyzed

side when protruded because the projecting apparatus on that side is weakened and does not oppose the genio-hyo-glossus. Inasmuch as this muscle also retracts as well as protrudes the tongue, the organ is not so high on the paralyzed side when in repose. The muscles of respiration and the muscles of the face and trunk are not involved in ordinary action. The movements of the superior intercostal muscles are greater on the paralyzed side, though if the patient take a deep respiration there is defective chest expansion of the affected side.

In general it may be said that in hemiplegia the extent

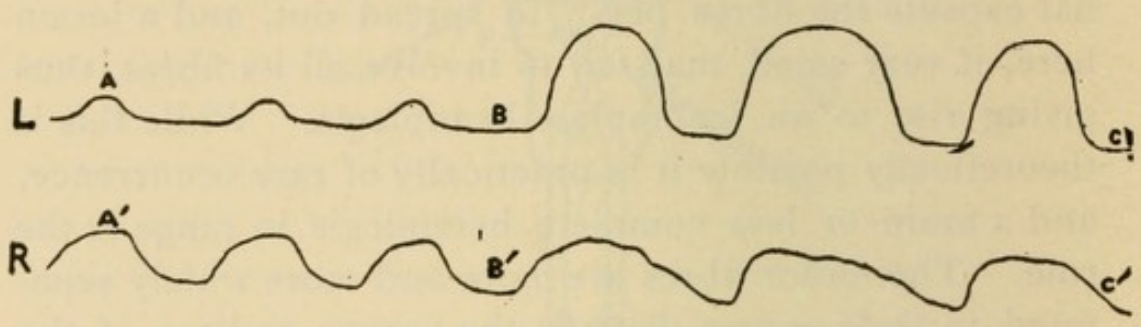


FIG. 61.

Respiratory movements in a case of right hemiplegia. R, right side ; L, left side ; A B and A′ B′, normal respiration ; B C and B′ C′, forced respiration.

of the paralysis in the part is proportional to the degree in which the part is capable of unilateral use. Thus the muscles of one hand are habitually employed alone, are chiefly innervated from one side of the brain, and suffer severely, while the intercostals of one side are never used by themselves, are innervated from both hemispheres, although not quite equally. The leg, which, though generally used with its fellow, is capable of some unilateral use and is also represented to some extent in both hemispheres, suffers less, and usually recovers more power than the arm. This recovery is through compensation by the normal hemi-

sphere, whose nerve impulses probably travel over other than pyramidal tracts. In early life the amount of recovery is usually extensive. Recovery is more rapid in the arms and thighs than in the hands and feet. The hemiplegia is always on the side opposite to the lesion. This is explained by the crossing of the pyramids in the medulla. A few exceptions to this rule are met with, and often the parts on the same side of the lesion are slightly weakened, due to an incomplete crossing of the pyramids and the persistence of a larger bundle in the direct pyramidal tracts.

Double hemiplegia or diplegia is a paralysis of all four extremities, and may result from two extensive lesions or a single diffuse lesion covering both hemispheres, as from meningeal hemorrhage in birth palsy.

Common hemiplegia, such as has been described, is not often accompanied with hemianæsthesia, but it may be associated with partial loss of tactile sensibility in the extremities for a time, if the lesion involve the sensory tracts lying far back in the capsule, or when the optic thalamus is injured.

The processes which most frequently give rise to hemiplegia are cerebral hemorrhage from arterial rupture, thrombosis, acute softening, embolism, tumor, and abscess.

Instead of the face, arm, and leg being paralyzed on the same side of the body, it is not very rare for the face to be paralyzed on one side and the limbs on the other. The condition is known as crossed hemiplegia. It occurs when the lesion is so placed that it interrupts the motor path to the limbs *above its decussation* in the medulla, and the downward facial path of the same side after it has crossed the median line in the pons from the opposite facial tract. The lesions must be below the

middle of the pons, as the crossing of the facial path occurs just above. (See Fig. 62.)

This form of paralysis is crossed hemiplegia in the restricted sense of that term. Taken in a wider sense the term refers also to the paralysis of one or more of the other cranial nerves on one side with paralysis of the extremities of the opposite side. The nerves most frequently involved in such a lesion are the third, sixth, and seventh. If the third nerve is paralyzed on one side and the face and limbs on the other the lesion is in the crus or presses on it on the side of the nerve, and this is the only situation in which a lesion can cause such a paralysis. In such cases the face paralysis has the usual character of a complete peripheral facial palsy; involvement of upper as well as lower muscles of the face, R. D., etc. (See Fig. 62 for explanation of these anatomic facts in paralysis.)

Hemiplegia from a disease in such region is by no means always of the crossed type, since there may be only a slight weakening of some one or more cranial nerves, the lesion being so small or situated in the motor tract only.

Spinal Hemiplegia or Brown-Séquard's Paralysis is a type of hemiplegia that results from lesions interrupting the motor path of one half of the cord. Both arm and leg are involved when the lesion is high in the cord; the paralysis of the leg may be incomplete, while that of the arm is complete, as fibres for the leg cross lower down in the cord. The lesion which causes spinal hemiplegia is seldom confined to the motor tract or even to one side of the cord. Hence there is not only anæsthesia on the side *opposite* the lesion, but there is commonly some weakening of that side also. The anæsthesia never extends quite to the level of the lesion, as the sensory

fibres cross in the cord a little above their level of entrance. The symptoms on the side of the lesion are: Motor palsy, slight hyperæsthesia of skin, impairment of muscular sense, reflex action at first lessened and then increased, and the temperature slightly raised. The symptoms on the side opposite the lesion are: Muscular power nearly or quite normal, loss of skin sensibility, temperature same as that above the lesion. The typical symptoms are less definite as the lesion approximates the lumbar enlargement. A lesion on one side of the lumbar enlargement often affects sensation on the same side as motion because it damages the sensory path before it has crossed. In all cases of crossed motor and sensory paralysis the sensibility of the muscles differs from the other forms of sensibility. And if muscular sensibility is affected on one side this is the side of the motor palsy and not of the cutaneous anæsthesia.

Paraplegia is a term used generally to designate a paralysis of both lower extremities. When all parts below the head are paralyzed the condition is termed cervical paraplegia. The intercostal muscles are involved and respiration is diaphragmatic. Anæsthesia may or may not accompany paraplegia, and there may or may not be involvement of the bladder and rectum. The conditions which determine the absence or presence of these symptoms will be discussed elsewhere.

Paraplegia is most frequently a symptom of spinal-cord disease, but may result from disease elsewhere. In the pons and medulla the motor tracts are close together, and it sometimes happens that a lesion there has sufficient transverse extent to involve the fibres of both sides (such as tumors), when paraplegia results. More rarely bilateral involvement of the crura leads to a similar result. Occasionally paraplegia results from bilateral

disease of the motor area of the cortex, as that seen in spastic paraplegia of infants. It has been already alluded to as double hemiplegia.

Paraplegia is a rather common occurrence in peripheral nerve affections, as in alcoholic multiple neuritis. Compression of the cauda equina by tumor or fracture ordinarily gives rise to it.

But paraplegia is not always referable to organic disease. There is a form known as hysterical paraplegia, which is a functional affection. A functional paraplegia from premature senile changes in the arteries to the lower part of the cord and paraplegia in chlorotic women sometimes develops independently of hysteria.

Monoplegia signifies paralysis of one limb or of one side of the face, and, according to the part involved, the paralysis is termed a brachial monoplegia, a crural monoplegia, or a facial monoplegia. The lesion may be cerebral, spinal, or peripheral.

There are certain general facts about the distribution of paralysis which aid one in determining whether a palsy be of peripheral, spinal, or cerebral origin. If the paralysis be confined to a single muscle or group of muscles known to be innervated by a single peripheral nerve branch the paralysis is unquestionably peripheral. If groups of muscles having a certain function in common are involved, as for example the dorsal flexors or adductors of the foot, the lesion is probably in the anterior horn of the spinal cord, as muscle groups related to particular functions are represented in the cord. When paralysis involves all the muscles of an arm or leg, that is of a segment of the body, the seat of the lesion is probably the upper portion of the motor path of the brain, for here movements of entire segments of an extremity are represented rather than of individual

muscles or groups of muscles. While these general rules are often insufficient in themselves to establish the location of the lesion they should always be taken into consideration.

It is to be noted that paralysis when due to lesions of the central nervous system is usually referable to disease of a definite region. This is especially true of localized paralysis, which is consequently often spoken of as a focal symptom in distinction to symptoms which, like general convulsions, point to an extensive disturbance of function and are hence called diffuse. While this distinction between focal and diffuse symptoms is useful it cannot always be made in practice. For example, some symptoms may be signs of focal disease at one time and at another of diffuse disease. This is the case with headache and with optic neuritis.

The designation paralysis is correctly applied to all grades of motor loss from slight weakness to more or less complete abolition of function. Sometimes the term "paresis" is applied to slight grades of weakness. The difference is purely one of degree, and the latter term should not be used, as it is often employed in another sense to designate a form of mental disease. The terms complete and incomplete are much the better expressions to qualify the degree of paralysis present in any given case under description.

Convulsions.—By this term is generally understood involuntary, paroxysmal, purposeless, muscular contraction, of variable intensity and duration and of extensive or limited distribution.

Convulsions may occur in consequence of disease situated in the cerebrum, basal ganglia, pons, medulla, or spinal cord, but are most often seen in cerebral disease, and their relation to such disease is of especial

importance. Convulsions may occur from disease of any part of the nervous system where there is an aggregation of motor cells forming part of a reflex arc.

They may arise (1) as a result of active irritation of brain tissue, particularly of the cortex, such as occurs in meningitis or meningo-encephalitis and active brain tumors; (2) in consequence of more or less stationary, non-active lesions, in which some nerve-cells are still able to generate nerve force, but do not possess the power of perfectly controlling their discharge, as in epileptic convulsions following infantile cerebral hemiplegia; (3) from spontaneous discharge of nerve force without a demonstrable gross lesion or irritation in the cortex, as in idiopathic epilepsy; (4) from discharge of ponto-bulbar centres or from centres in the cord itself, as in myelitis and polio-myelitis resulting from an acute morbid blood state.

Convulsions may be local or partial in their commencement, that is confined to particular groups of muscles or to one muscle of an extremity, and subsequently become general; or they may remain localized or be general from the beginning.

General convulsions occur in organic disease of the brain and from a large number of conditions external to and within the nervous system, which, when the ganglion-cells of the cerebral cortex are in a state of unstable equilibrium from defective nutrition or from other causes such as toxic and autoxic agents, seem capable of determining a discharge of nerve force from them, though they may be the seat of no gross structural change. Thus general convulsions are very commonly observed in idiopathic epilepsy, in uræmia, and especially in children at dentition, and at the commencement of febrile diseases, from gastro-intestinal disturbances, etc. It is

an important fact that a very large majority of all the cases in which general convulsions occur are *not* examples of gross organic disease of the brain.

In organic disease of the brain convulsions are usually general when the lesion is a diffuse one such as meningo-encephalitis and not circumscribed in the motor area of the cortex or the subjacent white matter. On the other hand, convulsions that are local in their commencement or remain limited in extent always indicate disease in or adjacent to the motor area of the cortex. The disease is usually structural in character, but in a few instances no structural change can be detected to account for the local beginning, and here there are probably nutritional changes that give rise to a pathological local instability. In other words, localized convulsions constitute a focal symptom, general convulsions a diffuse symptom.

The general convulsions from organic disease of the brain bear a close resemblance to the convulsions of idiopathic epilepsy, that is to say, there is usually a period of tonic followed by clonic spasm, during which the patient becomes rapidly or suddenly unconscious. A focal beginning in a general convulsion, however short, probably indicates an organic disease of the brain, if uræmia can be excluded.

In local or partial convulsions the local onset is referable to the nervous discharge commencing at the seat of irritation. Thus the spasm begins in one side of the face from irritation of the facial centre and in the arm or leg from the irritation of their respective centres. If a convulsion is very slight it may be limited to the muscles in which it begins, and there may be no perceptible loss of consciousness.

If of greater severity the spasm may extend gradually or rapidly to other parts of the side of the body on which

it begins. The order of its extension is usually from the free end of an extremity toward the trunk, involving an entire segment or extremity before extending to other parts of the same side.

In such cases consciousness is usually lost as soon as the convulsion is well advanced. The degree of loss of consciousness and the length of time consciousness remains absent is proportional to the rapidity and completeness of nerve discharge from the cortical centre. If the discharge of nerve force is of the highest grade of intensity the convulsions may involve both sides of the body at the same time and in such cases loss of consciousness is usually complete from the start.

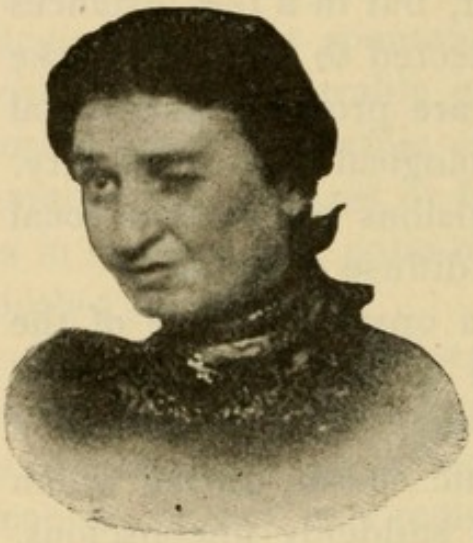


FIG. 62.

Localized spasm of the left side of the face in a patient suffering from Jacksonian epilepsy. (Starr.)

Strictly speaking it is probable that no convulsions are general from the very commencement. For example, even in infantile eclampsia and uræmic convulsions it is very common for the spasm to begin in one extremity a few seconds before it passes to the remaining half of the body or becomes general. True local convulsions, however, repeatedly commence in one extremity, and if they do not remain local at least continue so for an appreciable time.

It is not uncommon for a warning or aura to immediately precede a convulsive attack. The aura is always sensory in character and often begins in the part which is afterwards the seat of convulsion, and this is particularly true if there be a general convulsion with a local onset. The sensation may ascend the limb first in-

volved, and even pass to the second extremity affected, before the convulsion comes on. Such sensory auræ undoubtedly result from a nervous discharge from sensory cells which precedes that which occurs from motor nerve-cells, the functional control of which is one of their chief functions. The motor area of the cortex contains sensory as well as motor cells, and the presence of a sensory aura before the spasm indicates that the convulsion has its initiative in sensory elements, that is; cerebral convulsions are a sensory-motor reflex and are largely sensory in origin with a resultant motor expression.

Auræ of the special senses, as a flash of light, an odor, or a sound, are not uncommon in idiopathic epilepsy, but they are of relatively rare occurrence in organic disease of the brain. When they do occur they indicate that the lesion has its maximum intensity at or near the special sense centre corresponding to the aura—in the occipital lobe when there is a visual aura, etc.

Slight weakness in the part or parts involved is usual after a local or partial convulsion. If the convulsions recur frequently at short intervals the weakness may be very decided. In most cases it soon passes away, but in rare instances permanent paralysis may result. In such cases the convulsions have probably given rise to a destructive lesion by hemorrhage. If the convulsions happen to involve the extremities of one side of the body the weakness is, as might be expected, hemiplegic, but it is readily distinguished from hemiplegia by wearing off in two or three days or earlier. The weakness following convulsions is called post-convulsive or exhaustion paralysis. It is explained on the supposition that a severe convulsive seizure exhausts the motor nerve-cells related to the part. The resultant paralysis is greatest

in those particular parts which are most actively engaged in the preceding convulsions. Exhaustion paralysis occurs most frequently in those cases suffering from a previous incomplete organic hemiplegia of children in which epileptic convulsions have been superinduced in later life. The general exhaustion seen after general convulsions is probably analogous in cause and effect to the weakness which occurs in certain parts following local convulsion.

As to the diagnostic significance of local convulsions it may be said that this is the same whether the convulsions remain localized or subsequently become unilateral or general. They do not quite prove the presence of structural disease because in rare instances idiopathic epilepsy, uræmic convulsions, and diabetic convulsions may begin locally. But every case where convulsions begin locally should excite suspicion of organic disease and should lead to a minute and careful search for it. It is also to be noted that the significance of repeated local convulsion is not diminished by the occurrence of general convulsions at other times.

Convulsions which are general from the commencement, on the contrary, point to idiopathic epilepsy. But they do not exclude the possibility of structural disease, because general convulsions are not uncommonly symptoms of organic diseases of the brain. In such cases the distinction must be made by establishing the presence or absence of other symptoms of organic disease. It cannot be made from the character of convulsions alone.

General convulsions, besides being met with in organic diseases of the brain and spinal cord, are observed in idiopathic epilepsy, tetanus, uræmia, diabetes, and hysteria. It is not very uncommon for hysterical convulsions to occur in the course of organic disease of the

brain, and this sometimes leads to errors in diagnosis. Such convulsions not rarely result in the course of intracranial tumors; occasionally they are seen in meningitis.

Localized convulsions (monospasm, hemispasm) are much more frequently met with in intracranial tumor than in any other form of disease. The neoplasm causing them is usually situated very near the motor cortex and usually quite superficially. When the tumor is situated at a considerable distance from the motor area the local convulsions are probably the result of meningitis over the cortex.

Convulsions are frequently classified on a purely mechanical basis according as the tonic or clonic element predominates. Clonic spasm does not necessarily differ from tonic convulsion in its origin or essential character, but the impulses in the nerve discharge are infrequent enough to permit the muscular apparatus a temporary intermittent relaxation. A tonic or tetanic convulsion is one in which the muscular contraction continues for an appreciable but short period of time. Such spasm is met with in many conditions, for example at the onset of a grand mal epileptic paroxysm, in some cases of petit mal, in major hysteria, and in tetanus. Tetanic seizures lasting for hours or even days sometimes result from disease involving the gray matter in the floor of the fourth ventricle and perhaps from cerebellar disease and secondary pressure on the cruri. From their resemblance to tetanus the convulsions are often designated tetanoid.

Purely tonic spasm (Trousseau's symptom) may be excited by pressure on the larger nerves and arteries, as in laryngismus stridulus. The mechanical and electrical irritability of the peripheral nerves is increased during the intervals.

Localized tonic spasm is sometimes a symptom of considerable importance. It is seen as trismus (spasm of the muscles of mastication), in tetanus (early symptom), in some cases of hysteria, and sometimes in meningitis. Tonic spasm of the calf muscles (cramp) is observed in hysterical, neurasthenic, and alcoholic subjects as a consequence of over-use of these muscles. Another form of tonic spasm is that of the sterno-cleido-mastoid, causing "wry-neck."

When tonic spasm is of considerable duration or of gradual onset, it is perhaps more properly spoken of as rigidity, and will be considered under this title (p. 92), although it must be understood that a sharp distinction cannot always be made.

Clonic convulsions are characterized by the rapidly intermittent character of the muscular contractions which produce them. They are distinguished from tremor by being usually wider in range, less rhythmical in character, and of shorter duration; but here again the distinction between the two kinds of spasm is not always absolute.

Clonic convulsions occur in various forms in a great variety of morbid conditions of the nervous system, alone, or with those that are tonic. In the condition known as paramyoclonus multiplex we sometimes have examples of purely clonic convulsions. In this disease there is rapidly repeated clonic spasm occurring at varying intervals. The spasms vary from 50 to 180 per minute, and involve several muscles of a physiological group at once, thus giving the appearance of purposive action.

The term "internal convulsions" has been applied, not inaptly, to conditions of muscular spasm in internal organs. Thus, the laryngeal spasm of rickety children, known as laryngismus stridulus, may be regarded as analogous to the general convulsions which sometimes occur in these

same subjects. The spasm is thought to result from discharges from lower nervous centres in the medulla.

Tremor.—Tremor or trembling may be defined as a rapid involuntary to-and-fro movement of a part resulting from the alternate and usually rhythmical contraction of antagonistic muscles. Tremor in the strict sense of the word is always an oscillation of narrow range. It may or may not be present when the part in which it occurs is at rest—most tremors are not then present. The tremor of paralysis agitans usually continues during rest, and the tremor which results from cold and from emotion originates when the body is at rest. The majority of tremors continue during voluntary motion and are increased by the extra muscular tension. The tremor of paralysis agitans may be inhibited at will by voluntary effort for a short time, but it soon recommences.

The term "intention tremor," borrowed from the Germans, is used to designate tremors which occur only during voluntary movement causing increased muscle tension. It is more accurately named action tremor. It is frequently seen in disseminated sclerosis and in mercurial poisoning. It is often wide in range, irregular in rhythm, and ataxic in character.

Roughly speaking, the extent of a tremor is inversely proportionate to its rapidity, but the same tremor may vary somewhat in range at different times. There is considerable difference in the rapidity of different kinds of tremor, and these differences are sufficiently constant to be of some diagnostic value. For instance, the tremor of alcoholism is rapid and varies from seven to eight, and that of paralysis agitans from four to seven per second. Tremor is a symptom in a variety of diseases of the nervous system. It is sometimes the result of organic disease, but is commonly met with in diseases of

a nutritional character in which no gross structural changes in the nervous elements have been detected. Thus it is seen on the one hand in disseminated sclerosis after hemiplegia, in senile dementia, and certain forms of toxic neuritis; and, on the other hand, after exhausting diseases, in neurasthenia, paralysis agitans, hysteria, and Basedow's disease. Very frequently tremor is a toxic symptom, and as such it is seen in alcoholism, in poisoning from lead, mercury, opium, cocaine, and after the excessive use of tea, coffee, tobacco, and quinine.

But tremor is also encountered in cases where no assignable exciting cause can be detected and in which there is no associated abnormal state. It is sometimes not only congenital but also hereditary, and may be present throughout a lifetime without any other nervous affection being present. It may affect several members in a family through several generations. Sometimes this simple tremor, as it is called, seems to be related to emotion or to prolonged anxiety, and not infrequently a neuropathic heredity can be traced. Simple tremor neither shortens life nor influences the general health. Senile tremor occurs in extreme old age or in early senility, and while it does not exhibit the same rigidity and muscular weakness observed in paralysis agitans it is doubtless closely associated and probably dependent upon an allied structural change in the nerve elements. Most tremors cease during sleep; rarely a tremor is diminished or unaffected. The upper extremities are most frequently the seat of tremor, and the head is more involved than the leg.

With regard to the pathology of tremor nothing definite can be said. We know that when a voluntary movement is made every contraction of one set of muscles is accompanied by a corresponding relaxation of

antagonistic muscles. This arrangement serves to make motion even and regular and is doubtless subserved by a close anatomical association in corresponding motor centres. A continuous voluntary muscle contraction probably consists of a series of single contractions following each other at very short intervals, due to innervating impulses from the cerebral cortex, which are liberated at the rate of about ten or twelve per second, according to the position in which they are generated. So long as these impulses follow one another rhythmically, and simultaneously reach the antagonistic muscles involved in a voluntary movement, the motion is a continuous one. It is conceivable that the nice mechanism in which these simultaneous liberations of nerve force occur may be readily deranged to a slight degree by nutritional disease which is far short of being a gross structural change and that the parallelism of the innervating rhythm in opposed muscles would suffer slight derangement. The opposing muscles would then act, not simultaneously, but alternately, and the expression of such alternation would be tremor. But the disturbance of the cortical gray matter in the brain in which motor impulses are controlled and generated is probably not the only manner in which tremor may arise. Structural changes in the nerves which conduct voluntary impulses may damage the axis-cylinders enough to modify without destroying their conductivity and it is probable that some forms of tremor, as that of disseminated sclerosis and of neuritis, depend on such interference with conduction. The majority of tremors, however, undoubtedly depend on nutritional alterations and less commonly on structural changes in sensory inhibiting cells in the cerebral cortex. There is good reason to believe—although the evidence is not conclusive

—that in paralysis agitans, dementia paralytica, hysteria, fear, and in alcoholism the tremor is of cortical origin. Inasmuch as the pathology of tremor is still obscure its diagnostic significance is somewhat vague. Roughly speaking tremor is to be regarded as one evidence of nutritional derangement of functions of the sensory-motor cortex, which may or may not present gross structural changes. More cannot be said. In order to determine the location of the lesion and whether the change is simply one of nutrition and not one of gross structural change, the conditions associated with the tremor must be interrogated. Not infrequently these points cannot be decided. Myographic tracings of various forms of tremors are shown in Fig. 63.

Fibrillation.—Fibrillary twitchings or fibrillations are involuntary contractions of small numbers of muscle-fibres. As they give rise to no movements of the parts in which they occur, they are recognized only when occurring in muscle-fibres which lie just beneath the skin. The contraction appears as a small wave-like elevation running rapidly along the muscle in which it occurs. It may be confined to a part of one muscle or may be seen in all the muscles of an extremity. It is chiefly met with in the extremities, face, and tongue. The contractions may be almost constant or may be separated by considerable intervals. It is often easy to produce them at will by cooling or tapping the skin over the muscles. In those conditions giving rise to fibrillation the mechanical irritability of the muscle-fibres is increased. Fibrillary tremors occur under two distinctly different clinical conditions—as the expression of general, slight, functional derangement of the nervous system and as the result of a degenerative process in the muscle-fibres. Thus it is met with in neurasthenic

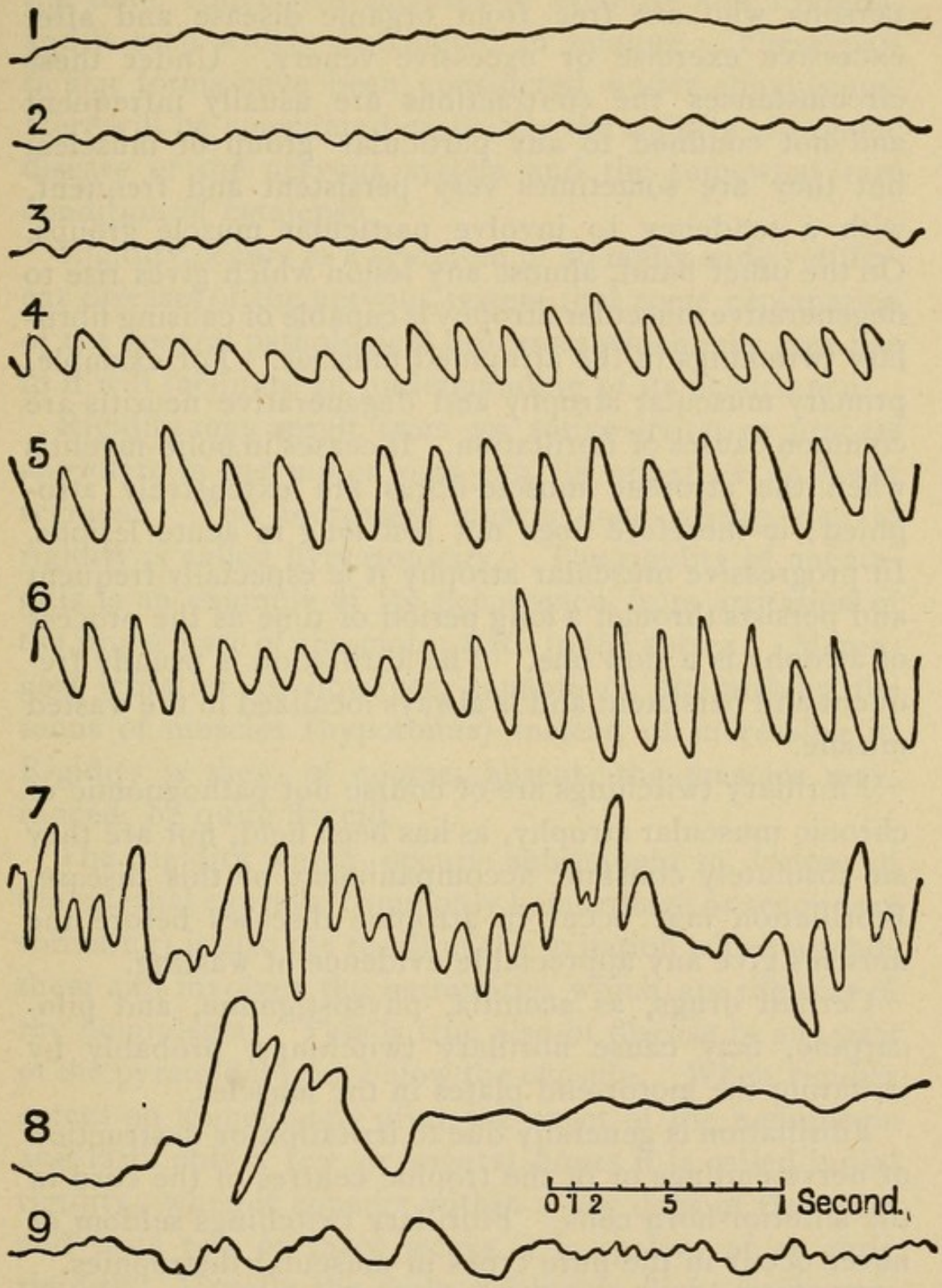


FIG. 63.

Myographic tracing of various forms of tremor taken directly from the moving parts. Reduced to one half. 1, tremor after hemiplegia, very fine, rather irregular; 2-6, paralysis agitans—2 and 3, very fine tremor, 4, 5, 6, coarse tremor; 7, insular sclerosis, very irregular in range, although in time; 8, general paralysis of the insane; 9, hysterical tremor. (Gowers.)

persons who are free from organic disease and after excessive exercise or excessive venery. Under these circumstances the contractions are usually infrequent and not confined to any particular group of muscles; but they are sometimes very persistent and frequent, with a tendency to involve particular muscle groups. On the other hand, almost any lesion which gives rise to degenerative muscular atrophy is capable of causing fibrillary twitchings in the atrophied muscles. For example, primary muscular atrophy and degenerative neuritis are common causes of fibrillation. It ceases in polio-myelitis when the atrophic muscle-fibres are extensively atrophied; it therefore does not last long in acute lesions. In progressive muscular atrophy it is especially frequent and persists through a long period of time as the process of atrophy is a slow one. The fibrillation is usually frequent and persistent and is always localized in the wasted muscle.

Fibrillary twitchings are of course not pathognomic of chronic muscular atrophy, as has been held, nor are they an absolutely constant accompaniment of this disease. Fibrillation may occur in atrophic diseases before the muscles give any appreciable evidence of wasting.

Certain drugs, as aconitia, physostigmine, and pilocarpine, may cause fibrillary twitchings, probably by irritating the motor-end plates in the muscles.

Fibrillation is generally due to irritation or destruction of nerve-endings or of the trophic centres of the cord in the anterior-horn cells. Fibrillary twitchings seldom or never occur in the pure types in muscular distrophies.

Rigidity.—The tonic muscular spasm, which shows itself as rigidity in one or more extremities or in the muscles of the trunk, is met with in both nutritional and structural diseases of the nervous system. It occurs in

the tonic convulsive seizures of epilepsy and hysteria, and is seen widely distributed in tetanus. These particular forms have been considered under convulsions. Here will be considered especially the rigidity of organic disease of the nervous system and the somewhat rare condition of catalepsy.

Rigidity occurs as a symptom of so many widely different diseases of the nervous system that some explanation of the general pathological processes which may give rise to it will facilitate an understanding of its occurrence.

Rigidity may result from an active irritative process anywhere in the motor path—in its upper or its lower segment. The increased muscle tone giving rise to rigidity is called hypertonicity. The rigidity of meningitis is an example of its occurrence from irritation of the upper part of the motor path in the cortex. Meningeal irritation is, however, capable of diminishing the tonus of muscles (hypotonus) instead of increasing it. Rigidity is then, of course, absent; the muscles may, indeed, be quite flaccid.

The rigidity which occurs subsequent to lesions of the internal capsule (commonly hemorrhage or secondary softening) is also the result of the irritation which attends them and involves the extremities which are the seat of the hemiplegia. This is true also of disease in any part of the pyramidal tract below the capsule. When rigidity comes on immediately after the onset of the hemiplegia and lasts only a few or several hours it is called initial rigidity; when it appears within a few days of the onset and lasts two or three weeks it is spoken of as early rigidity. Usually the early rigidity is slight in degree, occasionally it is very marked. Neither early nor initial rigidity is a constant accompaniment of hemiplegia. Both forms are certainly the result of irritation—initial rigidity

resulting from the mechanical irritation and shock of motor fibres by the lesion that interrupts their continuity, early rigidity from the inflammation which often follows. Lesions which may produce irritation and therefore rigidity in the pyramidal tracts of the cord are rather uncommon. External cord lesions however are common (meningitis, compression paraplegia from tumors) and rigidity is a frequent accompaniment of such affections.

But there is an influence other than irritation which is a common cause of rigidity. The normal tone of muscles depends on the integrity of the ganglion-cells of the anterior horns with which they are connected. Destruction of these cells results not only in paralysis of the muscles but in a loss of tone, as evidenced by their relaxation. Now these ganglion-cells are capable under normal circumstances of liberating more nervous energy than is required to give the muscles their normal tonicity, and are continually held in check or inhibited to prevent such overaction, by stimuli descending the pyramidal tracts from the cortex of the brain. If these inhibitory stimuli are cut off by the processes of disease, overaction of the spinal centres is the necessary result. There is hypertonicity or rigidity of the muscles. This rigidity may be constant, or it may diminish considerably during rest. Degeneration of the pyramidal tracts effectually interrupts the inhibition from the higher centres, and hence it is that we find rigidity in the many types of disease in which such degeneration occurs. After hemiplegia, for example, there occurs a secondary degeneration of the pyramidal tract in which the lesion lies. In the course of a few weeks the rigidity appears,—late rigidity it is termed,—and remains for months or years. If the degeneration be great in degree the rigidity may be

permanent. A similar secondary degeneration may result from interruption of the fibres in the pons, medulla, or cord, from hemorrhage, softening, or inflammation. The primary degeneration of the pyramidal tracts in the cord as seen in lateral sclerosis also gives rise to rigidity in paralyzed muscles. In the case of amyotrophic lateral sclerosis the presence of rigidity depends on the condition of the ganglion-cells of the anterior cornua. If these are only slightly injured and can still maintain the muscle tonus, this is increased by the degeneration in the lateral tract. If they are so extensively altered as to lose their control of the muscle tonus, no degree of degenerative change in the lateral tracts is capable of restoring or increasing the muscular tone. Hence it is that we have a tonic type of chronic muscular atrophy. (See p. 450.)

In general, we may say that rigidity is due to irritation of motor structures in acute processes, and to deficient control of the reflex mechanism which subserves muscular tone, in chronic forms of disease.

The motor overaction is seldom equal in degree in antagonistic muscle groups. Either the flexors or the extensors predominate and determine the position of the limb. If the spasm causes persistent shortening of a certain set of muscles the resulting condition is a contracture.

Contracture and Contraction.—These words are used in different senses by different authors, and this has given rise to considerable confusion. According to the best English usage the word *contracture* is applied to the shortening of a muscle or group of muscles from active contraction of the muscular fibres, and not from structural changes in these fibres. The contracture may last for a few minutes or for years. Good examples of

contracture are seen in cerebral tumor involving the motor tract, in the late rigidity of hemiplegia, and in hysteria. The contracture of hysteria may persist many years. A contracture can always be overcome temporarily by gentle, firm extension of the muscle.

A contraction is the shortening of a muscle or group of muscles from structural changes in the muscle-fibres —structural contracture it is sometimes called. A contraction cannot be reduced by extension. Contraction of a muscle from structural changes is always preceded by a period o. active contracture in which connective-tissue alterations have not yet occurred. The causes of contraction are:

a. The unequal paralysis of antagonistic muscles. The stronger muscles assert themselves and give rise to serious deformity of the limb, though there is no spasm. This is often seen in poliomyelitis.

b. From long-continued spasmodic contracture from any cause whatever. The structural changes in such cases develop very slowly—often not until many years have passed. Such structural changes occur even from hysterical contracture in some of the rare instances in which such contracture has existed unaltered for years.

Since contractures are generally associated with some degree of motor weakness, even when they result from spasm, they are often regarded as late conditions of paralysis, though it is to be remembered that they sometimes occur independently of motor loss. Contractures may result from paralysis of cerebral, spinal, or peripheral origin. By far the most common cerebral cause of contracture is hemiplegia. The contracture is produced by the late rigidity already mentioned. In the upper extremity the position which results from the contracture is one in which there is flexion of the elbow and

pronation of the wrist, and flexion, often great, of the fingers, especially of the second and third phalanges. By passively flexing the wrist these phalanges can be readily extended because such flexion lengthens the course of the flexor tendons, but if the wrist is extended somewhat, the fingers return to their original state of flexion. The contracture thus preponderates in the flexors, but there is usually some rigidity in the extensors. In the leg there is usually extensor contracture, the antagonistic muscles being much more equally balanced, and the leg is straight. In the foot there is a tendency, often marked, to take the position of talipes equinus or equino-varus.

The great cause of contracture from spinal-cord disease is poliomyelitis, and especially the infantile form. It is unusual for all the muscles of an extremity to be involved or to be involved equally. In the lower extremity the tibialis anticus and peronei suffer most frequently and there is a resulting contracture of the calf muscles, which are either unparalyzed or only slightly involved. Hence talipes equinus results; and it is equino-varus or equino-valgus according as the peroneal group or the tibialis anticus is most involved. When the calf muscles suffer most, as they occasionally do, talipes calcaneus results. In the arm contractures are much less apt to result than in the leg, and when they do occur give rise to less deformity.

The contracture of poliomyelitis early becomes structural contracture.

Contracture from disease of the peripheral nerves is not common. It occurs chiefly as the result of long-continued irritation, especially of the anterior nerve-roots, or as the result of injury to motor nerves.

Catalepsy.—This is a condition of infrequent occur-

rence in organic disease of the nervous system. It is practically confined to major hysteria and to the katatonic stupor of dementia precox. Trance, lethargy, tetanic rigidity, and cerea flexibilitas are all states of the same general condition. Catalepsy may be defined as a state of general or local muscular rigidity associated with a state of perverted consciousness. It may occur spontaneously in disease or it may be in-

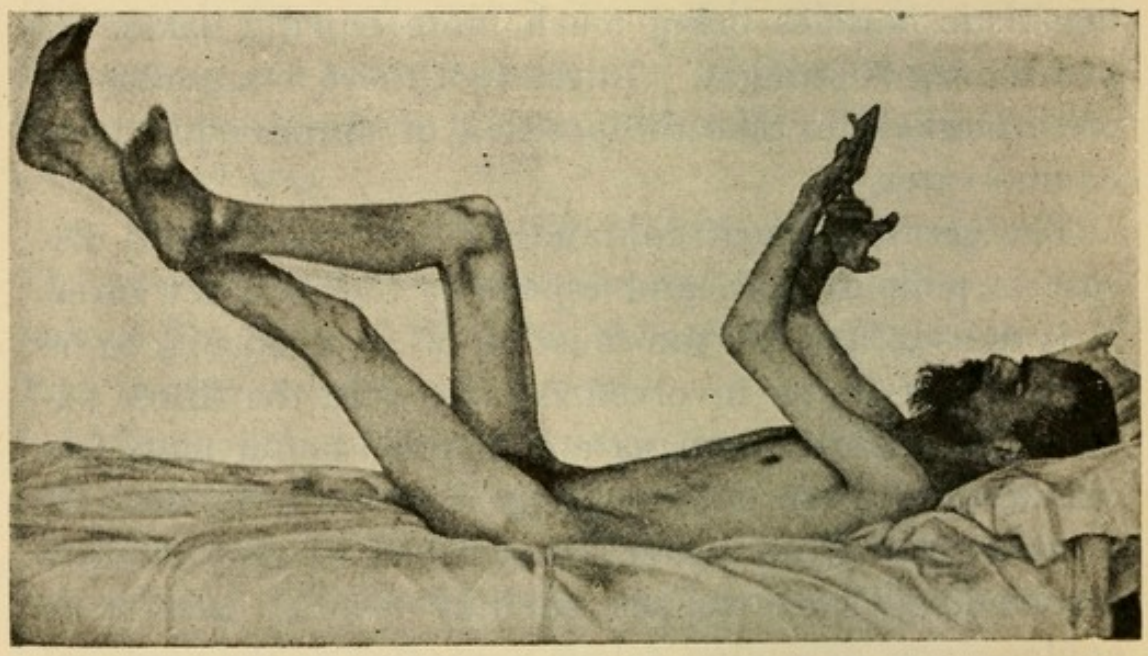


FIG. 64.

A case of catalepsy (Dana).

duced in a state of hypnotism. It has been noted in brain abscess, tumor, hemorrhage, softening, and meningitis, but only when consciousness has been much impaired. Its practical diagnostic significance at present is confined to hysteria and the adolescent insanities. In the absence of hysterical stigma and the presence or a history of stupor, negativism, automatism, excitement alternating with depression, stereotypy, verbigeration, etc., it points to pronounced mental disease. In the latter

disease the origin of the katatonic stupor is commonly rapid with partial or complete obscuration of consciousness. The features grow expressionless, the respiratory movements and heart action are depressed. The voluntary impulses seem to be overcome by counter impulses. An act may be begun but almost immediately is checked by a counter impulse which produces an act contrary to the one desired.

Athetosis and Athetoid Movements.—The word athetosis (meaning, "without form") was introduced to designate a peculiar form of chronic spasm—"mobile spasm" it has been aptly called—which gives rise to slow, irregular inco-ordinated movements. The movements usually involve the hand and foot, sometimes also the face, but they are always most marked in the hand. In distribution the movements may be either strictly confined to one side of the body, or rarely they may be bilateral.

This condition of athetosis has been looked upon as a primary type of disease since it is said to occur occasionally in persons apparently healthy, unassociated with any other abnormal state.

Movements of essentially the same character as those of athetosis are not infrequently seen as a post-hemiplegic condition. They are often met with in children and sometimes in adults. As in athetosis they affect the hand more often and in greater degree than the foot. Usually the lesion which causes them is unilateral and hence the movements are generally one-sided. When the lesion is bilateral the movements may involve both sides of the body. These "athetoid movements," as they are called, never occur while the hemiplegia remains absolute, but develop with the return of voluntary power, usually some months after the onset of the

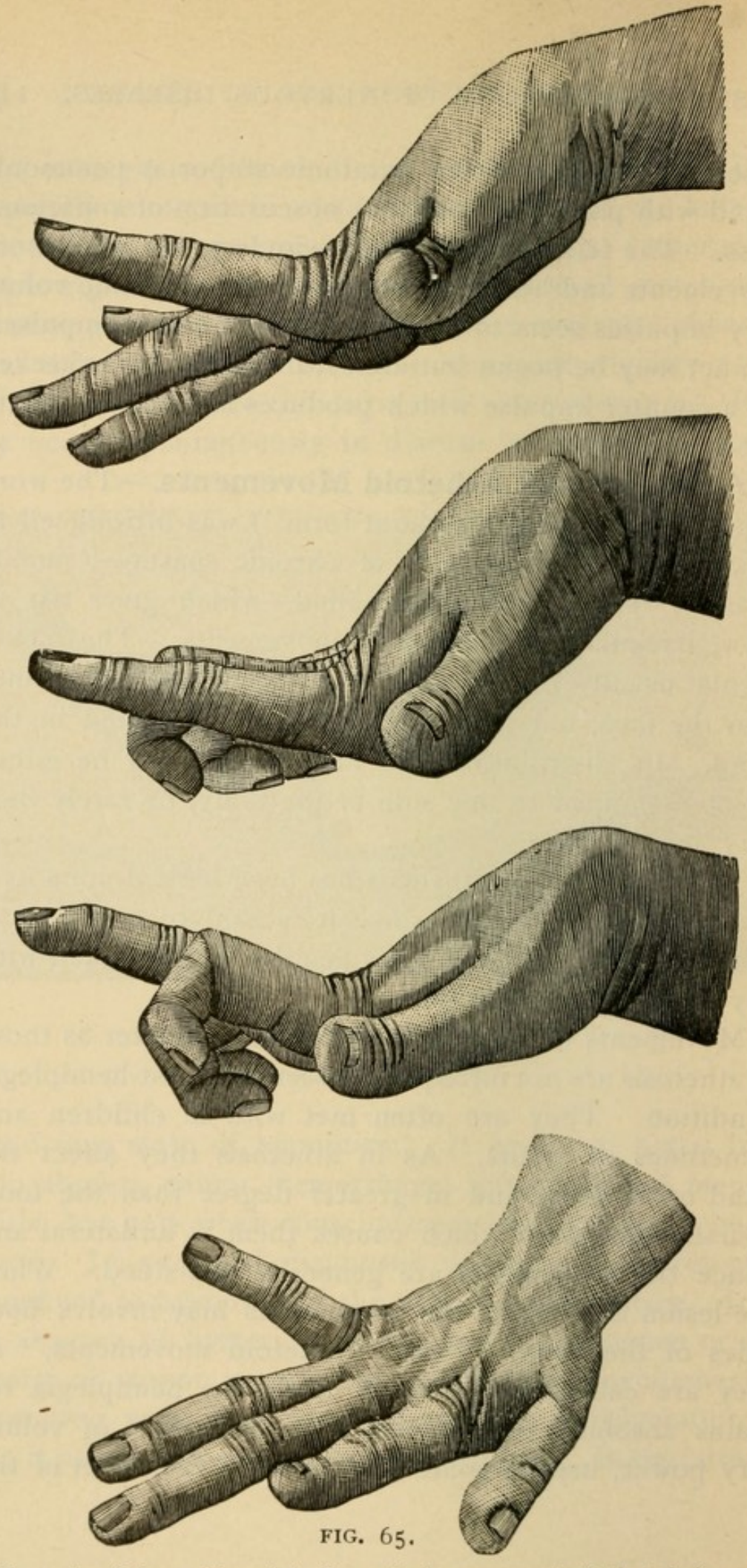


FIG. 65.

Examples of the position of the fingers in the movements of athetosis (personal observation). (Strumpell.)

paralysis. There is commonly associated with these spasmodic movements a certain amount of muscular rigidity which tends to give to the part some particular attitude, and this rigidity is related to the degree of paralysis—being slight when the weakness is inconsiderable. The recognition of the dependence of this symptomatic form of athetosis, as it may be termed, upon a cerebral lesion is usually not difficult. The history of a hemiplegic attack, the loss of power and rigidity in the extremity which is the seat of the athetoid movements, and in children the arrest of development in bone and muscle, all point clearly to an organic cerebral process. But it is well known that a slight lesion, especially during childhood, may be so far recovered from, in the course of time, that there remain only slight objective evidences of a cerebral process. Under these circumstances the distinction between a symptomatic athetosis and a primary athetosis may be exceedingly difficult or impossible. Hence it is not difficult to understand that from time to time cases of what are evidently symptomatic athetosis (athetoid movements) should be described as cases of primary athetosis. This circumstance, and the fact that a careful examination of a supposed case of primary athetosis in the light of our present knowledge of infantile hemiplegia often reveals traces of an old hemiplegia which ten years ago might have been overlooked, has led some observers to deny the existence of a primary athetosis. But while we have reason to think that many so-called cases of athetosis are of post-hemiplegic origin, we are not at present justified in maintaining that this is true of every case which has been described as athetosis.

The athetoid movements are usually slow, and always independent of voluntary motion, though they may be increased by it. Sometimes the movements are not slow

but quick, and they may then bear some resemblance to chorea. On the strength of this occasional resemblance these athetoid movements have been described as post-hemiplegic chorea, or choreatic paresis—an objectionable term, because the condition has nothing to do with the true chorea.

As already mentioned, athetoid movements are a sequel to hemiplegia, particularly of that which occurs in childhood. The lesion is usually meningeal hemorrhage or venous thrombosis; sometimes it is embolism, sometimes hemorrhage. In adults the disease may succeed cerebral hemorrhage or softening from any cause. In general, the movements are more likely to follow cerebral softening than hemorrhage. As regards the position of the lesion which underlies these athetoid movements or choreatic paresis, no accurate generalization can yet be made. Frequently the motor cortex has been the seat of disease, and it has been shown that disease in this situation is by itself competent to occasion the symptom. Disease of the internal capsule may cause the symptom, and involvement of the adjacent gray matter (optic thalamus and caudate nucleus) is probably not an essential condition for its production. Any slight diffuse lesion of the motor path of the brain during development may give origin to the symptom.

Associated Movements.—When a voluntary movement of one group of muscles is accompanied by an involuntary contraction of muscles in another part of the body this is termed an associated movement. Associated movements are met with generally as the result of hemiplegia, particularly the hemiplegia of infancy. Usually the movements are confined to the upper extremity, the fingers of the paralyzed side undergoing involuntary movement, of limited range, during the voluntary activ-

ity of the other arm. Sometimes these movements are associated only with extensive movements of the normal limb, and are then themselves extensive in range, as when the paralyzed arm is lifted above the head, when the other is employed in an action which, like pulling the strap in a car, requires the elevation of the arm above the head. More commonly the associated movements are narrow in range, and are best seen when the hand of the unparalyzed side is employed in fine co-ordinated movements, as in the buttoning or unbuttoning of the coat, or in the fingering of any small object. Associated movements have a significance similar to that of athetoid movements. Their explanation is doubtless to be found in the connection of the subsidiary centres through which they are produced. They possess little diagnostic importance.

Inco-ordination.— During an ordinary muscular movement there occurs not only an accurately proportioned contraction of the various muscles which produce the movement, but a less powerful contraction of antagonistic muscles. This adjustment of opposed muscles makes the movement which results from their activity regular and continuous. If the contraction of muscles in either group is excessive, or less than normal, the movement which results is not that intended, but is irregular and broken, and efforts to remedy it are often excessive in degree and increase the difficulty. The condition of irregular, inco-ordinated voluntary movement is termed ataxia. It is difficult to say just what defects of co-ordination the term ataxia should include; whether it should be applied to all forms of inco-ordination, or whether it should be strictly limited to that form of inco-ordination in which there is loss of harmony in the various muscle-groups used in the purely voluntary

execution of particular acts and in which an effort to correct the defect only serves to increase it. We shall employ the term in this restricted sense. Under this definition ataxia does not include jerky inco-ordination or ataxic tremor such as is seen in multiple sclerosis and general paralysis, nor mere uncertainty in voluntary movements, nor the staggering which results from defective equilibration.

According to the position of the lesion in the nervous system it is customary to distinguish several varieties of ataxia. Thus there are described a cortical ataxia, a cerebellar ataxia, a bulbar ataxia, a spinal ataxia, and an ataxia from peripheral nerve disease. Probably all are cortical in their primary origin. Some of these regional forms, however, are not ataxias, strictly speaking, and we shall therefore speak of them as varieties of inco-ordination, specifying which are true ataxias.

Cortical Inco-ordination.—Inco-ordination of movement from cortical lesions is usually associated with paralysis of the same distribution. The inco-ordination which is observed in athetoid movements (so-called post-hemiplegic chorea) has been mentioned. In cases of infantile hemiplegia, however, inco-ordination may be developed only during voluntary movement and be of a jerky, oscillatory character, deserving the name of ataxia (hemiataxia it is called, when due to a unilateral lesion). Sometimes it is difficult to say whether we should class a given form of post-hemiplegic inco-ordination as an active tremor or as a true ataxia. The same difficulty is met in the inco-ordination of dementia paralytica, which is sometimes truly ataxic, but more generally consists of movements of narrow range which are more properly designated tremor.

The disturbances of co-ordination which occur in

hysteria are probably of cortical origin. They may exist alone or be accompanied with loss of power or anæsthesia. It is doubtful whether the movements are ever those of true ataxia. In one form movements are well controlled and regular while the eyes are directing them, but when the visual control is withdrawn they become grossly irregular. In other cases there is good co-ordination while the patient lies in the horizontal position, but in standing she begins to sway from side to side. Again hysterical patients in whom there is no loss of cutaneous or muscular sensibility, though quite steady with the eyes open, may oscillate from side to side or even fall when the eyes are closed. The term "astasia" is sometimes used to designate the unsteadiness in standing, with or without visual guidance, that is seen in some cases of hysteria. The word "abasia" is used to designate the unsteadiness in walking, which often does not amount to inco-ordination, seen in the same class of cases.

The effect of withdrawing the visual guidance in these cases is greater than it ever is in true ataxia without defective sensibility. This latter form of inco-ordination may be of considerable diagnostic value, since it may exist when other marked evidences of hysteria are absent.

Why ataxia sometimes occurs in cortical disease but more frequently does not occur, it is impossible at present to say. Sometimes it seems to be due to damage of the cortical centre for the muscular sense (superior parietal lobule), sometimes to the disturbance of cutaneous sensibility which the lesion produces. The inco-ordination seen in alcoholic and other forms of intoxication is probably in part cortical.

Cerebellar Inco-ordination (*cerebellar titubation*).—This form of motor disturbance consists in an inability to

maintain the erect position or to walk straight, in consequence of deficient equilibrium. The patient, in order to increase his base of support, stands with the feet apart. The body is bent forward and the arms are used to help in the maintenance of the equilibrium, but there is no true ataxia, although the condition is often called cerebellar ataxia. The forefinger can be placed on the nose without difficulty, and if the patient is supine, there is no difficulty in moving the lower extremities as desired. The disorder is chiefly one of defective equilibration and the gait frequently is much like that seen in intoxication from alcohol. Rarely the upper extremities present marked jerky inco-ordination. This symptom is generally due to damage of the middle lobe of the cerebellum, sometimes to disease in it, sometimes to pressure upon it from disease of one of the cerebellar hemispheres. Disease of the cerebellar hemispheres is *per se* not productive of cerebellar inco-ordination; but wild inco-ordinated movements, resembling those of cerebellar titubation, may result from pontine and medullary disease, probably from damage to the sensory tract (fillet). A peculiar difficulty in maintaining the equilibrium in the act of walking is sometimes seen in paralysis agitans, and may bear some resemblance to cerebellar titubation. As the patient tries to walk, the first steps may be taken with difficulty and slowly, but they gradually increase in rapidity and soon the patient may appear about to run in a staggering way. This tendency to run is termed "festination." The body often inclines forward, and the patient may seem to be about to fall as he staggers forward—"propulsion" the tendency is called when marked. More rarely it is easier for the patient to walk backwards than forwards, and there then exists an unavoidable inclination to fall backwards; this is termed

"retropulsion." Lateral pulsion may also occur. The occurrence of festination has been accounted for on the supposition that the forward inclination of the body and head makes the patient run after his centre of gravity, but this is certainly not the entire explanation for retropulsion, which doubtless has a similar mechanism, may occur though the head and body still incline forward.

Bulbar Inco-ordination.—Occasionally inco-ordination is met with as a result of disease of the medulla involving the fillet (see page 60), which is probably one path for sensory impressions from the muscles. This is an unusual cause of inco-ordination. The characters of bulbar inco-ordination appear to be largely those of the inco-ordination of spinal-cord disease.

Inco-ordination from Spinal-Cord Disease.—The muscular inco-ordination which constitutes so important a symptom in certain forms of spinal-cord disease is probably of sensory origin, though it is usually classed with motor symptoms. The underlying cause of the symptom, which in chronic disease of the cord usually amounts to ataxia, is in the majority of instances disease of the posterior columns of the cord, especially the posterior median column, occurring alone or in combination with disease of the posterior nerve-roots. Disease of the posterior median columns, containing, as we have seen (page 60) one of the paths for muscular sensibility, is probably operative in the production of inco-ordination by the arrest of the upward conduction of impressions from the muscles to the cerebellum. The impressions thus carried are probably an important factor in cerebellar co-ordination, and it is not difficult to conceive how interruption of this path may bring about disturbance of co-ordination. In the condition known as ataxic paraplegia this is the probable mechanism of the ataxia that

is observed. In cases of unilateral lesion of the cord, also, loss of the sense of posture, followed by ataxia, has been observed. This ataxia is referable to arrest of sensory conduction from the muscles, and not to anæsthesia, for the ataxia is on the same side as the lesion, while the cutaneous anæsthesia is on the opposite side, and the ataxic muscles have been known to lose their sensitiveness to pressure. The injury to the direct cerebellar tract, one of the paths of muscular sensibility, which occurs in these cases of unilateral lesion, may also be a factor in the production of the ataxia.

But the mechanism of inco-ordination in spinal-cord disease is not always so simple, for in ataxia in its most typical form there occurs an almost constant process of disease in the posterior nerve-roots and often there is also disease of the sensory peripheral nerve-fibres. Among other effects resulting from the involvement of these structures is cutaneous anæsthesia, which though not an absolutely constant condition is rarely entirely absent. This cutaneous anæsthesia has been regarded by some as a very important factor in the production of ataxia. But it is highly probable that while cutaneous anæsthesia may be an accessory cause of ataxia it cannot operate as a primary cause. It has been shown that there is no definite relation between the degree of inco-ordination and the degree or extent of the anæsthesia in conditions where both are present, and it is not rare to meet with absolute and extensive anæsthesia without the least ataxia, while on the other hand, there are cases in which there is marked ataxia without anæsthesia. Yet there is good reason to believe that anæsthesia may increase pre-existing ataxia, and it probably does so by withdrawing the guidance to the motor cerebral centres which the ordinary cutaneous impressions continually

furnish. It is interesting to note in this connection that visual impressions give similar guidance and that the withdrawal of this aid by closure of the eyes always increases pre-existing inco-ordination. We may therefore regard anæsthesia as an element in the production of inco-ordination of locomotor ataxia. As in ataxic paraplegia, the primary factor is usually the interruption of impressions from the muscles to the cerebellum consequent upon sclerosis of the postero-median columns or more precisely disease of the afferent muscle nerves. We shall presently see that disease of the sensory peripheral nerves is competent to give rise to inco-ordination and such disease is always encountered in locomotor ataxia. We must therefore admit that this factor is the essential one for the ataxia of tabes.

Inco-ordination from Disease of Peripheral Nerves.— Inco-ordination of movement may occur from disease involving exclusively the peripheral nerves. It may be dependent in part upon the resulting anæsthesia as just explained, but the important factor is probably the interruption of impressions from the muscles to the cerebellum, the result of disease of the sensory muscle nerves. The process may involve the sensory nerves throughout their extent, from the muscles to the cord, or may involve them in a limited part only of their extent, for example at the posterior nerve-roots as in locomotor ataxia. Inco-ordination of similar origin is seen in alcoholic, diphtheritic, and some rarer forms of multiple neuritis. In the alcoholic form the inco-ordination is sometimes indistinguishable from that of tabes.

The sense of posture is undoubtedly derived in great part from the stimulation of the sensory muscle-nerves by muscular contraction and tension, but it is probable that nerve filaments supplying adjacent joint-surfaces

play a rôle in the recognition of posture by centripetal transmission of impressions from the apposition of joint-surfaces at different parts, according to the position of the segments above and below the joint. These sensory impressions probably help in the co-ordination of muscular movements, and their interruption may lead to some disturbance of co-ordination, but how far this mechanism may be effective we do not yet know.

Certain symptoms referable directly or indirectly to the ocular muscles may be conveniently considered here. These are conjugate deviation, strabismus, diplopia, nystagmus, and ptosis.

Conjugate Deviation of the Eyes and Head.—In many cases of brain disease there is a lateral movement of the eyes (by the external rectus of one side and the internal rectus of the other) and often a rotation of the head (chiefly by the opposite sterno-mastoid) toward one or other side. This is known as conjugate deviation. The axes of the eyes are always parallel in conjugate deviation, whatever may be their direction.

Conjugate deviation occurs most often as an accompaniment of hemiplegia, whatever may be its cause, and may occur when the lesion is in the cortex (probably the posterior part of the second frontal convolution), in the subcortical white substance, in the internal capsule, or in the pons. The symptom may depend on a destructive process (paralytic conjugate deviation) or on an irritative process (spasmodic conjugate deviation). In the former case the deviation is to the side of the lesion; the patient is said to "look at his lesion." In the latter case the deviation is to the side opposite the lesion—that is, to the paralyzed side. Spasmodic conjugate deviation is generally transient. It is often present for a short time after acute hemiplegias, and is not rarely seen in

acute meningitis. The spasmodic form also occurs in some cases of uræmia, generally associated with convulsions. It then depends on cortical irritation by morbid blood products.

In cases where conjugate deviation occurs the associated condition of the limbs shows whether the symptom is paralytic or spasmodic in origin.

In some cases of disease of the pons there occurs a peculiar form of associated paralysis of the external rectus of one side with the internal rectus of the other, in which the eyes can be turned towards the lesion to the middle line, but not beyond it.

Strabismus.—Strabismus is loss of parallelism in the visual axes of the two eyes. This loss of parallelism is a conspicuous symptom of many cases of paralysis of the ocular muscles. But strabismus is not always of paralytic origin; it may depend on spasm of ocular muscles. Spasmodic strabismus is usually transient, with one exception—the ordinary form of squint (due most often to myopia), in which it is persistent.

In cases of strabismus dependent on paralysis there occurs what is known as "secondary deviation." Primary deviation is the deviation of the axis of the eye paralyzed, from parallelism with that of the normal eye. Secondary deviation is something quite different. It is an excessive movement on the part of the sound eye, which increases the deviation from parallelism, when the sound eye is prevented (as by interposing the hand or a piece of ground glass) from seeing an object looked at with the eye paralyzed.

By means of this secondary deviation it is possible to distinguish between paralytic and spasmodic strabismus, for secondary deviation always occurs in paralytic, never in spasmodic strabismus. Spasmodic strabismus is the

same in all positions of the eyes; paralytic strabismus occurs only in those positions of the eyes that necessitate the use of the muscle or muscles affected.

Strabismus may be divergent (external strabismus) or convergent (internal strabismus) or may cause a difference in the level of the two eyes.

Transient spasmodic strabismus is not rarely seen in irritative brain diseases, as meningitis, tumor, abscess, hemorrhage, or softening, and occasionally occurs in hysteria.

Diplopia.—Double vision is present in all recent cases of strabismus, and is due to the erroneous projection of the field of vision of the affected eye. When the strabismus is of long duration (whatever may be the cause) the patient learns to disregard the image formed on the retina of the affected eye ("false image"), and he then becomes aware only of those things that are seen with the normal eye. In such chronic cases the false image may be brought out by placing a bit of colored glass before one eye (preferably the sound eye) in order to color either the true or false image (the true image in the case of the normal eye).

Double vision is often an early symptom of brain disease, as meningitis, tumor, abscess, multiple sclerosis. In many cases it accompanies the onset of sudden lesions, especially those in or near the pons. It depends here on spasmodic strabismus, but this is of such duration as to be unrecognized. The occurrence of diplopia in children who are free from eye disease (errors of refraction) is suggestive of meningitis. When not dependent on eye disease diplopia is usually a sign of serious nervous disease, but this is not always the case. It occurs, for example, as a transient symptom in many cases of acute

alcoholism and certain drug intoxications and in some cases of cerebral concussion.

Nystagmus is the term applied to rapid involuntary and rhythmical movements of the eyes. The movements are usually bilateral and similar in character in both eyes. Most often the movements are horizontal; sometimes they are rotary; rarely they are vertical. They vary in frequency from 60 to 200 movements per minute in the same direction. All forms cease during sleep. Usually the rhythm of movement is regular.

Nystagmus is usually constant. Occasionally it occurs only when the eyes are moved in certain directions. Often it persists in central fixation. In cases that date from early life, the patients are not conscious of the movement of their eyes; but in some cases that begin in adult life there is apparent movement of objects at least for a time.

Nystagmus is observed in a number of different conditions. It occurs (*a*) from conditions external to the nervous system, and (*b*) in many different diseases of the nervous system. Of the conditions external to the nervous system that cause nystagmus, the following are the chief: albinism, a very common cause, and local eye affections causing defective sight, as opacities of the cornea or lens and diseases of the choroid or retina (effective chiefly in childhood). It is also of frequent occurrence in miners who work in coal mines in a dim light in postures that require excessive tension in certain eye muscles for a long time.

Of the diseases of the nervous system in which nystagmus occurs the most important are these: multiple sclerosis, hereditary ataxia, meningitis, meningeal hemorrhage, and cases of tumor, and hemorrhage and softening

in almost any situation. Cerebellar disease (especially tumor) is often associated with nystagmus, and pontine disease is occasionally the apparent cause of nystagmus. In chronic ear disease and in mastoid disease nystagmus is sometimes developed, in association usually with symptoms indicating irritation of the adjacent membranes. Severe cerebral concussion is sometimes followed immediately by nystagmus (the oscillations may be very rapid) of temporary duration, and may depend in these cases on minute hemorrhages into the brain substance. Nystagmus is occasionally observed in the course of uræmic convulsions and exhaustion of status epilepticus.

The mechanism and significance of nystagmus are but imperfectly understood. It is certainly central in origin and probably due to a disarrangement of function of the third nerve nuclei of the two sides or their connection with centres of equilibration. The symptom certainly has little or no localizing value, for it arises from disease in a great variety of positions in the brain. It is, however, an important symptom, for it usually indicates more than purely functional disorder, especially when associated with other evidence of nervous disease.

When it develops gradually in adults as the result of nervous disease this is most often degenerative in nature.

Ptosis, or drooping of the eyelid, is a symptom of several distinct conditions. Most often it is the result of disease of the third nerve (generally syphilitic disease), and is then associated with other symptoms. But it may result from disease of the third nerve nucleus, or of the tract of the third nerve anywhere between the nucleus and the cortical centre. (The position of the cortical centre is uncertain ; perhaps it is in the lower parietal lobule.) Generally, ptosis from disease of the third nerve tract is unilateral, but in some cases of nuclear disease

and disease of the nerve itself the symptom is bilateral.

Ptosis from paralysis of the sympathetic is occasionally seen, and is distinguished by other evidences of sympathetic involvement, especially contraction of the pupil on the same side. In these cases the drooping of the lid probably depends on paralysis of the smooth muscular fibres of the fascia of the orbit (fibres of Müller), which are supplied by the sympathetic, and normally act on the tarsal cartilages.

Some cases (usually bilateral and partial) of ptosis are congenital, and ignorance of this fact may lead to errors in diagnosis. Hysterical ptosis is sometimes seen, and may be bilateral or single. It is generally associated with spasm of the orbicularis.

Reflex ptosis, a rare form, generally depends on some morbid state (irritative usually) of the fifth nerve, as extraction of a tooth, or injury of the nerve. In some neurasthenics ptosis develops in middle life. It is always double. "Morning ptosis" is a form in which the patient, on waking, is temporarily unable to lift the lid. Ptosis also occurs in the relapsing and recurring palsies of the third nerves. Some other forms of ptosis are associated with over-action of the occipito-frontalis.

Pupillary Symptoms.—Deviations from the normal reaction and size of the pupils are of frequent occurrence in disease of the nervous system, and possess considerable diagnostic importance. The most important of these deviations are the following:

(1) *Dilatation of the Pupils* with normal reactions to light and during accommodation. Such dilatation is often observed in children and in nervous and irritable adults in apparently good health. It is frequently seen in neurasthenic, hysterical, and maniacal persons, and

also occurs as a result of irritation of the cervical sympathetic, either direct or through the irritation produced by certain lesions of the cervical spinal cord. Stimulation of the skin of the neck normally causes temporary dilatation of the pupils.

(2) *Contraction of the Pupils* is observed in senility, in locomotor ataxia, in dementia paralytica, in acute lesions of the pons and medulla (hemorrhage and softening), in many irritative diseases of the brain (meningitis, tumor, abscess), in irritative conditions of the eye (iritis, cilliary neuralgia), and in some forms of poisoning, especially opium and chloral poisoning. In locomotor ataxia and dementia paralytica the myosis is associated with loss of light reflex and preserved accommodation reflex, or with total immobility. A high grade of myosis in persons in the first half of life, who are free from refractive errors, is always pathological and suggests organic disease of the nervous system. Dilatation of the pupil in the second half of life, in those without eye-disease, has the same significance.

(3) *Dilatation of the Pupils with Immobility* is observed in almost all forms of coma, in cases of advanced glaucoma, and in lesions of the third nerve. The pupil is contracted in coma only when the condition results from poisoning, or occurs in a person who has myosis from an organic degenerative disease of the nervous system. Dilatation of the pupil, with paralysis of accommodation and sluggish but preserved contraction to light, is often seen in diphtheritic paralysis. In these cases there is usually contraction on an endeavor to accommodate.

(4) *Loss of Reaction to Light with Preservation of Reaction during Accommodation* (Argyll-Robertson pupil) is the most significant of all pupillary symptoms. It probably always indicates organic disease (especially degenerative

disease) of the brain, which interrupts the reflex path between the optic nerve and the nucleus of the third nerve. The Argyll-Robertson pupil is a characteristic symptom of locomotor ataxia and of general paresis, and is an occasional symptom of multiple sclerosis. In rare cases it is observed in persons who have syphilis but have no signs of organic brain disease. The Argyll-Robertson pupil is usually associated with contraction of the pupil; occasionally with dilatation, or with pupils of normal or unequal size.

(5) *Mere Difference in the Size of the Pupils* has no single significance. Such difference is common in cases where the two eyes refract differently. A high grade of inequality is sometimes seen when there is little defect in vision. Inequality is often observed in irritative diseases of the brain—meningitis, abscess, tumor, extra-dural hemorrhage, etc., and in degenerative diseases, such as locomotor ataxia and dementia paralytica—but is seldom of more than secondary importance in diagnosis.

Changes in the size and reaction of the pupils from nervous diseases are readily distinguishable from the changes in size and immobility that depend on iritic adhesions. The immobile pupils of iritis are almost always irregular in form, and proper illumination will reveal the presence of synechiæ.

(6) *Hemianopic Pupillary Inaction.*—This term is applied to the imperfect reaction which occurs when light is thrown on the blind half of the retina in certain cases of hemianopsia. The cases of hemianopsia in which pupillary reaction is diminished or lost when the blind half-field is stimulated, are those in which the lesion is in the optic tract; the cause of the phenomena being the interruption of the path to the corpora quadrigemina (or ganglion habenulæ). In cases of hemianopsia due to

lesions of the hemisphere the hemianopic pupillary inaction does not occur. Hence the symptom, if carefully determined, is of value in distinguishing between lesions of the tract and those of the intra-cerebral optic path and centres.

Laryngeal Symptoms.—Disordered action of the larynx, the organ of voice, may give rise to important symptoms and signs. Of these symptoms the chief are: (1) altered phonation, or entire loss of voice: (2) respiratory symptoms, as stridor and dyspnœa, from imperfect regulation of entrance of air in breathing, or cough from imperfect closure of the glottis. The signs of laryngeal disorder consist chiefly of defective movement of the vocal cords, which may be observed by means of the laryngoscope.

It is convenient to consider the grouping of these symptoms and signs in the various forms of laryngeal disorder (especially paralysis) that are met with as the result of nervous disease.

(1) In *Total Bilateral Paralysis* of the larynx (or what is more common, paralysis of all the laryngeal muscles except the crico-thyroid) the vocal cords are in the "cadaveric position"—that is, in a state of slight abduction from the median line, and cannot be moved. When an attempt at phonation is made the cords cannot be approximated; hence there is loss of voice. There is also inability to make a natural explosive cough—a symptom of much importance and one which always indicates laryngeal paralysis, usually organic in character. There may be stridor during deep inspiration.

(2) In *Total Unilateral Paralysis of the Larynx* the paralyzed cord lies motionless in the "cadaveric position." The unparalyzed cord moves freely and may even pass

beyond the median line of the glottis during phonation. In consequence of this compensating action phonation is possible, but the voice is hoarse and low in pitch. There is no stridor, and an explosive cough is impossible. Total unilateral paralysis of the larynx may result from disease of the spinal accessory nucleus or its nerve-roots by the side of the medulla, or from disease of the root of the pneumogastric. But the most common cause of unilateral paralysis, and indeed of all laryngeal paralyses, is disease of the recurrent laryngeal nerve. Aneurism of the vessels round which the nerve passes, thoracic tumor, enlarged bronchial lymph-nodes, and enlargements of the thyroid are all causes of pressure upon the nerve. The crico-thyroid muscle is of course exempt in these cases, but the symptoms are practically the same as when all the muscles are involved. The causes of total bilateral palsy are the same as those of the unilateral form, acting on both sides.

(3) In *Total Abductor Paralysis of the Larynx* (paralysis of the posterior crico-arytenoids) the cords lie close together (much nearer than in the position of cadaveric rigidity), and are not abducted during inspiration. The effect upon respiration is that inspiration is difficult and accompanied with stridor. There may even be alarming dyspnœa, with lividity of the face, etc. Expiration is unembarrassed, cough is normal, and the voice is little changed.

(4) In *Unilateral Abductor Paralysis* the recession of the unparalyzed cord prevents both stridor and the dyspnœa which characterizes the bilateral palsy. Neither cough nor voice are distinctly affected. The symptoms are therefore chiefly negative.

Abductor paralysis of the larynx may be due to local palsy, as laryngeal catarrh, or to central disease. Some-

times it appears to be due to disease of the recurrent laryngeal nerve. Occasionally it occurs as an hysterical paralysis, and is then double.

(5) In *Adductor Paralysis* the vocal cords are separated and cannot be brought together nearer than the cadaveric position, but abduction is possible during deep inspiration. The voice is lost, but cough is usually perfect, because the paralysis is generally partial and the cords can be approximated during the attempt to cough, though not for phonation. There is no stridor and no dyspnœa.

Adductor paralysis is only rarely due to organic disease. It is most often of hysterical origin, and gives rise to the aphonia of hysteria. Occasionally it results from simple catarrhal laryngitis.

(6) *Adductor Spasm of the Larynx* is a common occurrence in many laryngeal diseases. It always depends on a state of abnormal central excitability. Under favorable conditions of excitability in the laryngeal mechanism (such as are frequently present in children) almost any nerve irritation, whether direct, reflex, or central, may determine a paroxysm of adductor spasm. The paroxysms consist of sudden and intense difficulty in breathing and loud stridor, lasting only a few minutes.

In children very slight nerve irritation often suffices to determine the paroxysm. In children with rickets laryngeal paroxysms of peculiar type, associated with cardiac and vaso-motor disturbance and known as "laryngismus stridulus," are not uncommon. These paroxysms are related in their pathology to the general convulsions which may occur in rickets. Any irritative lesion of the superior or inferior laryngeal nerve may cause adductor spasm, which may be bilateral or unilateral. Adductor spasm occurs also at the onset of attacks of grand mal,

causing the "epileptic cry," during hydrophobia, and occasionally in hysteria and tetany.

Abnormal Reflex Action.—The modifications of reflex action which possess diagnostic value may conveniently be considered here.

Loss of Reflex Action occurs whenever the reflex arc which subserves the reflex in question is interrupted.[1] As every reflex arc consists of a centripetal portion (sensory nerve), a centrifugal portion (motor nerve), and a central portion (motor ganglion-cell of cord), the disease abolishing reflex action may be in any one of these three positions. In which particular one it lies in a given case is to be determined by the associated conditions. If the disease is in the sensory path, there is loss of sensibility as well as of reflex; if in the peripheral motor path, or in the motor centre, there is corresponding loss of motion, with nerve degeneration and atrophy. This is true both of the superficial and of the deep reflexes, but in the case of the former, it has little diagnostic value, while as regards the latter (especially the knee-jerk), it is of diagnostic importance, for the degenerative atrophy which results from implication of the motor elements is readily appreciated in the latter but not in the former instance.

Disease within the reflex arc is not the only cause of arrested reflex action, for both superficial and deep reflexes may be temporarily arrested by irritative inhibition from above—that is, from irritative disease at any level higher than the motor ganglion-cells on which the reflex depends. This is the explanation of the unilateral reflex loss which is frequently encountered immediately after hemiplegia, and of the bilateral reflex loss which is seen below the lesion for a variable time after sudden

[1] The different levels of the spinal cord upon which the more important reflexes depend, are given on pages 24 and 25.

damage to the cord—as from fracture or dislocation of the cervical vertebræ. Persistent loss of cutaneous reflex (unilateral or bilateral) occasionally occurs as a result of disease of the hemispheres. Persistent loss of tendon reflex probably never occurs from brain disease, but temporary loss of knee-jerk occurs sometimes in irritative disease (tumor) of the cerebellum, and is not uncommon in lesions which irritate the cortex of the convexity (convexity meningitis, subarachnoid hemorrhage). Probably this is due to irritative inhibition.

Loss of Patellar Tendon Reflex (knee-jerk) is of much diagnostic significance. The more important conditions in which it occurs may be tabulated as follows :

(A.) Diseases of the brain :

Almost any irritative brain disease involving motor structures (tumor, meningitis, abscess, hemorrhage, acute softening) ; loss usually unilateral, always temporary (hours, days or weeks) ; in convexity meningitis and subarachnoid hemorrhage (bilateral) the loss may be bilateral.

(B.) Diseases of the spinal cord :

1. Locomotor ataxia. Very early and constant symptom ; bilateral (in very early stage may be present on one side) ; due to destruction of reflex arc by disease of posterior nerve-roots ; persistent.

2. Poliomyelitis of lumbar cord. Immediate onset ; generally unilateral, sometimes bilateral ; due to interruption of reflex arc by disease of ganglion-cells of anterior horns.

3. Any diffuse destructive process in lumbar cord, as transverse myelitis.

4. Spinal meningitis. After stage of irritation ; due to compression of anterior or posterior nerve-roots by exudation.

(C.) Diseases of the peripheral nerves :

1. Alcoholic neuritis. Loss usually occurs early ; not absolutely constant, for knee-jerk may occasionally be present throughout the disease when muscle-nerves for extensors of knee are not involved ; due to interruption of reflex arc by disease of sensory muscle-nerves.

2. Diphtheritic neuritis. Frequent loss when there is no subsequent paralysis ; almost constant loss when there is post-diphtheritic paralysis ; comes on about one month after onset of disease ; usually bilateral, rarely unilateral ; return of knee-jerk after variable period.

3. Acute ascending paralysis. Bilateral and early loss very constant ; usually persistent, but knee-jerk may return.

4. Any severe lesion of anterior crural nerve, sensory or motor fibres, or both. Immediate unilateral loss of knee-jerk.

Besides the conditions that have been given as causes of loss of knee-jerk, there are others whose mechanism is at present obscure. Thus, the knee-jerks may be lost during the febrile stage of secondary syphilis and in severe forms of malarial fever,—possibly in both instances from slight neuritis of the sensory muscle-nerves on which the knee-jerk depends ; for it is certain that very slight disease of these nerves may cause temporary loss of myotatic irritability. The knee-jerk may be permanently lost in diabetes mellitus.

It should be remembered that in very rare instances persistent absence of knee-jerk is observed in persons in good health. It is claimed by some that this is not such an uncommon occurrence, but we believe that if the precautions recommended on page 589 be observed in testing the knee-jerk such cases will be found to be very rare.

Thus it is seen that loss of knee-jerk is by no means a pathognomonic symptom. Its chief general significance is interruption of the reflex arc on which it depends. The exact character and seat of the disease which is thus operative must be determined by the study of other symptoms. But it should not be forgotten that interruption of the reflex arc is not the only significance of the symptom.

Excess of Reflex Action is of frequent occurrence in disease of the nervous system, and is evidence for each reflex that its arc is intact. Increase in cutaneous reflex action is of little diagnostic significance, partly because of the variable character of the different skin reflexes in different normal individuals, and the consequent difficulty in determining what constitutes a pathological change. An increase is, however, seen in conditions which, like strychnia poisoning and tetanus, are characterized by an increased irritability of reflex centres generally, and is an inconstant effect of lesions, which, like certain cerebral and spinal diseases involving the pyramidal tract, diminish spinal inhibition. In locomotor ataxia the superficial reflexes are sometimes much increased early in the disease, the plantar increase being particularly noticeable. As the disease progresses upwards, the increase may be seen in the abdominal reflex. Probably this increase is the effect of irritation in the reflex arc.

Increase in the activity of the deep reflexes, particularly the knee-jerk, is a sign of considerable diagnostic importance. It is sometimes due to primary over-action of the reflex centres, as in tetanus and strychnia poisoning. Usually it depends on defective inhibition from higher (cerebral) centres, and this defective inhibition may be brought about by organic or functional disease. Permanent exaggeration of the knee-jerk is almost always

consequent on degeneration of the pyramidal tracts. The excess is at first slight in degree and increases gradually.

Since the conditions that lead to degeneration of the pyramidal tracts are numerous, it is not singular that exaggerated tendon-reflex is seen in a variety of organic processes. The following are the main conditions in which it is observed :

1. Hemiplegia from any cause; tumor, hemorrhage, softening ; increase comes on in about a week ; especially noticeable on side opposite lesion ; may be bilateral ; regular symptom, except in slight cases.

2. General paresis with ataxic paraplegic symptoms ; common and early symptom ; excess bilateral.

3. Transverse myelitis above lumbar enlargement ; bilateral excess usually great.

4. Slow compression of the cord above lumbar enlargement, as from caries, tumor, etc. Early and great excess always present ; may occur when there is no secondary degeneration, and may disappear when compression is removed.

5. Primary degenerative diseases of the cord ; primary lateral sclerosis ; spastic paraplegia ; amyotrophic lateral sclerosis; disseminated sclerosis.

6. Injuries of spinal cord ; hemisection, complete transverse section ; sudden destruction at one level above lumbar enlargement from fracture or dislocation of vertebræ. When injury is one-sided, excess is unilateral and on same side ; excess following fracture or dislocation is preceded by complete loss of reflex if transverse destruction is extensive.

Bilateral increase of the knee-jerks is seen in functional (nutritional) diseases ; in hysteria, hypochondriasis, neurasthenia, epilepsy, and in certain neuroses that follow traumatism (traumatic neuroses).

Increase of other tendon reflexes, triceps, and supinator longus, has the same general significance as increase of patellar tendon reflex, but is of less practical diagnostic value. Very active knee-jerks are frequently seen in perfectly healthy persons.

Ankle Clonus ("foot-clonus") consists of a series of to-and-fro movements (flexion and extension) of the foot at the ankle joint, which may sometimes be induced by suddenly flexing the foot (dorsal flexion) on the extended and relaxed leg. It may also occur spontaneously when the patient sits with the tips of the toes on the ground and the heel raised. The movements are highly rhythmical, and number from six to ten per second. The general significance of ankle clonus is that of exaggerated tendon reflexes, but the phenomenon usually occurs only in conditions where the knee-jerk is very much exaggerated, and true clonus is never a symptom in functional affections.

Ankle clonus is usually evidence of organic disease, especially of degeneration of the pyramidal tract, but it is frequently very marked in cases of compression of the cord in which secondary degeneration has not occurred. A spurious, irregular, and varying clonus is also seen in the paraplegic contracture of hysteria, and in the "traumatic neurosis." Ankle clonus may be obtained in some normal individuals after they have stood on tiptoe for some time. It is very exceptional to meet with ankle clonus without exaggeration of the knee-jerks. But in rare lesions in the sacral cord it may be present when knee-jerks are absent. Clonus of the rectus muscle (rectus clonus) is sometimes obtained by striking the tendon just above the patella. This condition is usually present when there is ankle clonus, but may occur unassociated with it.

Sensory Symptoms are of frequent occurrence in both organic and functional disease of the nervous system, and of the various forms that are encountered loss of sensibility is the most important. Certain forms of sensory loss possess important localizing significance, but many forms are at present of little value, owing to our imperfect knowledge of the sensory paths.

It is important to remember that sensory structures (especially fibres) often preserve their functional activity entirely or in part, when damaged to a degree (for example by pressure) that in motor elements would produce complete loss of function. Hence it is that lesions involving both motor and sensory structures frequently either give rise to no sensory loss, or are soon followed by partial or complete recovery of sensibility.

Different forms of sensory loss (anæsthesia, analgesia) often coexist, but not infrequently certain forms are present while others are lost. It is convenient to consider separately the different forms of sensory loss.

Anæsthesia (the word anæsthesia is also used in a general sense to include loss of pain sense), loss of *tactile* sensibility, is met with in diseases of the brain, spinal cord, and peripheral nerves, and in some nutritional affections (hysteria, traumatic neuroses). Certain types of sensory loss, based on distribution, may be distinguished.

Hemianæsthesia, or loss of tactile sensibility involving one lateral half of the body—face, trunk, and extremities of one side,—is invariably of central origin. The hemianæsthesia may be complete or partial, and may or may not be associated with hemiplegia, which may be of any degree. When of organic origin, complete hemianæsthesia is rarely associated with a high grade of motor paralysis (unilateral). Loss of pain and temperature senses may be associated with hemianæsthesia, but the three forms of sensibility seldom suffer equally. The

anæsthesia usually does not extend quite to the median line (within one or two centimetres), but occasionally it may extend a few cm. on the other side. It involves not only the skin but the mucous membranes as well (lips, conjunctiva, rectum, urethra). Hemianæsthesia of organic origin is always due to interruption of the sensory tract between the junction of the ascending and descending roots of the fifth in the pons (upper extremity of the pons) and the cortex of the opposite side or to disease of the cortex itself. Its exact position must be determined by other symptoms. If the disease is in the pons, the region supplied by the fifth nerve (face) escapes (because the path from the fifth crosses to the opposite side high in the pons), unless the lesion be one involving the entire formatio reticularis in the upper part of the pons. The most common seat of the lesion causing hemianæsthesia is the posterior third of the hinder limb of the internal capsule (see Fig. 27) between the lenticular nucleus of the corpus striatum and the optic thalamus. The sensory tract is here contiguous to the motor path, but owing to the arrangement of the vascular supply of the region it is common for a lesion to injure the former without the latter, or the latter without the former, though an extensive lesion may damage both, and cause both motor and sensory symptoms. The sensory path in the capsule also lies close to the path for the special senses, which may be involved on the same side as tactile sensibility, with the exception of vision, the defect of which is hemianopsia (see page 163). Hemianæsthesia may occur from disease of the cortex itself, or of the subjacent white substance, but complete hemianæsthesia from this cause is very rare. This is because the fibres of the sensory tract are here greatly spread out, to cover a large area, and only a very ex-

tensive lesion can involve them all. Hemianæsthesia is not infrequently of hysterical origin, and it may be one manifestation of certain traumatic neuroses. In both these cases it is probably of cortical origin (nutritional disease). The points of distinction between functional and organic disease are considered in chapter VI.

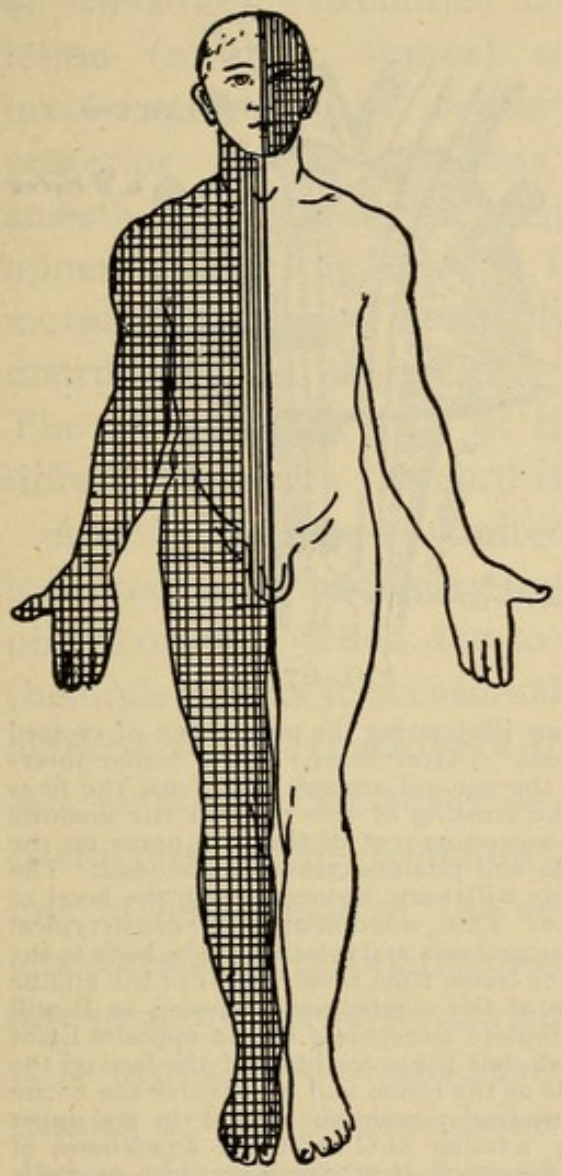


FIG. 66.

Diagram illustrating the distribution of the sensory line in a case of crossed hemianæsthesia. The horizontal lines show the distribution of the analgesia, which was complete above, partial below. The vertical lines show the area of hyperalgesia. In this area, contact with cotton caused a peculiar prickly sensation.

A partial form of hemianæsthesia which is of considerable importance from its frequent occurrence is that which accompanies ordinary hemiplegia. The loss of sensibility does not extend to the median line. It exists chiefly or exclusively in the limbs, the loss being most distinct in the hands and feet. The sensibility to pain is usually diminished in these cases, but not in the same degree as the loss of tactile sensibility.

Crossed Hemianæsthesia —(alternate hemianæsthesia).—Loss of tactile sensibility of one side of the face and of the opposite limbs is of rare occurrence. The sensory loss in the face is due to damage of the ascending root of the fifth

nerve of the *same* side in some part of its course; the loss on the trunk and limbs to damage of the sensory tract from these parts of the opposite side. In the pons (below the upper third), and in the medulla, these tracts lie close together, hence a lesion in the pons or upper part of the medulla involving the entire lateral extent of the formatio reticularis and the fifth nerve-root gives rise to crossed hemianæsthesia. (See Fig. 67). Crossed hemianæsthesia is never of functional origin.

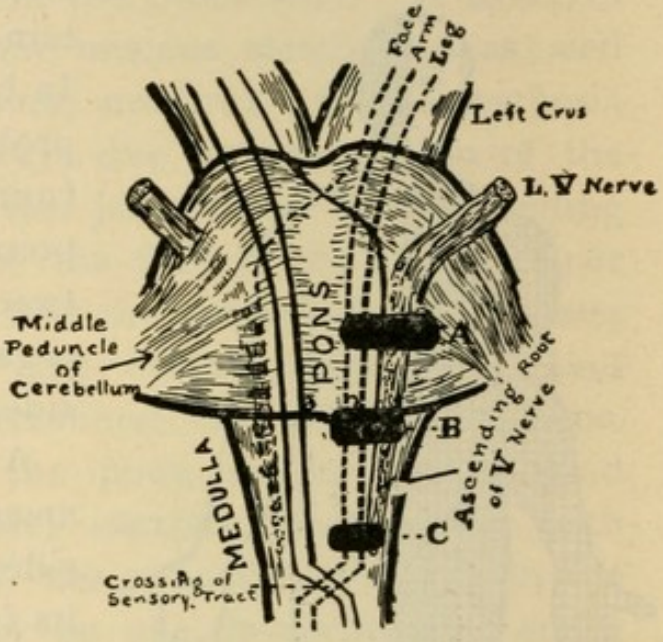


FIG. 67.

Diagram illustrating the mechanism of crossed anæsthesia. (After Starr.) Any lesion interrupting the general sensory tract from the lines above the crossing of this tract in the medulla and the ascending root of the fifth nerve on the same side will produce crossed anæsthesia. The conditions will vary, however, with the level of the lesion. Thus, a lesion at A will cause typical crossed anæsthesia and rotation of the body to the side of the lesion from involvement of the middle peduncle of the cerebellum; a lesion at B will cause complete anæsthesia of the opposite limbs and trunk, but the anæsthesia of the face on the same side as the lesion will not involve the entire face, sensation remaining normal in its upper portion; a lesion at C will cause anæsthesia of entire opposite half of the body, but *probably* only partial and greatly limited anæsthesia of the face on the same side as the lesion.

Bilateral Anæsthesia, involving the limbs on both sides, may occur as a consequence of disease of the pons damaging the sensory tract on both sides. The anæsthesia is seldom complete.

Para-Anæsthesia.—Anæsthesia involving both extremities ("anæsthesia of paraplegic distribution" it is sometimes called) is generally dependent upon disease of the spinal cord (but not infrequently on peripheral neuritis), sometimes upon disease of the cauda equina,

though in this case the anæsthesia usually involves the skin on the lower extremities irregularly. Anæsthesia of the lower extremities may be due to a transverse lesion (myelitis, tumor) or to a systematic lesion involving only the sensory tract of the cord, the posterior median columns. The upper level of the anæsthesia affords an important indication of the upper level of the lesion, a more accurate one than the motor palsy, which generally coexists, but whose exact distribution and degree it is often difficult to determine. The exact upper limit of the anæsthesia in lesions of different levels in the cord is given on page 438.

Anæsthesia may be limited to one extremity, and such localized anæsthesia may be of cerebral, spinal, or peripheral origin. When due to organic disease of the brain (hemiplegia), as it occasionally is, the anæsthesia is most marked at the extremity of the limb, and grows gradually less marked as the trunk is reached—that is, it has the characters of the common form of anæsthesia that is met in hemiplegia of the severe or grave type. If the anæsthesia corresponds closely to the area supplied by one or more nerve-trunks and there is no cincture pain, it is probable that the lesion is in one or more nerve-trunks, or the plexus above them. When there is evidence of a segmental loss of sensibility (inner border of arm, forearm, and hand, for example) and there is a girdle-pain or sensation, the lesion may be referred to a corresponding segment or nerve-root of the spinal cord (especially the arm). It is very important to know that anæsthesia of one extremity, with a sharply limited upper border and of the severe or grave type, is frequently of hysterical origin.

Anæsthesia limited to well defined areas of the extremities or trunk, and which corresponds to the distribution of

one or more nerves or nerve branches, generally indicates disease of the nerve itself,—more rarely disease of the nerve-roots. In studying the distribution of the sensory loss from lesions of mixed nerves, it is helpful to bear in mind Schroeder van der Kolk's law, that the sensory fibres of a mixed nerve supply, generally speaking, the integument over the muscles innervated by the motor fibres of the same nerve.

Irregular areas of anæsthesia, of indeterminate nerve supply, are not infrequently met with in the trunk or limbs. Such patches may be due to disease of the sensory nerve-endings in the skin, to the irregular incidence of disease in the peripheral nerves, to disease of the nerve-roots, to systematic disease of the cord (posterior sclerosis), or to the occurrence of irregular islets of sclerosis in the cord (multiple sclerosis), and possibly also in the brain. Perhaps the most frequent structural cause of anæsthetic patches is disease involving several nerve-roots, as spinal meningitis. Usually the localization of the process causing the anæsthesia is easily accomplished with the help of other symptoms. It is to be remembered that irregular areas of anæsthesia are not infrequently met with in hysteria, and are sometimes seen in neurasthenia and alcoholism.

Very rarely cases are observed in which there is general loss of tactile and pain sense, associated with tremor, increased reflexes and mental depression, and in which the autopsy gives no explanation of the phenomena.

Analgesia.—Loss of sensibility to pain may occur with or without loss of tactile sensibility. In the spinal cord and medulla we know the paths for these two forms of sensibility to be distinct, and this explains

the frequency with which one form of sensibility suffers without, or in less degree than, the other, from disease in these situations. In the brain we know little as regards these paths. They probably lie close together, and both forms of sensibility usually suffer together, though one, usually tactile sense, is generally more affected than the other. In disease of the peripheral nerves and nerve-roots, also, both forms generally suffer, though not necessarily in equal degree. Where the two paths lie adjacent, as in the nerves and nerve-roots, a slight grade of damage frequently arrests the conduction of tactile sensation, while painful impressions continue to be transmitted. This probably depends on the greater molecular disturbance created in the nerve path by painful stimuli.

What was said of the varieties of anæsthesia in their bearing on the position of the lesion applies in general to analgesia. As regards the spinal cord the indications of the level of the lesion are the same in disturbances of tactile and pain sense, though the indications as to the horizontal position of the lesion are different. Analgesia may be of functional origin. In this connection may be mentioned the delay in the transmission of sensory impulses (as pain), which is seen in some forms of spinal-cord disease (notably in tabes, and in compression of the cord). The delay may amount to five, ten, twenty, or even sixty seconds. When marked, such delay is evidence of organic disease (usually of the cord). It must be carefully distinguished from the apparent delay which is seen in psychic conditions (dementia, delusional states).

Loss of Temperature Sense is generally observed to be associated with loss of sensibility to pain, but frequently both forms of sensibility do not suffer in equal

degree, and one may be involved without the other. The character of the loss varies in different instances. Sometimes there is absolute inability to recognize either heat or cold. In other cases considerable degrees of heat and cold are recognized, while slighter degrees are not perceived. The sensibility to heat and to cold is not always affected in the same degree (this being due in all probability to the fact that the conduction of heat and cold are subserved by different nerve-fibres). As regards the topographical distribution of loss of sensibility to pain in disease, the same may be said, in general, that was stated in regard to anæsthesia.

In the brain, nerve-roots, and peripheral nerves—that is, wherever the sensory paths for touch, pain, and temperature lie close together, all three forms of sensibility are apt to suffer together in disease, and have the same general distribution. But in the spinal cord and medulla the tracts for touch are separated from those for pain and temperature (which probably lie close together) by such an interval (p. 33), that the former is readily affected without the latter, and *vice versa*. Some observers place the paths for pain and temperature in the antero-lateral ascending tracts of the cord. Others believe that the central gray matter of the cord is concerned, at least in part, with the upward transmission of these forms of sensibility. In the peculiar condition of the cord, known as syringomyelia, there is observed loss of pain and temperature sense, of one extremity or of one side of the body, with little or no disturbance of tactile sensibility. Focal processes and injury of the cord may produce the same effect.

Loss of Muscular Sensibility is observed when the path which conducts impressions from the muscles be-

comes the seat of disease. As already stated (p. 43), this path, after reaching the posterior nerve-roots, with other sensory fibres, probably passes up the cord in the posterior median column and direct cerebellar tract, without undergoing decussation. After reaching the post-pyramidal nuclei, the tract decussates in the interolivary tract, and passes by the fillet to the level of the posterior corpora quadrigemina, whence fibres probably pass to the superior parietal lobule of the cortex. The direct cerebellar tract goes to the cerebellum. Practically loss of muscular sense is more frequently observed in disease affecting the peripheral nerves (multiple neuritis) and the posterior nerve-roots (tabes) than in disease nearer the brain. Yet it is met with in marked degree in unilateral and transverse lesions of the cord. In unilateral disease of the cord the loss of muscular sense exists only *on the side of the lesion*, that is, on the side of the motor loss, and opposite the general sensory loss. The muscular sense may also be lost in consequence of disease confined to the interolivary tract in the medulla, and the sense of posture of a limb during active and passive movement, which is dependent in part on the muscular sense (cutaneous and joint sensations being the other factors), may be much impaired or lost from disease of the cortex, and probably also of the central ganglia. There is every reason to believe that the sense of posture is regularly impaired in the limbs of the opposite side whenever there is destructive disease of the superior parietal lobule.

Hyperæsthesia and Hyperalgesia.—Increased sensibility to pain is designated hyperalgesia, or, less correctly, hyperæsthesia. Probably there is no cutaneous hyperæsthesia in the sense of increased tactile sensibility, with recognition of two points of the æsthesiometer

at smaller distances than normal. Sometimes a light touch on the skin causes pain, or an unpleasant sensation, but this is probably due to irritation of over sensitive end-organs which subserve common sensibility (pain), and not to increased tactile acuteness. This increased sensitiveness is frequently due to irritation of sensory fibres in some part of their course—peripheral, spinal, or cerebral. Probably the irritation is operative by exalting the sensory impression as it passes, but it may also act by increasing the irritability of the cortical sensory centres. The distribution of the sensory disturbance is sometimes of service in localizing the position of the lesion, and the same general statements are applicable that have been mentioned in connection with anæsthesia. Increased cutaneous sensibility is observed in a variety of processes—as meningitis, cerebral and spinal tetanus, hydrophobia, hysteria, traumatic neuroses, etc. It is a common and important symptom of neuritis and multiple neuritis. When tactile or painful stimuli give rise to perverted forms of sensation, as numbness, tingling, or formication, these are designated paræsthesiæ. Paræsthesiæ may have a purely subjective origin, or they may be of cerebral, spinal, or peripheral origin.

Pain, another expression of sensory irritation, is an important symptom of organic disease of the nervous system. It is, however, so common in functional affections, that many varieties of pain are in themselves of little diagnostic importance. Severe recurrent limb pains may arise from any form of nervous disease which involves the sensory tract and partially, but only partially, destroys it. As examples, may be mentioned the pains of multiple neuritis from neural irritation, of spinal meningitis, spinal tumor, spinal compression, and locomotor ataxia, from irritation of the posterior nerve-

roots, of myelitis from irritation of sensory structures within the cord, of cerebral hemorrhage, softening, or tumor involving the sensory tract in the internal capsule, and of irritative processes in the motor cortex. Pain in the distribution of the fifth nerve is an important and early symptom of disease of the nucleus or trunk of the nerve. The pains that result from irritation of the nerve-roots by vertebral disease are generally increased by movement. This is often marked in vertebral cancer and caries.

Pain Referred to the Spinal Column is not infrequently observed in disease involving the vertebræ and the dura mater. Occasionally it is met with in spinal-cord disease of long standing, as myelitis. It is then often referred to the parts in the vicinity of the spine, as the sacrum and loins, as well as to the spine itself. Its mechanism and exact significance are not known. Where there is organic disease of the bones, pain, often severe, is a persistent symptom, and is usually associated with local tenderness, but the same association of spontaneous pain and tenderness to pressure is seen in the purely neuralgic affection of the spine known as "rachialgia."

Sometimes the pains of chronic spinal-cord disease, and of acute processes, are not acute, but dull and persistent. These pains are often regarded as rheumatic, particularly when exacerbations occur, as they frequently do in chronic disease, during wet or cold weather. Persistent or rheumatoid pains in the limbs should always lead to a search for more unequivocal evidences of spinal-cord disease—such as slight localized loss of power, anæsthesia, or altered reflex action.

Cincture Pain (girdle-pain) is a persistent sense of painful constriction, as of a band encircling the body, the limbs, or the neck. It is most frequently observed

in cases where the cord is damaged transversely, as in myelitis, and fracture or dislocation of the vertebræ. The pain is due to the irritation of sensory structures just above the seat of the lesion—that is, in the lowest part of the healthy cord, and is referred, as is the rule, to the peripheral sensory nerve-endings. It possesses considerable diagnostic significance, for it is not only evidence of organic disease, but indicates the upper level of the lesion in the cord. A cincture-pain also occurs in locomotor ataxia, but is far less frequently observed than in myelitis.

Muscular Tenderness (muscle pain sense) is a symptom which, from its constancy in some diseases of the peripheral nerves (multiple neuritis) and muscles (polymyositis), is of diagnostic importance. In polymyositis the muscles are exceedingly painful on pressure, though there may be little spontaneous pain, except on movement. In multiple neuritis there are neuritic pains and tenderness of the nerve-trunks, but the sensitiveness of the muscles to pressure is even more constant. The occurrence of muscular tenderness in trichiniasis must be remembered in this connection. It is usually associated with considerable spontaneous pain.

Headache.—The head is very frequently the seat of pain. In a large majority of cases this pain is the expression of functional derangement, which is of widely different character in different cases. The headaches which result from gastric disturbance, anæmia, febrile conditions, renal disease, various forms of toxæmia and errors of refraction, probably constitute more than ninety-five per cent. of all the headaches met with in general practice. But organic disease within the cranial cavity is also a frequent cause of headache, and the importance of distinguishing between functional dis-

turbance and organic intracranial processes makes it necessary to study carefully the character of every case of headache.

The pain of organic disease is usually constant. It varies in degree from time to time, but the patient is seldom entirely free from it. The severity of the pain is also a suggestive feature. In a considerable proportion of cases the pain is at times intensely severe. It persists through the night, and either prevents the patient from sleeping or awakens him from his sleep. This is an important diagnostic point, for the headache of functional disease rarely prevents sleep. Whenever, therefore, a patient complains that his headache frequently keeps him awake during the night, the possibility of organic brain disease should be thought of, and other evidences of such disease should be sought. But it must not be forgotten that in point of severity alone some functional headaches, as neuralgia and migraine, resemble closely the pain of organic diseases. Their paroxysmal character and short duration make an error in diagnosis impossible.

The pain of organic disease is increased by anything that leads to increased vascular tension within the cranium, as stooping or coughing, but this is not a distinctive feature of such disease. The headache of organic disease is sometimes diffuse, sometimes frontal or occipital, or both; sometimes it is distinctly one-sided, and occasionally it is sharply localized. Pain which is very limited in extent is more apt to be functional than organic in origin. Pain that is one-sided is seldom due to disease of an opposite region of the brain, but it is never possible to say, from the headache alone, whether there is a close correspondence between the seat of pain and the seat of the lesion. If the disease is at the surface of the brain, or in the

meninges, or very near the surface, there is probably a close correspondence between the two. When the disease is thus superficial there is often local tenderness on light percussion over the seat of the lesion. Sometimes no correspondence exists between the seat of the headache and the lesion—for example, the headache is sometimes frontal when the lesion is in the cerebellum.

The intracranial processes which give rise to headache are generally irritative and progressive in character, as, for example, tumor, abscess, and the different varieties of meningitis. Nevertheless, little or nothing is known as to the mechanism by which these processes give rise to pain ; even the structures which are the seat of pain cannot be positively designated. The dura mater receives sensory nerve filaments and may unquestionably be the seat of pain when the membrane is the seat of disease, and probably also when it is not the seat of appreciable structural change. The brain of a highly organized animal may be mutilated without giving rise to evidences of pain, but neither this fact nor the circumstance that sensory fibres have not yet been discovered in the pia-mater or the cerebral substance can be regarded as showing that those structures are insensible to pain under conditions of disease. It has been supposed that increased intracranial pressure is operative in the production of headache in certain diseases, but while this may be a factor in some cases, it has been shown that processes which, like internal hydrocephalus, increase the intracranial pressure as much as it is ever increased, but cause no tissue irritation, often run their course without causing headache.

We have seen that the constancy and severity of a headache may be suggestive, perhaps highly suggestive, of intracranial disease. Yet these characteristics of a

headache never *prove* the existence of organic disease. Actual proof can be supplied only by the association with headache of other evidences of disease. Of these optic neuritis is the most significant. But double optic neuritis and headache occur as symptoms of certain constitutional states, notably lead poisoning, chronic diffuse nephritis, and a high grade of anæmia. If we can exclude these conditions, we may regard the occurrence of marked double optic neuritis with headache as evidence of some form of organic intracranial disease.

It is hardly necessary to say that neither the absence of headache nor its slight character in any particular case renders it possible to exclude organic disease. In the presence of symptoms of doubtful character, the absence of headache is of some significance in lessening the probability of the existence of organic disease, but in the presence of unequivocal symptoms, such as local paralysis or convulsions, its absence does not materially modify the diagnosis.

Cephalic Sensations Other than Pain are frequently complained of by patients who are hypochondriacal or neurasthenic, or whose brains are overworked. The sensations are variously described. Very frequently there is a sense of pressure, slight or great, as if the top of the skull were being forced in. Sometimes the patient feels as if the sides of the head were being forced in, but generally the sensation is referred to the vertex. A feeling of expansion, as if the top of the skull were being lifted, is often complained of. Another and more common sensation, described chiefly by neurasthenics, is a crackling feeling within the skull as if something had broken, followed often by a curious sense of emptiness or lightness of the head. Sometimes there is simply a sense of fulness of the head or a sense of throb-

bing. These sensations and many more are very often described in extravagant language. They are seldom constant, and are always functional. They are generally aggravated by attention. We are in ignorance as to how these sensations are produced, but there is no reason for attributing them to cerebral congestion, or to congestion at the base of the brain.

Symptoms Referable to the Special Senses.

Olfactory Symptoms.—Anosmia, loss of smell, is much more frequently the result of disease of the olfactory mucous membrane (chronic inflammation, polypi), than of intracranial disease, and such local disease must be excluded before the symptom can be considered significant of brain disease. Blows on the head have been known to cause anosmia by mechanically tearing the olfactory filaments from the bulb, but this is an infrequent occurrence. Another rather infrequent cause of anosmia is pressure on the nerve or bulb by tumor in the anterior fossa of the skull, or by bone disease. Meningitis may also involve the nerve, and it has been compressed in internal hydrocephalus. Rarely anosmia occurs in degenerative diseases of the nervous system, as locomotor ataxia, and dementia paralytica. Disease in the sensory part of the internal capsule (posterior limb) has caused anosmia on the side opposite the lesion. An extensive cortical or subcortical lesion occasionally produces the same effect. Disease of the tip of the temporo-sphenoidal lobe, involving the olfactory centre, may unquestionably give rise to anosmia on the same side as the lesion. Functional loss of smell may occur in hysteria. It is always associated with other forms of sensory loss, hemianæsthesia, crossed amblyopia, loss of taste, etc. In attaching significance to the presence

of anosmia, it must be remembered that it is sometimes congenital (due to congenital absence of the bulbs), and that it may result indirectly from disease of the trigeminus, through trophic changes in the olfactory mucous membrane, or from defective local secretion.

Hyperosmia or Olfactory Hyperæsthesia is occasionally observed in hysteria. It consists in the development of great acuteness in the sense of smell, so that odors are perceived which are ordinarily not recognized. The symptom has little diagnostic significance.

Olfactory Hallucinations.—Subjective sensations of smell may be of functional or organic origin. In insanity they are not very uncommon. The epileptic paroxysm is sometimes ushered in with an olfactory aura, usually unpleasant in character. In several instances the development of gross organic disease (tumor) of the anterior part of the temporo-sphenoidal lobe has been accompanied by olfactory auræ. An aura may also result from disease involving the olfactory tract. The repeated occurrence of such an aura is of great diagnostic significance in the presence of evidence of organic disease, for it gives a clue to the position of the lesion, sometimes when other indications are wanting.

Visual Symptoms.—*Amblyopia* and *Amaurosis* are terms used to designate different degrees of visual defect, the former meaning a partial loss or blurring of sight, the latter complete blindness. Complete blindness (of nervous origin) of one eye, indicates that the optic nerve is damaged between that eye and the chiasma—that is, in the orbit, at the optic foramen, or within the skull between the foramen and the chiasm. Such damage may result from pressure by tumors, from basal disease, from inflammation, and from embolism of the central artery of the retina, etc., but in these cases the blindness is not always absolute.

Frequently there is concentric limitation of the field, and sometimes there are regular defects. Amblyopia of one eye may also result from disease of considerable extent in the cortex of the opposite hemisphere, involving the lower and posterior part of the parietal lobe (angular and supra-marginal convolutions). There is usually considerable limitation of the visual field in such cases, and though the patient does not notice any defect in the vision of the other eye, examination usually reveals a slight concentric restriction of the field of that side. Hemianæsthesia is also frequently observed in these cases, and its occurrence helps in the distinction of crossed amblyopia from the amblyopia of optic-nerve disease. Other points of distinction are as follows: (1.) the pupillary reaction is diminished in disease of the optic nerve, but is normal in cortical disease; (2.) The motor nerves of the eyeball are frequently involved in optic-nerve disease, never in disease of the parietal cortex. Functional loss of vision of one eye only is exceedingly rare, but may result in a reflex manner from irritation of the fifth nerve, and, rarely, from hysteria. In hysterical amblyopia, which is almost always unilateral, vision may amount to complete blindness. The fields are contracted concentrically, and since the retina exhausts rapidly, the limitation may be more marked with each succeeding test at the one examination. A functional amblyopia affecting both eyes, as in organic crossed amblyopia, and associated with hemianæsthesia, is sometimes observed in hysteria. This functional form is much more frequent than the organic form, though it is probably not as common as has been supposed.

Bilateral loss of vision, partial or complete, is infrequently observed in the course of organic disease of the nervous system, as the result of optic neuritis or optic atrophy, primary or post-neuritic. The relation of these

processes to intracranial disease will be discussed elsewhere. It is important to bear in mind the fact that a considerable error in refraction often gives rise to amblyopia, and that such an affection must be excluded before we can attribute the visual defect to disease of the optic nerve.

Defective Central Vision sometimes results from irregular patches (scotomata) of retinal anæsthesia involving the fixing point (macula lutea), and extending a very little distance about it. Such central scotomata tend to be symmetrical in situation, though the vision of one eye may be more impaired than that of the other. The visual loss is seldom absolute, even in the portion of the field where it is most decided, and the loss for colors is usually more extensive than that for white light. This form of amblyopia is met with in those who use tobacco to excess ("tobacco amblyopia"), and occasionally occurs from the excessive use of alcohol. It is generally a transient affection, and for this reason is often classed with functional amblyopia, but it is really due to an inflammation of the central fibres of the optic nerve (axial neuritis).

Temporary amblyopia, involving both eyes, occurs in certain functional diseases of the brain, notably epilepsy and migraine, but there is little danger of confounding this with amblyopia of organic origin, as the loss of vision is not only transient but distinctly paroxysmal in character. In epilepsy the loss of sight usually just precedes loss of consciousness. In migraine the defect of vision is always partial, and may consist in general blurring of sight, or in unilateral defect in each eye—hemianopsia.

Functional amblyopia of one eye from irritation of the fifth nerve has been alluded to. But such reflex amblyopia is more often bilateral than one-sided, though

the loss of sight is frequently most marked on the side of the nerve which is the seat of irritation. The pain of neuralgia may be effective in producing reflex amblyopia ; sometimes the cause is the irritation of a carious tooth, generally a molar. How such causes are effective is not definitely known. Probably the arrest of visual function is the result of inhibition exerted on the nervous elements of the retina, or on the visual centres of the cortex.

Bilateral loss of sight, usually of short duration, but often complete in degree, is sometimes met with as a consequence of toxic blood-states—"toxic amblyopia" it is called. Uræmic poisoning is the most frequent cause of such blindness (which is frequently of sudden onset). It may also occur in malaria, which disappears on exhibiting full doses of quinine. Oddly enough, a similar defect also occurs in poisoning by quinine. Lead occasionally produces similar results. There are usually no ophthalmoscopic changes to which the blindness in these cases can be ascribed.

Concentric Limitation of the Visual Field, both for white light and for colors, is observed in many of the affections that have been mentioned as causes of amblyopia. If we exclude those cases in which the limitation is the result of structural change in the optic nerve (neuritis, atrophy) there remain a number of conditions of the nervous system, both functional and organic, in which this limitation is of frequent occurrence. Of these conditions the following are of especial importance : crossed amblyopia of organic origin, crossed amblyopia of hysterical origin, crossed amblyopia occurring in the course of the traumatic neuroses, the amblyopia of epilepsy and of neurasthenia, and the amblyopia of multiple sclerosis. The acuity of central vision may be impaired in any of these conditions, but in some of them (*e. g.*, the traumatic neuroses) it is frequently normal.

Hemianopsia.[1]—Hemianopsia is loss of vision in one lateral or vertical half of the visual field. It almost always involves both visual fields, but partial damage to one optic nerve occasionally gives rise to a unilateral hemianopsia. In bilateral hemianopsia either one horizontal or vertical half of each field may be obscured. A horizontal defect is, however, exceedingly rare. Vertical hemianopsia, on the contrary, is not very infrequent, and constitutes a symptom of the greatest importance for the localization of cerebral lesions. Several varieties of bilateral vertical hemianopsia (which is always due to an intracranial lesion) must be distinguished.

A.—Temporal or Bi-Temporal Hemianopsia, is that variety in which the temporal half of each field is obscured. It therefore represents a defect of the nasal half of each retina, which can result only from disease of the central portion of the optic chiasm, involving the decussating fibres of each retina. Often the process (tumor or inflammation) which gives rise to temporal hemianopsia extends laterally, and involves the non-decussating fibres of one side of the chiasma, thus causing total blindness of the corresponding eye, or, if both sides of the tract are involved, of both eyes. In some cases of temporal hemianopsia from syphilis at the base, a rapid and frequent variation of the dark half-fields has been noted (so called "oscillating bi-temporal hemianopsia"). It is probably of some diagnostic importance, as evidence of basal syphilis (gumma, chronic meningitis), but cannot be regarded as pathognomonic.

[1] The word hemiopia is properly used to designate loss of sight in one half of the retina. A left hemiopia is therefore equivalent to a right hemianopsia, and, conversely, a right hemiopia to a left hemianopsia. The word hemianopia is used synonymously with hemianopsia. Lateral hemianopsia is also known as homonymous hemianopsia.

B.—Nasal Hemianopsia.—Obscuration or the nasal half of each field from anæsthesia of the temporal half of each retina is not only the rarest form of hemianopsia but is the rarest of all visual defects, since it can result only from a lesion which involves each side of the optic chiasm without damaging the central part. In one case, such hemianopsia was produced by the pressure of calcified and enlarged carotid arteries on the sides of the chiasm.

C.—Lateral, or Homonymous Hemianopsia, is that form of hemianopsia in which corresponding visual fields are obscured. Thus, in *right* lateral hemianopsia *both right half-fields* are blind. In all cases of lateral hemianopsia the lesion is *posterior to* the optic chiasm. It may be in one optic tract, in one of the primary optic centres (corpora geniculata externa), in the pulvinar of the thalamus, in the sensory tract of the internal capsule, in the optic radiation, in the occipital lobe, or in the half-field centre of the cortex (cuneus). In whichever one of the enumerated positions the lesion lies, it is on the side *opposite* the half-fields obscured. A lesion of the angular gyrus may also cause lateral hemianopsia, but probably it can do so only by injuring the immediately underlying optic radiation. The determination of the position of the lesion in the optic apparatus is based on the associations of the hemianopsia. The extent of the half loss of the visual fields varies in different instances. The line of division between the obscured and seeing halves may pass through the fixing point (corresponding to the macula lutea), or may pass just to one side of it, leaving it within the region of sight. These are the typical forms of hemianopsia. In other instances the line of division is irregular, or inclines obliquely to one side or the other above and below the fixing point. These variations do not depend on the position of the

lesion causing the hemianopsia, but on individual peculiarities in the decussation in the optic nerves. Certain other variations, however, are related to the situation of the lesion. In the first place, hemianopsia may be incomplete, a portion only of the half-fields being obscured. The area of defective vision is in these cases sector-like, being frequently irregularly quadrantic in outline. This incomplete loss is probably dependent on partial destruction of the visual path, or half-vision centre, most frequently the latter. Secondly, lateral hemianopsia may be associated with concentric limitation of the half-fields of vision that remain. This limitation of the remaining fields is always greatest in the eye of the side *opposite* the lesion. It probably indicates that the lesion involves not only the half-vision centre of the cortex, or the optic fasciculus in the occipital lobe, but also the higher visual apparatus (see page 50), which is supposed to lie in and beneath the inferior parietal lobe. In about one half of the cases of hemianopsia the symptom is associated with transient or permanent hemiplegia of organic origin, both symptoms being effects of the same lesion. The hemiplegia is on the side of the loss of vision ; the patient "cannot see his paralyzed side." Complete loss of sight in *both* eyes occasionally results from *double* lateral hemianopsia. It depends on the occurrence of two lesions, one in each hemisphere, involving the visual path posterior to the chiasm.

Lateral hemianopsia which has been complete for several months, seldom passes away entirely. It may remain, with little change, during many years ; in one case it is known to have endured twenty-three years. It is important to know that hemianopsia may exist for a considerable time without being noticed by the patient.

Lateral hemianopsia is not a very uncommon symptom of migraine. It is of short duration, and often the loss

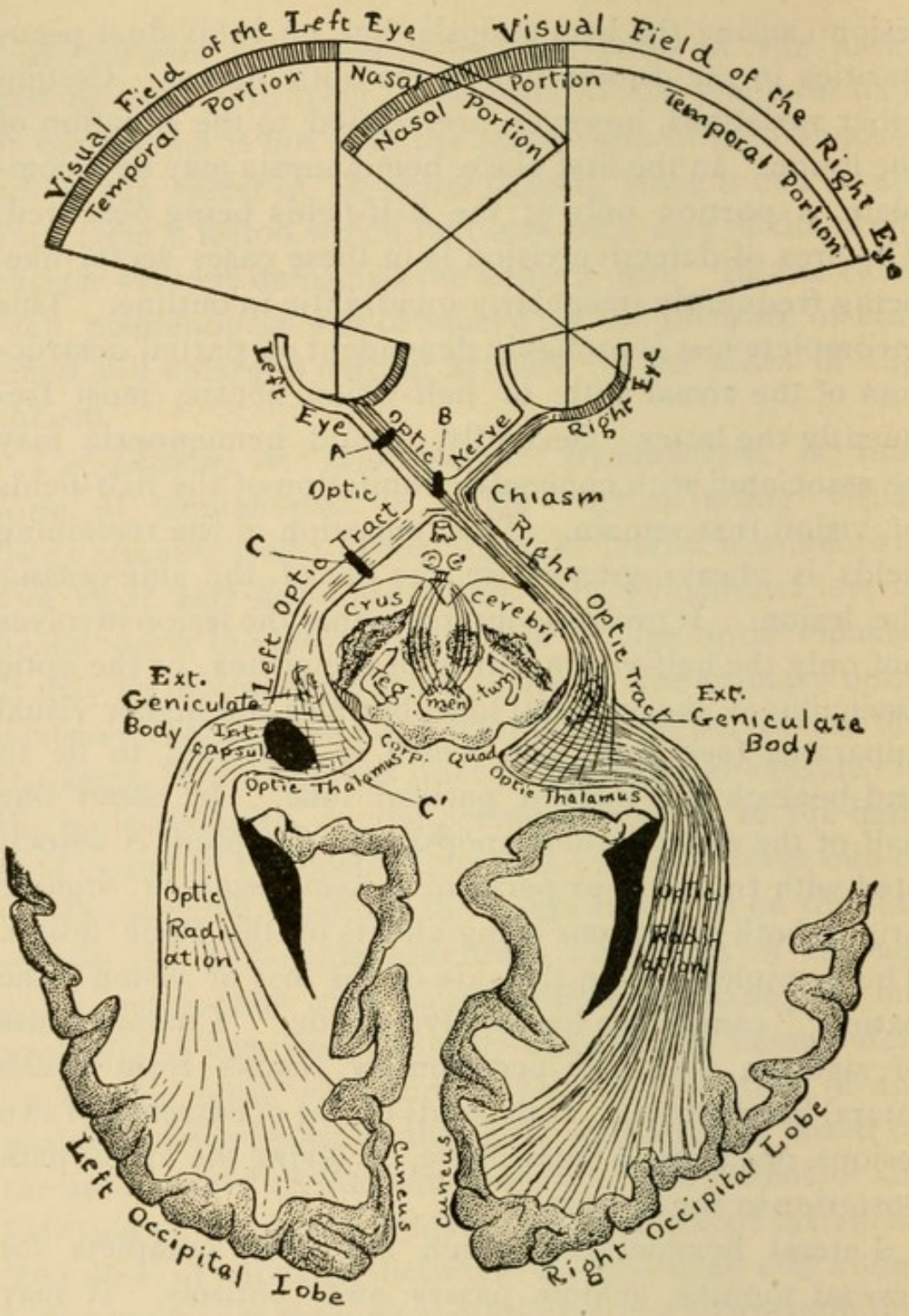


FIG. 68.

Diagram illustrating the course of the optic and visual tracts. (After Starr.)
A, lesion of left optic nerve, causing total blindness in left eye; B, lesion causing bilateral temporal hemianopsia; C, C′, lesions in the optic or visual path POSTERIOR to the chiasm. All such lesions cause bilateral homonymous hemianopsia, but give rise to different associated symptoms according to the position of the lesion. Thus, a lesion in the int. capsule, as at C′, causes also hemiplegia of the opposite side of the body.
The rest of the diagram requires no explanation.

involves the right-half fields in one attack, and the left-half fields in another. Not infrequently other forms of visual disturbance occur during other attacks. It is well to bear in mind the fact that the hemianopsia of migraine may occur as an isolated symptom, though it is usually associated with some degree of headache and gastric disturbance. It is probable that migraine is the chief functional affection in which hemianopsia occurs. It is exceedingly probable that the symptom is only rarely of hysterical origin. Transient hemianopsia is frequently observed during the onset of cerebral hemorrhage, but in this case it is probably an indirect symptom, due to inhibition of the cortical centre by irritation. In almost all cases of hemianopsia the loss of vision for white light is associated with a corresponding loss for colors. It occasionally happens, however, that there is hemianopsia for colors—"hemi-achromatopsia," it is called—when there is no loss whatever for white. In these cases an object, when moved across the field, is seen in all positions, but becomes uniformly gray when moved beyond the vertical line of separation between the half-fields. The explanation of this remarkable phenomenon must lie in the existence of a distinct centre for color vision, adjacent, in all probability, to the half-vision centre in the cuneus.

Symptoms referable to *irritation of the optic apparatus* occasionally appear in the course of organic processes which eventually give rise to atrophy or inflammation of the optic nerve, but such phenomena are of much more frequent occurrence in functional and nutritional disturbances of the nervous system. Muscæ volitantes, sparks, etc., not infrequently appear to pass before the eyes of neurasthenic and hysterical patients. A zigzag appearance, described as the "fortification spectrum," is often seen in migraine, but is not met with in any

other neurosis, except in rare cases of epilepsy. It usually lasts many minutes. Sparks, flashes of light, colors before the eyes, and other irritative symptoms related to the visual apparatus occur in migraine, and also in epilepsy. In epilepsy a visual aura is not uncommon; indeed, such an aura is more frequent in epilepsy than all other warnings of the special senses combined. Sometimes these impressions are very elaborate, and may represent a complex perception, as of a human face; usually they consist of crude sensations, such as luminous flashes, etc. Rarely a visual aura is a precursor of convulsions due to organic cerebral disease. In most cases of this kind the lesion has been located in the cortex or white substance of the occipital lobe.

Ophthalmoscopic Changes.—Pathological changes in the fundus of the eye, and particularly in the optic nerve, are of frequent occurrence in the history of structural disease of the nervous system. Many of these alterations in the optic nerve may be carefully studied during life with the aid of the ophthalmoscope, for in the optic disc we have presented to direct view the termination of the optic nerve. The information thus obtained is of the highest importance in diagnosis, and renders a knowledge of ophthalmoscopic technique indispensable to the student of nervous diseases. Of the changes which are observed, some are the direct consequence of the cerebral or spinal cord lesion—that is, they are *consecutive* to the process in the nervous system; others are not related directly to the morbid process in the nervous system, but to the ultimate cause of this process—that is, they stand in the same relation to this cause as does the process within the nervous system, and are hence denominated *associated* changes. The consecutive changes are optic neuritis (or inflammation of the optic nerve) and some forms of atrophy of the optic nerve.

In *Optic Neuritis* the optic nerve may be inflamed throughout its entire extent, or the inflammatory process may be most marked in one particular part of its course. When the inflammation is most intense behind the eyeball (retro-bulbar neuritis), the visible signs of neuritis may be slight or absent, though there is considerable loss of vision. When the inflammation is most marked as it generally is, at the optic papilla within the globe, there are always distinctive ophthalmoscopic signs (papillitis). At first the disc becomes slightly swollen, and its edge, which is normally sharply defined, grows less distinct. This loss of distinctness or "softening" of the edge of the disc is the important characteristic of beginning papillitis, the swelling of the disc being at first only very slight and causing little appreciable projection of the papilla. The nasal side of the disc (which possesses more nerve-fibres than the temporal side) is generally the first to grow indistinct; later the blurring involves the entire circumference of the papilla. The normal white central depression (physiological cup) is soon obliterated, and the whole disc, which at first shows only slightly increased vascularity, assumes a full red tint, or a grayish opalescence. On direct examination (p. 605) the disc has a distinctly striated appearance, the striæ radiating from the centre of the disc and corresponding to the course of the nerve-fibres. Early in the process the vessels show little change; when the swelling is marked the veins become wider and tortuous, and may be lost as they pass over the edge of the disc. The arteries may remain normal in size; frequently they are contracted. They may be partially hidden from view by the opacity. As the papilla grows more and more prominent the breadth increases, until it may attain twice the diameter of a normal papilla. The prominence of the

disc is so great in some cases that it can be distinctly seen with a 7 D convex lens. A gradual subsidence of these changes takes place after a few weeks or months. The opacity of the disc slowly disappears and the outlines of the papilla again become recognizable, although the disc may continue for some time to look hazy ("woolly disc"). In slight degrees of neuritis the disc may regain its normal appearance. In more severe cases the papilla grows white or pale, and the arteries continue narrow or grow even more contracted; "consecutive" or "post-neuritic" atrophy sets in.

The inflammatory changes which are seen with the aid of the microscope in the papilla can often be observed, though in less degree, throughout the course of the optic nerve, and may be traced through the chiasma. Immediately behind the eyeball the space between the inner and outer sheath of the nerve is generally distended with fluid inflammatory products.

An intense grade of optic neuritis (papillitis) always gives rise to impaired vision. The acuity of sight is diminished and the field of vision becomes restricted both for white and for colors. Sometimes the impairment of vision is very great, particularly for green and red, but not infrequently it is inconsiderable, even with a high grade of neuritis. The grade of visual disturbance is often greater during the subsidence of the neuritis than during the period of most intense congestion. In optic neuritis of slight or moderate intensity there may be a corresponding degree of visual defect, but it is an important fact that even a considerable grade of neuritis frequently causes no appreciable disturbance of sight. We can never exclude optic neuritis because a patient has a high grade of visual acuity.

Optic neuritis may occur in consequence of several

different intracranial processes. Of these causes tumor is by far the most frequent. The nature, size, and seat of the tumor appear to exert little influence in the production of the neuritis. The neuritis of tumor is often intense, and it is of diagnostic importance that an intense degree of neuritis is not often the result of any other process. Optic neuritis occurs in about nine tenths of all intracranial tumors. Meningitis is the next most frequent cause, and the neuritis is somewhat more frequent in meningitis of the base than of the convexity. Cerebral abscess (though less frequently a cause than meningitis) is certainly not an infrequent cause of optic neuritis. About six per cent. of all cases of multiple sclerosis are accompanied with optic neuritis, which is frequently slight and of short duration. In these cases there are inflammatory or sclerotic patches in the optic nerve. In thrombotic softening and hemorrhage, optic neuritis is of rare occurence, but in embolic softening it is less uncommon. Occasionally optic neuritis is met with in diffuse cerebritis. It is also seen after sunstroke and in idiopathic hydrocephalus. The optic neuritis of brain disease is almost always double, but the process is often more advanced in one eye. In multiple sclerosis the neuritis is very often one-sided, in consequence of unilateral involvement of the nerve by a sclerotic patch, and it is occasionally one-sided in other forms of brain disease, but, in general, unilateral neuritis is much more often due to disease within the orbit or at the optic foramen than to cerebral processes.

In rare instances optic neuritis has been developed in the course of acute or subacute disease of the spinal cord—transverse and disseminated myelitis, for example. It is probable that in most of these cases the neuritis is to be regarded as an associated and not as a consecutive process

Not infrequently optic neuritis occurs in consequence of disease outside of the nervous system. It is often seen in the course of chronic diffuse nephritis, and occasionally occurs in chlorosis, marked anæmia, diabetes, lead poisoning, scarlet fever, typhoid fever, and erysipelas. In some of these conditions, notably in anæmia, lead poisoning, and nephritis, there may be considerable headache, and the association of this with optic neuritis may give rise to a suspicion of intracranial disease, if the occurrence of papillitis under these circumstances is not borne in mind. Degenerative changes are often seen in the retina in cases of nephritic optic neuritis (neuro-retinitis). These changes consist of small white patches of fatty degeneration, often characteristically grouped about the macula lutea. When these changes are present they show the neuritic process to be dependent on kidney disease. Their absence, however, does not by any means exclude the possibility of the nephritic origin of the neuritis.

As regards the mechanism by which optic neuritis is produced, there is still considerable difference of opinion, but it is probable that the most important single factor is a descending inflammation along the optic nerve, or along the sheath by which it is invested. This view is supported by the pathological findings in a large number of cases of optic neuritis, and it is the only mechanism which will explain the occasional occurrence of unilateral optic neuritis on the side opposite a cerebral tumor. Distension of the optic sheath immediately behind the globe with serous fluid is of frequent occurrence in optic neuritis, and may be a factor in heightening the inflammation of the papilla, by preventing the escape and absorption of products of inflammation. It is also possible that this fluid may at times contain irritating pathogenic

material which is capable of inducing a certain degree of neuritis. There can be no doubt, however, that the first-mentioned mechanism is frequently alone effective. The theory that increased intracranial pressure is operative in causing optic neuritis by causing pressure on the cavernous sinus is now untenable : (1) Because a free anastomosis has been shown to exist between the orbital and facial veins ; (2) because large tumors of the brain and great hydrocephalus may exist with little or no neuritis ; (3) because tumors so small as to cause no appreciable change in intracranial pressure often produce a high grade of optic neuritis. It is to be regretted that this theory once gained so firm a hold that many still cling to it in the face of the objections mentioned.

In the minds of many there is even at the present day a belief that optic neuritis is a symptom which is of use in the localization of cerebral lesions. This is an error. Optic neuritis is usually a diffuse and not a focal symptom, and while it is a little more common with lesions in certain locations in the brain than in others, the symptom possesses pratically no localizing value.

Atrophy of the Optic Nerve.—Several clinical varieties of atrophy of the optic nerve, of importance in their bearing on the diagnosis of disease of the central nervous system, must be distinguished. Primary or simple atrophy, that is, atrophy which is not preceded by any recognizable inflammatory change in the papilla or surrounding structures, occurs in the degenerative diseases of the brain and spinal cord. It is probably of more frequent occurrence in multiple sclerosis and locomotor ataxia than in any other forms of disease. In multiple sclerosis it is much more common, in some degree, than is generally supposed. In a small proportion of these cases the atrophy is complete ; in a consid-

erable proportion (about 20 per cent.) the atrophy is partial, but involves the whole papilla, and in nearly the same number of cases the atrophic change is partial and involves only the temporal half of the disc. In locomotor ataxia, optic atrophy is present in about 15 per cent. of all cases, and is frequently an early symptom. The form of atrophy is rather characteristic, the disc being usually gray, as seen by the ophthalmoscope, with little or no diminution in the size of the vessels,—"gray atrophy." In a closely allied disease, dementia paralytica, optic atrophy occurs in a small proportion of cases (5 per cent.), but here, as in multiple sclerosis, the atrophy is probably to be regarded as an associated and not as a consecutive change.

Primary atrophy also occurs unassociated with central disease. Such atrophy has been attributed to cold, menstrual disturbance, migraine, exhausting diarrhœas, and, very rarely, to diabetes, syphilis, and various specific diseases. Tobacco, alcohol, and lead may lead to partial atrophy, but this is frequently preceded, in the case of tobacco and alcohol, by an axial neuritis. Primary atrophy generally involves both eyes, but one is often involved much earlier than the other. Not very rarely one eye alone is affected.

Disease of the optic nerve behind the eye or at the chiasm may cause atrophy (usually gray) of the optic nerve. Occasionally such atrophy is preceded by slight neuritis; usually it is not. This form is denominated "secondary atrophy." Loss of sight generally precedes visible signs of atrophy by a considerable period.

Neuritic or papillitic atrophy is that variety which follows optic neuritis. Frequently, but by no means always, the neuritis leaves changes in the choroid and in the vessels which make it possible to distinguish neuritic

atrophy from the simple form. Another variety of atrophy is that which occurs after disease of the choroid and retina.

The ophthalmoscopic appearances of atrophy vary somewhat in different cases. In all cases the disc is whiter than normal, but the degree and character of the pallor presents differences. The pallor may affect the whole disc or its temporal side only. But the temporal side of the disc is normally somewhat pale, because the nerve-fibres are there least numerous, and this may give rise to some difficulty in diagnosis when the pallor of atrophy is confined to the temporal side of the papilla. The pallor of atrophy is due to the wasting of the capillaries of the disc which accompanies the degeneration of the nerve-fibres. The tint of the disc varies, being in some cases milk-white, in others grayish, and in others bluish or yellowish. There is no diminution in the size of the optic disc in atrophy, because, although the nerve usually shrinks, the size of the papilla is dependent on the size of the sclerotic opening. Frequently the edge of the disc becomes very sharp and distinct. Sometimes the vessels are diminished in size, but this is not a constant change.

When optic atrophy is pronounced there is some diminution in the acuity of vision. In general, the degree of visual loss is related to the degree of visible change (pallor) in the optic nerve, but to this rule there are important exceptions. In some cases there is a high degree of visual defect with little or no pallor. When it is remembered that the whiteness of the disc depends on wasting of the capillaries, while the visual loss depends directly on the degree of change on the nerve-fibres of the optic nerve, it is not surprising to find that there is not infrequently a want of correspondence between visual acuity and the tint of the papilla.

The visual field, both for colors and for white, shows some degree of peripheral limitation in almost all cases of atrophy. In some cases this limitation is concentric, in others sector-like. Impaired color-vision is another accompaniment of optic atrophy, which, in some degree, is present in a large proportion of cases. Green is usually the color first lost ; red the next.

Of the *Associated Ophthalmoscopic Changes* of importance in relation to nervous disease, the following are the principal :

1. Primary atrophy of the optic nerve, occurring for the most part in association with degenerative diseases of the brain and spinal cord.

2. Albuminuric retinitis is of importance, because it is so frequently associated with atheromatous change in the arteries, and such degeneration may lead either to cerebral hemorrhage or to thrombotic softening.

3. Syphilitic choroiditis and the atrophy which follows it are frequently seen in brain disease from acquired and sometimes from congenital syphilis. Certain forms of atrophy of the choroid, however, result from other conditions than syphilis (posterior staphyloma, hemorrhage, senile change).

4. Tubercles of the choroid are in rare cases met with in tubercular meningitis. They are much less frequently observed when there is tubercular meningitis only than when such meningitis is associated with general tuberculosis. When they develop early in the disease they may be of the greatest assistance in diagnosis.

5. Embolism of the central artery of the retina very rarely occurs in cases of cerebral embolism.

6. Retinal hemorrhages occur under a variety of conditions—in albuminuria, leucocythæmia, purpura, etc. Occasionally small hemorrhages are found in the

meninges, particularly the pia mater, in these conditions, but they generally give rise to no symptoms.

The routine use of the ophthalmoscope in all cases of nervous disease in which there is even a possibility of discovering changes in the fundus cannot be too strongly urged. It is, however, important to remember that the condition of the circulation within the eye is not in any sense an index of the condition of the circulation within the cranial cavity.

Auditory Symptoms.—*Deafness* of nervous origin, that is, due to disease of the terminal auditory filaments in the labyrinth, or to disease of the auditory nerve or its central connections (p. 67), is of relatively uncommon occurrence as compared with deafness from middle- and external-ear disease. The character of such "nervous deafness" is the same, whether the disease causing it be central or peripheral; *there is defective conduction of sound through the cranial bones* as well as through the air. This constitutes a sharp distinction from the deafness dependent on disease of the middle and external ear, which impairs the perception of sounds conducted through the air, while it does *not* destroy the ability to perceive sounds conducted through the cranial bones. In making use of this method of distinguishing between deafness due to disease of nervous structures and that dependent on middle- or external-ear disease, either a vibrating tuning-fork or a watch may be employed. In persons more than fifty years of age it is not rare to find some diminution in the perception of vibrations transmitted through the bones of the skull, owing to changes in the labyrinth incidental to the degenerative period of life. This diminution is bilateral and never amounts to complete loss, though frequently the ticking of a watch is no longer perceived when the latter is in contact with the zygoma.

Since the character of the deafness is the same, whether it be due to labyrinthine, nerve, or central disease, the localization of the situation of the disease depends on associated symptoms. If there is paralysis of the facial nerve on the same side as the deafness, and there is no middle-ear or bone disease, the lesion is certainly in the auditory nerve, either within the auditory canal or between this and the superficial origin of the nerve from the upper part of the medulla. If deafness of rapid or sudden onset is associated with symptoms of medullary or pontine disease, especially with hemiplegia on the side opposite the loss of hearing, the lesion probably involves the auditory nucleus (p. 55). Deafness associated with hemianæsthesia or hemiplegia on the same side probably indicates disease of the internal capsule or extensive cortical disease involving the auditory centre in the first temporo-sphenoidal convolution. Deafness from disease involving this centre is exceedingly rare.

Much the most frequent cause of nervous deafness, both unilateral and bilateral, is labyrinthine disease. Such disease also gives rise to tinnitus and frequently to vertigo, and the association of one or both of these conditions with nervous deafness suggests, but does not prove, the existence of disease of the labyrinth. It does not *prove* its existence because disease of the nerve may have the same association, but in this case there is usually evidence of other nerve disease (facial paralysis). The nature of the labyrinthine disease varies in different cases. Acute and chronic inflammation, syphilitic disease, and degenerative processes occur. The deafness produced by certain drugs (quinine, etc.) is probably of labyrinthine origin.

The auditory nerve suffers chiefly from processes originating in neighboring structures—meningitis, simple and syphilitic, new growths, etc.

Disease involving the auditory nuclei is generally softening or hemorrhage, sometimes tumor.

Disease of the auditory path above the nuclei, in the tegmentum of the crus, in the internal capsule, or in the white substance of the temporo-sphenoidal lobe, is rare.

Occasionally deafness is of functional origin. It is not uncommon in hysteria in association with hemianæsthesia, etc.

Tinnitus Aurium is a term used to designate all sounds which are referred to the ear, and have no objective cause external to the body. These subjective sounds, as described by patients, are exceedingly varied in character. Generally they are simple in nature—that is, of a low degree of elaboration, and are spoken of as "singing," "ringing," "hissing," "buzzing," "whistling," etc. Sometimes they are more elaborate in character, and are compared to the ringing of bells, the rustling of wind in the trees, etc. The simpler sounds may be either aural or central in origin, but those of greater elaboration are almost always of central (cortical) origin. The sound may be constant or intermittent, continuous or pulsatory. In intracranial aneurism (carotid or vertebral) a pulsating sound has in very rare instances been produced, which could be heard by another person, and such a sound, if audible on auscultation, makes the existence of aneurism highly probable, and practically certain if there are at the same time evidences of a tumor at the base of the brain. But though the diagnostic significance of this sign is great, its extreme rarity makes it of little practical use.

In the great majority of cases tinnitus is of aural origin, for nearly all diseases of the ear, inner, middle, or outer, may be accompanied by it. Much less frequently the cause is organic irritative disease of the

auditory nerve or centre. Functional derangement of the cortical auditory centres occasionally gives rise to tinnitus. Thus, an epileptic attack may be preceded by a paroxysmal sound, due no doubt to nervous discharge from the nerve-cells constituting the auditory centre, and very rarely such sounds occur in migraine. Occasionally a continuous tinnitus occurs in cases of neurasthenia, mental depression, etc. Intense habitual stimulation of the auditory nerve, as by the sound of cannon, musical instruments, etc., may cause tinnitus.

Usually some degree of deafness coexists with tinnitus. When this is the case both deafness and tinnitus are dependent on the same cause, and what this cause is must be determined by a study of the associated symptoms, as already mentioned in speaking of deafness.

Vertigo.—This word has been loosely used to designate almost any subjective sensation that is associated with a feeling of imperfect equilibrium, or of transient mental confusion. When correctly used, the term is applied only to sensations in which there is experienced a feeling of movement, often of turning, or in which surrounding objects, which are really at rest, have the appearance of moving in a definite direction. These subjective sensations are often accompanied by a compensatory movement of the body of the patient, which may result in a fall. Some impairment of consciousness, never amounting to actual loss, always exists in vertigo.

Vertigo is a frequent symptom of organic brain disease, but it is a much more common symptom of peripheral disturbance. Occasionally it occurs where there is no discoverable cause for it, and then it is sometimes termed "essential" vertigo. It is, however, never to be regarded as a definite disease, but always as a symptom,

although in many cases it is the most obtrusive symptom of the condition on which it depends.

Before mentioning the varieties of vertigo, it is desirable to consider briefly the manner in which the equilibrium of the body is maintained. When the body is in any position requiring the active contraction of muscles for the maintenance of its balance, the centrifugal motor impulses which leave the brain for the establishment of this balance are influenced through the mediation of cerebral centres by certain sensory impressions which serve for the guidance of these centres in adapting the position of the body to its environment. The most important of these sensory impressions are those derived from the semicircular canals and from the motor nerves of the eyeball. Other impressions that furnish the brain with guiding information as to the relation of the body with surrounding objects are derived from the skin of those parts which are in contact with external things, and from the muscles whose activity determines posture. If any one of these different kinds of guiding sensory impressions becomes imperfect or deranged its expression in consciousness is felt as a derangement of the cerebral centre—that is, as vertigo. Derangement of the aural impressions gives rise to aural vertigo, derangement of the guiding impressions of the muscles of the eyeball results in ocular vertigo.

Aural Vertigo—that is, vertigo dependent on disease of the semicircular canals—is by far the commonest and most important variety of vertigo, constituting at least nine tenths of all cases of true vertigo. The disease of the semicircular canals is usually evidenced by tinnitus aurium and by nervous deafness. Often the latter is slight. Sudden lesions of the labyrinth generally give rise to violent attacks of vertigo and marked audi-

tory disturbance, and this variety of aural vertigo is properly known as Ménière's disease, although this name is sometimes made to include slighter forms of the affection. It is common for aural vertigo to be mistaken for evidence of organic disease of the brain, particularly for hemorrhage and softening.

Ocular Vertigo is a rare and usually slight form of vertigo which is due to paresis of an ocular muscle. The vertigo depends on the erroneous projection of objects caused by the diplopia resulting from the paresis. The occurrence of diplopia makes the diagnosis of the trouble easy.

Vertigo is not infrequently associated with dyspepsia, and the occurrence of vertigo in connection with gastric symptoms has led to the description of what is called *gastric vertigo.* It is doubtful whether vertigo is ever really referable entirely to gastric disorder. The majority of the cases described as gastric vertigo are due to labyrinthine disturbance and are really varieties of aural vertigo. But frequently the evidences of labyrinthine trouble are slight, and the true character of the disorder is readily overlooked. It must not be supposed, however, that gastric disorder has no influence in bringing about vertiginous attacks. It is easy to understand how a disordered stomach may act, through the central connections of the vagus, as an important exciting cause of vertigo, by disturbing the balance of an already unstable cerebral centre.

Vertigo sometimes constitutes an aura of the seizures of petit mal, and, at times, of grand mal. The distinction of this form from aural vertigo is described on page 582. The convulsions of organic disease of the brain are also at times preceded by vertigo. As a symptom of organic brain disease, vertigo has little diagnostic significance,

for it may occur in connection with a variety of lesions. It is especially frequent in lesions of the cerebellum and pons, and disease of the middle peduncle of the cerebellum gives rise not only to vertigo, but to an actual forced rotation in the long axis of the body towards the side of the lesion. The mere occurrence of vertigo, however, is of no localizing value, as it may be produced by disease in almost any part of the brain. It may occur in the course of cerebral tumor, abscess, chronic meningitis, general paralysis, multiple sclerosis, and at the onset of cerebral hemorrhage, embolism, or thrombosis.

When vertigo is severe it is apt to be accompanied by vomiting, which seems to be entirely dependent on the severity of the vertigo and not upon its cause.

Slight vertiginous attacks, consisting merely in a sense of defective equilibrium, may occur in the course of hysteria, neurasthenia, anæmia, etc.

Gustatory Symptoms.—The more important facts relating to loss of taste have already been sufficiently considered in connection with the anatomy of the fifth nerve (p. 61). There remain to be mentioned only the perversion of the sense of taste (parageusia) which is occasionally observed in hysteria, insanity, and other neuroses; the increased sensitiveness of taste, which is of occasional occurrence, under the same conditions; and the various subjective sensations of taste, usually unpleasant in character, that occur in insanity, and, rarely, as the aura of an epileptic paroxysm. With the possible exception of auræ of taste these disturbances are of slight diagnostic importance.

Trophic Symptoms.—The nutrition of the various tissues of the body is to a considerable extent under the domination of nervous structures, but we know little of the exact character of this influence and of the conditions

on which it depends. In the case of certain tissue elements the changes in nutrition that result from morbid conditions of the nervous system, in consequence of this dependence, are very striking and possess considerable diagnostic significance. The principal structures in which such alterations occur are the skin, the bones and joints, and the muscles.

Trophic Changes in the Skin may result from peripheral, spinal, or cerebral lesions. They vary considerably in character according to the acuteness of the lesion which gives rise to them, and are much more marked and frequent in peripheral and spinal than in cerebral disease. When the changes are rapid and intense, as they are in acute disease of the nerves (for example, acute neuritis), or spinal cord (acute myelitis), the skin becomes hot and red, bullæ form, and slight pressure may give rise to sloughing. In chronic lesions of the nerves and cord (chronic myelitis, syringomyelia) there is a gradual change in the nutrition of the skin, which grows thin, red, and glossy, and the nails become retarded in growth, brittle, and thick. In chronic cerebral disease (hemorrhage, softening, etc.) the skin sometimes becomes rough, dry, and thick, and the nails occasionally grow curved and brittle. In general, it may be said that the most serious trophic changes in the skin take place when the gray substance of the cord or posterior nerve-roots are involved in irritative processes; and there can be little doubt that the nutrition of the skin is dependent on fibres which pass through the posterior nerve-roots. There are no special trophic nerve-fibres. The situation of the trophic centres is also obscure, but it is probable that the posterior root-ganglia are in some way related to the nutrition of the skin.

One form of eruption is unquestionably dependent on

a nervous lesion: the eruption of herpes zoster. There is reason to believe this affection to be dependent on an inflammatory process involving one or more intervertebral ganglia, a nerve-trunk or its terminal nerve-filaments.

It is very possible that other forms of eruption (psoriasis, scleroderma) are dependent on nervous lesions, and several varieties of cutaneous ulceration (bed-sores of myelitis, perforating ulcer of foot) have long been regarded as true trophic lesions. But it is probable that in the case of these ulcerations the death of tissue is caused by external agencies, such as injuries and micro-organismic infection, which is rendered possible by the anæsthesia that exists in these cases. Probably in addition to the anæsthesia, the nervous lesion causes, in these cases, some modification of the nutrition of the parts, which predisposes them to necrosis from trivial injuries.

The trophic changes that occur in the eyeball in consequence of disease of the trigeminus may be mentioned in this connection. Lesions of the trigeminus, particularly irritative lesions involving the first division (ophthalmic) of the nerve, are frequently followed by ulceration of the cornea and subsequent perforation. This is especially apt to occur when the lesion involves the Gasserian ganglion, or the nerve in front of it. Whether these alterations in the nutrition of the eyeball are to be regarded as true trophic changes, or simply as consequences of injuries which the accompanying anæsthesia makes it difficult to guard against, cannot be positively stated. It is certainly true that under strict antiseptic care, ulcers of the cornea can be made to heal rapidly and perfectly, while the nerve-lesion continues in progress.

Trophic changes in the *Bones and Joints* are observed in the course of spinal cord and cerebral disease. Retar-

dation in the growth of the bones, without pathological changes in their histology, occurs in children when the ganglion-cells of the anterior horns are the seat of acute inflammation, and in most cases of hemiplegia coming on in the course of childhood. In rare instances similar changes occur after peripheral nerve-lesions. Extreme atrophy of the bones, together with atrophy of the skin and subcutaneous tissue, occurs in the rare condition known as progressive facial atrophy (facial hemiatrophy). The affection is strictly unilateral, and probably depends on some derangement in the trophic function of the fifth nerve, possibly on disease of its trophic root (see Fig. 44). Brittleness of the bones (*fragilitas ossium*) leading to fracture from slight injuries, so-called "spontaneous" fracture, is occasionally observed in dementia paralytica, amyotrophic lateral sclerosis, and locomotor ataxia. It is a question whether the fragility in these cases is due directly to the nervous lesion, or is merely one consequence of defective general nutrition.

Inflammation in the larger joints is occasionally met with in acute cerebral and spinal-cord diseases. The cerebral affection with which this change is associated is usually hemorrhage or softening (the latter more frequently), and the joint changes are limited to the hemiplegic side. The synovitis comes on in the course of a few weeks after the onset of the hemiplegia. Bilateral effusion into the knee-joints is occasionally observed in the course of acute myelitis. Extensive changes in the larger joints (knee, shoulder, elbow) are sometimes observed in the course of locomotor ataxia ("spinal arthropathies," Charcot's joint disease). These changes consist of rather sudden and painless swelling of the affected joint, followed by destructive disease of the articulation itself,—erosion of the cartilage, atrophy of the heads

of the bones, and relaxation of the ligaments. Sometimes there are irregular osteophytic growths external to the articulation. At times these changes appear to be related to slight injury. Their painless character is an important diagnostic feature of these arthropathies. When these changes involve the tarsal articulation, the foot may become flat ("tabetic foot").

Trophic Changes in the Muscles. The trophic changes that occur in muscles exceed in diagnostic importance all other nutritive changes dependent on nervous disease. The diagnostic indications are derived, first, from changes in the bulk and consistence of the muscles, and, secondly, from alterations in their electrical excitability.

Muscular Atrophy is a frequent consequence of organic disease of the brain, spinal cord, and peripheral nerves, but a high degree of wasting is seen only in disease involving the lower segment of the motor path—that is, in disease of the motor ganglion-cells of the anterior horns, the anterior nerve-roots, or the motor nerves. Such a degree of wasting may follow acute lesions, or may occur in the course of those that are chronic in their progress. In the former case the atrophy is preceded by motor paralysis of sudden or rapid onset (acute poliomyelitis, acute myelitis, acute neuritis, injuries to motor nerves); in the latter, the paralysis progresses *pari passu* with the weakness, being due to the gradual wasting of muscular tissue (progressive muscular atrophy). In an important group of cases of muscular atrophy of gradual development, in which no constant changes in the spinal-cord or peripheral nerves have yet been detected, and which are looked upon as myopathic in origin (progressive muscular dystrophies—idiopathic muscular atrophy), a high degree of muscular wasting is frequently observed. These cases constitute the only exception to the rule that a high

grade of muscular atrophy is due to anterior cornual or peripheral nerve disease. Their distinction from forms of atrophy dependent on nervous lesions is considered elsewhere.

It must not be supposed that all cases of disease of the anterior cornua, or motor nerves, are productive of a high degree of muscular wasting. In lesions of moderate severity or of gradual onset the wasting may be moderate or slight in degree, and, unless the distribution be distinctive, may resemble that which occurs after cerebral lesions. The atrophy which follows disease of the upper segment of the motor path (cerebral disease, many forms of spinal-cord disease not involving the anterior cornua) is usually slight in degree; occasionally it is considerable, but it never reaches the grade commonly observed in disease of the lower segment (myelitis, progressive muscular atrophy, multiple neuritis).

The rapidity with which muscular atrophy develops varies greatly under different circumstances. In disease of the *lower* segment of the motor path the rapidity of wasting depends chiefly on the acuteness or chronicity of the lesion to which it is due. In acute processes the wasting progresses with rapidity, often reaching a high degree in the course of a month, and often being readily appreciable after the lapse of ten days (acute myelitis, acute poliomyelitis). In gradually progressive lesions the atrophy is proportionately slow.

Disease involving the *upper* segment of the motor path is generally followed by some muscular atrophy, but this is almost invariably late in its development. This atrophy is particularly noticeable in young subjects and is frequently a result of irritative lesions. In a very small proportion of cases, cerebral lesions, especially disease of the cortex and subjacent white substance, are followed not by late but by early atrophy—atrophy which reaches its height

in from three to six weeks after the onset of the lesion, which is generally irritative in character. These early cerebral atrophies are mainly observed during adolescence and during early adult life. Usually the degree of wasting is inconsiderable. Both late and early atrophies are probably dependent, in a measure, on secondary degeneration of the pyramidal tracts, which, when due to irritative processes, seems to exert a detrimental influence on the nutrition of the ganglion-cells of the anterior horns and through these on the muscles. In rare instances hypertrophy of paralyzed parts may occur, which is, as yet, without satisfactory explanation.

In disease of the lower segment of the motor path the muscles usually become flabby and lose their tone ; they undergo " atonic atrophy." In disease of the upper segment the muscles may retain their tone, or, if there be well-developed secondary degeneration of the pyramidal tracts and increased reflex action, may grow hypertonic and exhibit muscular rigidity. When there is secondary degeneration of the pyramidal tracts, muscular rigidity may be preserved in cases of atrophy from disease of the ganglion-cells of the anterior horns, *provided* some of the ganglion-cells of the anterior cornua are only partially damaged, and still exert enough control over the muscular tone to cause that over-action which develops under the influence of degeneration in the upper segment of the motor path. If, however, the ganglion-cells of the anterior cornua are so extensively diseased as to abolish the muscular tonus, the degeneration of the upper segment is incapable of causing rigidity.

The distribution of the atrophy corresponds closely with the distribution of the motor paralysis in cases where the wasting follows such paralysis. In cases where the wasting is not secondary in time or causation to motor

paralysis, the distribution of atrophy presents considerable variety and forms a basis for the distinction of clinical types.

True *Muscular Hypertrophy* rarely occurs as a phenomenon of disease, but an increase in the bulk of certain muscles, associated with a change in their consistence and diminution of power, and due to an overgrowth of the interstitial connective tissue, and subsequent deposition of fat, is observed in one variety of the progressive muscular dystrophies (pseudo-hypertrophic muscular paralysis). Muscular hypertrophy, both the true and false type, occurs in Thomsen's disease, and in the healthy leg, in some old cases of poliomyelitis, as a result of increased activity, and in rare cases of infantile cerebral hemiplegia with athetosis and without.

Altered Electrical Irritability of Nerves and Muscles—Reaction of Degeneration.—In health the muscles and motor nerves are excitable by galvanism and faradism. Either current, when applied to a motor *nerve*[1] (indirect stimulation), is capable of producing a contraction in the muscle or muscles supplied by this nerve. The faradic current causes a contraction which continues during its application, but, if the distinct shocks of which it consists are passed through the nerve at short intervals, each stimulus gives rise to a contraction of momentary duration. A galvanic current of moderate intensity applied to the nerve gives rise to a muscular contraction only when the circuit is made or broken—that is, when the current is passed or stopped—or when a sudden change is made in the current strength. A slight diminution in the irritability of the nerve to both faradic and galvanic currents

[1] The point on the surface at which a motor nerve passes to its muscle is called a "motor-point." It is customary, in electro-diagnosis to obtain contractions by stimulating motor-points. These points are given in most text-books on general medicine.

is observed in diseases of the nervous system which impair the nutrition of either the upper or the lower motor segment; considerable diminution or complete loss of irritability to the strongest currents is observed only in disease of the *lower* segment (degeneration of the nerve-fibres from any cause).

Either faradic or galvanic current, when applied to a *muscle* without the intervention of a motor nerve (direct stimulation), can be made to produce a contraction of this muscle. The muscle possesses two irritable structures, which it is convenient to think of as being physiologically distinct: (1) the intra-muscular termination of the motor nerve-fibres with their motorial end-plates, and (2) the muscular fibres themselves. In health both these structures may be excited by either current, but it is probable that it is mainly by the action of these currents on the motorial end-plates that the contractions are produced. When these end-plates undergo degeneration, as they do in consequence of degeneration of the motor nerve-fibres or spinal ganglion-cells with which they are connected, and the motor *nerve* is no longer excitable by faradism *(vide supra)*, the *muscle* also loses its faradic irritability, because faradism is incapable of stimulating the muscular fibres themselves when these have suffered in their nutrition. But the galvanic current, notwithstanding the existence of nutritive changes in the muscle-fibres and from degeneration of the nerve-fibres and motor nerve-endings, is still capable of exciting a muscular contraction, by *directly* stimulating the muscular fibres. The contraction, however, which is obtained under these circumstances differs from that which is obtained in health in several respects. First, it occurs more readily for a time than the contraction of health—that is, the muscle responds to a feebler galvanic current. Secondly, the contraction is not the quick, sharp, and complete contraction of

health, but is tardy, wave-like, and often imperfect. Thirdly, there is a change in the readiness of response at the two poles. Under normal conditions a contraction occurs more readily at the negative pole when the circuit is closed (kathodal closure contraction, KCC), than at the positive pole when the circuit is closed (anodal closure contraction, ACC),—that is, KCC > ACC, and with a current of sufficient strength to produce both ACC and KCC the latter is always more vigorous than the former. Under the conditions of disease of which we are speaking (complete degeneration of the lower motor segment), the anodal closure contraction either occurs with a weaker current than the kathodal closure contraction, or both contractions occur with equal readiness—that is, ACC > KCC, or ACC = KCC. These different changes in the contractility of the muscles—loss of faradic irritability, temporary increase of galvanic irritability, with changed polar reactions and sluggish responses—constitute what is known as the reaction of degeneration, which may be conveniently expressed by the symbol RD. The RD is so called because it is evidence of a degenerative change in the motor nerve-fibres, which, when stimulated, no longer react to either current *(vide supra)*.

We have here described what may be called the complete or typical form of RD—the form which is observed in the severer varieties of lesions in the lower segment of the motor path, acute degenerative or inflammatory processes in the nuclei of the motor nerve-cells anywhere in the spinal cord, medulla, pons, or crura cerebri, or in the motor nerves which spring from these cells. Other forms of RD exist, however, presenting innumerable differences in the degree and character of the changes in electrical excitability. Of these, there are two forms or types of

RD that require mention. The first of these is observed in chronic forms of degenerative disease of the lower segment of the motor path, in which the nutrition of the nerve-fibres and muscle-fibres is gradually more and more impaired. The nerve-fibres and their motorial end-plates suffer slowly in nearly equal degree, and there is consequently a *gradual* loss to faradism and to galvanism in both nerve and muscle. This form of RD differs from the complete form, in that at no period of the degenerative process is the irritability of the nerve-fibres lost while that of the muscle-fibres is preserved. There is consequently no time when the muscles show an increased irritability to galvanism and loss of irritability to faradism. The other phenomena of the complete form are, however, present ; there is the characteristic sluggish response to galvanism, and, generally, the alteration in polar reaction.

The other variety of degenerative reaction that remains to be mentioned is termed the "mixed form." It is characterized by the presence of slight or marked changes in the irritability of the *muscles*, while the *nerve* either reacts normally or shows only slight diminution in irritability to both currents. The changes in the muscle vary considerably in different cases. Usually there is increased or diminished galvanic excitability, changed polar reaction, and sluggish contraction. The galvanic irritability may be nearly normal, however, and the altered polar reactions and tardy contraction may not be very pronounced. The mixed form of RD is observed in cases where some muscle-fibres are degenerated and others in the same muscle are normal or nearly normal, this condition of the muscle-fibres being dependent on a corresponding mixture of normal and degenerated nerve-fibres in the nerve. Clinically it is observed in peripheral paralyses, in progressive muscular atrophy, acute and subacute poliomyelitis, etc.

It is important to note the fact that the slow, sluggish contractions which are observed in RD constitute its most important and characteristic feature. They are present even in the slightest grades of RD, in cases where the irritability of the nerves to both currents is retained, where the faradic irritability of the muscles is only slightly diminished, and where the polar reactions are normal or only slightly modified.

The development and course of the electrical changes which constitute the RD vary according to the severity and chronicity of the morbid processes on which they depend. In cases where the lesion is very severe and acute in its development (acute neuritis, acute poliomyelitis, severe nerve injuries), there is an early [1] and rapid fall in the irritability of the nerve, both to faradism and galvanism ; in the course of two or three weeks the irritability of the nerve becomes extinct. In the muscle there is a fall in faradic irritability which corresponds in time and extent (being due to the nerve-endings) with that observed in the nerve. The galvanic irritability of the muscles may remain unchanged for several days after the onset of the lesion, or may fall slightly. Then occurs a considerable increase in irritability, which reaches its height in about a month, and is followed by a gradual decline, until, after the lapse of a year, or, in some instances, two years, no reaction can be obtained with a current that can be borne.[2] In such a case there is no recovery of electrical irritability or of power of voluntary contraction. In somewhat less severe cases of acute

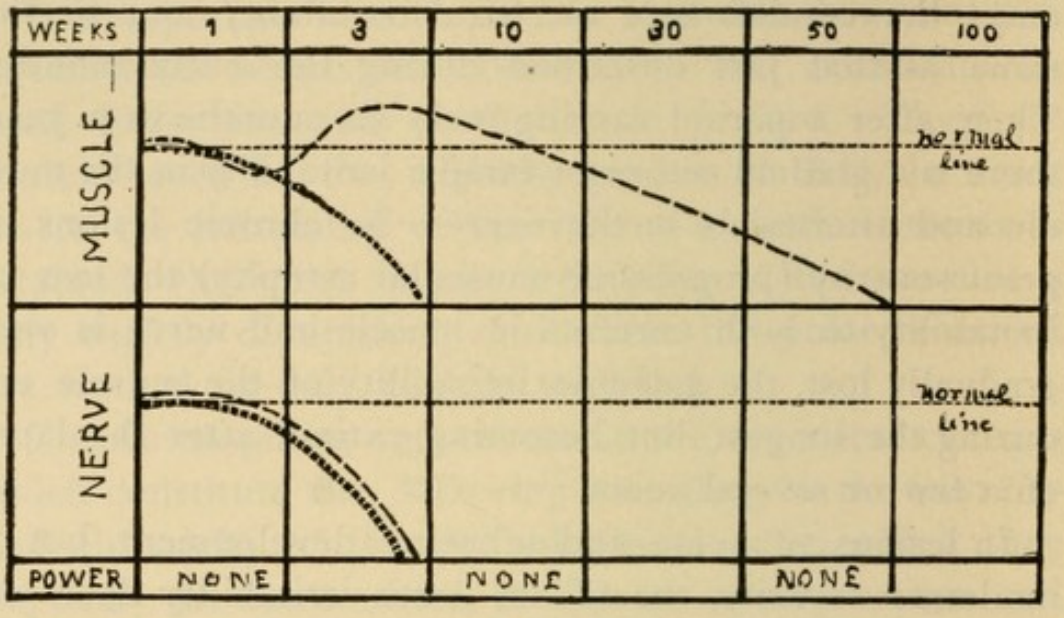
[1] This fall usually begins about the middle of the first week, sometimes not until its end.

[2] When galvanic irritability of the muscle is extinct to ordinary modes of stimulation, the insertion of a needle into the muscle as one pole of the battery may elicit a local contraction.

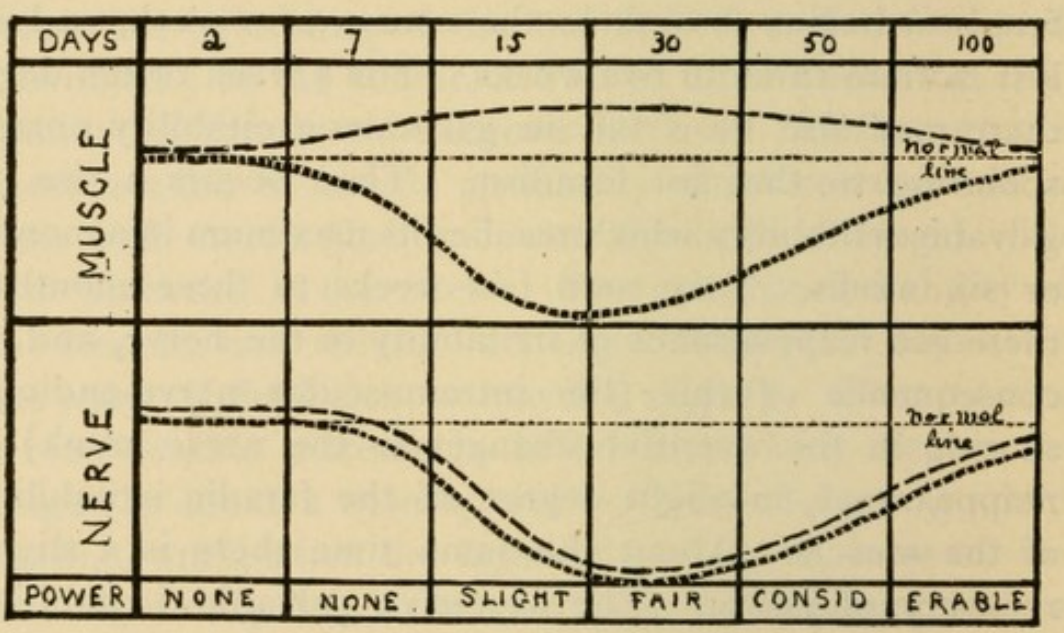
onset the condition of electrical irritability may be the same as that just described during the early months. Then, after a period varying from six months to a year, there is a gradual return of faradic irritability in the muscle and afterwards in the nerve. In chronic lesions of great severity (progressive muscular atrophy) the loss of irritability to both currents in muscle and nerve is very gradually lost, the galvanic irritability of the muscle enduring the longest, but becoming extinct after the lapse of a few or several years.

In lesions of acute and subacute development, but of moderate severity, the fall in nerve irritability does not differ greatly in rapidity of progress or degree from that observed in the severest forms. Muscle irritability to faradism begins to sink in the course of a week, and is lost in from three to five weeks. For a week or ten days there may also be a fall in galvanic excitability corresponding to that for faradism. Then occurs a rise in galvanic irritability which reaches its maximum in a month or six weeks. Very soon (six weeks to three months) there is a reappearance of irritability in the nerve, and in consequence of this (the intramuscular nerve-endings sharing in the nutritive changes in the nerve trunk) a reappearance, in slight degree, of the faradic irritability of the muscle. About the same time there is a slight recovery of power. The excessive galvanic irritability may continue a considerable length of time (three or five months) before returning to the normal, which it does gradually.

In lesions of slight severity there is frequently a slight rise in nerve irritability. This may last several weeks, and be followed by an inconsiderable fall in irritability, lasting a few weeks or months. Power of voluntary contraction is never entirely lost, and the loss which occurs



A



B

FIG. 69.

Fig. A shows type of electrical reactions in a case of severe and permanent damage to a nerve. The broken lines ▬▬▬ in this and the following chart show the state of galvanic irritability. The broken lines ▬ ▬ ▬ show the state of faradic irritability. The broken curves in the upper half of each chart show the state of electrical irritability in the *muscle;* the dotted lines in the lower half of each chart show the state of electrical irritability in the *nerve*. The horizontal dotted line represents the normal degree of irritability.

Fig. B shows type of electrical reaction in a case of slight damage to a nerve.

is recovered from in from three to six weeks. The irritability of the muscle to faradism corresponds, in general, to that of the nerve, but there is seldom increased faradic irritability even when this occurs in the nerve. The galvanic excitability has the same character as in lesions of moderate severity, but the increase is less and of shorter duration.

Since the extent of the changes in nerve and muscle irritability varies, in general, with the severity of the degenerative process in the nerve, we have in the RD a valuable aid in prognosis, for the severity of the case and its duration (in the case of acute lesions) are proportional to the loss of nerve irritability. If there is complete loss of nerve irritability, and greatly increased galvano-muscular excitability, with sluggish contractions, etc., the degenerative process is one of severity. The question then arises whether or not regeneration can take place within the nerve—that is, whether or not there will be recovery of trophic innervation, return of power, etc. The possibility of *some* recovery is not gone until there is complete loss of irritability to both currents in the muscle, but if there is no return of faradic irritability in the muscles at the end of a year, and their atrophy has been rapid and great, only slight improvement, at best, will occur. If the lesion is an acute one, and the muscles have lost their faradic irritability after the lapse of a week or ten days, considerable atrophy will certainly follow, and the loss of motor power will be of long duration. If the faradic loss does not occur until two or three weeks have passed, there will be less atrophy and earlier recovery, but the paralysis will last many months. If there is only slight loss of faradic irritability, or no loss whatever, recovery will begin in the course of a few weeks or months.

The presence of RD, as already stated, indicates disease of the lower segment of the motor path. The more important clinical conditions in which it is observed are poliomyelitis, progressive (spinal) muscular atrophy, amyotrophic lateral sclerosis, lesions of the ganglion-cells of the anterior horns from hemorrhage, tumor, syringomyelia, etc., bulbar paralysis, injuries of the peripheral nerves, neuritis of all kinds (rheumatic neuritis, multiple neuritis, toxic paralyses, and paralyses after infectious diseases). The absence of RD does not exclude the possibility of a slight affection of the anterior horns or motor nerves. It may be absent in very slight peripheral paralyses from injury. On the other hand, RD may be present, in slight degree, in cases of lead poisoning and slight injury to a nerve in muscles that show no appreciable loss of power.

Diminished Irritability of nerve and muscle to both currents, without RD, is due to slight changes in the peripheral nerves, which may or may not depend on corresponding changes in the motor ganglion-cells. In the case of lesions in the upper segment of the motor path (cerebral paralysis—pyramidal tract lesions) the electrical changes are due to the nutritive alterations produced in the motor nerve-cells by the irritative influence of descending degeneration in the pyramidal tract.

The clinical conditions in which simple diminution of irritability without qualitative change is observed, are as follows :

(1) Cerebral paralyses ; diminished irritability in paralyzed muscles in the course of three or four months, usually preceded by a rise in irritability.

(2) Spinal-cord diseases ; bulbar paralysis, amyotrophic lateral sclerosis ; sometimes difficult to distinguish from the slighter RD which occurs in some of these conditions.

(3) Peripheral nerve disease; after slight injuries, after recovery from RD, in multiple neuritis and in arsenical paralysis.

(4) Muscular conditions; atrophy from disuse, arthritic atrophy, progressive myopathic atrophies (muscular dystrophies).

3. *Increased Irritability* of nerve and muscle to both currents, without qualitative changes, is frequently observed in the early period of hemiplegia when irritative phenomena are present, in the early stage of acute and subacute myelitis, in the early stage of progressive muscular atrophy (the increase in irritability in these cases is usually very great), and in the first days of slight neuritis. An increase has also been observed in some cases of hemichorea, tetany, locomotor ataxia (pre-ataxic stage), and dementia paralytica. A peculiar variety of increased muscular irritability to both currents is that which occurs in myotonia congenita (Thomsen's disease), and is hence called the myotonic reaction. The myotonic reaction consists in increased faradic contractility, with prolonged contraction after the current ceases, and in excessive galvanic irritability when the circuit is closed (ACC and KCC), with exceedingly sluggish, lasting, and wavy contractions. The nerve excitability is normal.

Vaso-Motor Symptoms are of frequent occurrence in the course of both organic and nutritional disease of the nervous system, but the diagnostic indications furnished by them are of comparatively little importance and will be only briefly mentioned here.

The disturbances which are the expression of disordered vaso-motor function manifest themselves clinically as local or general changes in color and temperature, or by alterations in secretion, or by the presence of œdema. In general, all these changes are most marked in acute

iesions of an irritative character. Local changes in color and temperature are observed in many cases of cerebral, spinal, and peripheral disease. In hemiplegia there is often an increase of ½ degree F. in the temperature of the paralyzed limb, and this may be accompanied by distinct redness of the skin. In other cases there is a fall in the surface temperature of the limbs ; they become pale and livid. A slight degree of œdema is frequently observed in the paralyzed extremity, and occasionally there is a marked local increase in perspiration. Any or all of these changes may be produced by disease of the cortex or of the vaso-motor path from it to the posterior limb of the internal capsule.

In spinal-cord disease slight vaso-motor disturbances are very common, and more marked changes are always present in inflammatory processes, whatever may be the position of the disease. At first the changes are those produced by vaso-motor dilatation (redness, increase of temperature, sometimes sweating) ; later, there is depression in the temperature of the limbs involved, which become livid, often extremely so, at the extremities. Œdema is not uncommon in the early stages of acute processes, but may also accompany disease of exceedingly slow development (for example, syringomyelia).

In diseases of the peripheral nerves vaso-motor disturbance is of frequent occurrence and has the same character as spinal-cord disease. Very slight neuritis is capable of producing very marked changes in the temperature of a part for a considerable period of time.

It is thus seen that no deductions as to the position of the lesion can be made from the existence of vaso-motor symptoms ; these must be made from the associated conditions (motor paralysis, sensory symptoms, muscu-

lar atrophy, etc.) which are commonly present, and from the general distribution of the phenomena.

Peripheral vaso-motor disturbance is not at all infrequent in hysteria and allied conditions, and is often most marked in its manifestations. The distinction of vaso-motor symptoms of functional origin from those that depend on organic disease, is usually simple, and is based on the mutability, temporary duration, and dependence on emotional disturbance, which is observed in the former. The extent to which the vaso-motor apparatus is under the influence of psychical processes is illustrated by the fact that, in rare instances, local hyperæmia and even vesication have been produced by verbal suggestion in subjects, usually hysterical, in the hypnotic state.

The "*tache cérébrale*," or better, dermographism, in which slight cutaneous irritation is followed by distinct local congestion of the skin of considerable duration, is a vaso-motor disturbance dependent on loss of tone in the smaller vessels from defective innervation of the local ganglia in their walls. It is not diagnostic of meningitis, as has been supposed, but is observed in many different conditions in which the general nervous system suffers in its nutrition.

Mental Symptoms.—*Loss of Consciousness* is one of the most significant and frequent symptoms of organic disease of the brain, but since it is also a symptom of many conditions that do not depend on structural disease, but on nutritional disturbance of the cortex (functional disease), its exact significance depends largely on its associations. The expression "loss of consciousness," as employed in medicine, refers to loss of the external manifestations of mental activity, not to the loss on the part of the patient of purely subjective consciousness, though the two may coincide. Considerable variations in the degree of unconsciousness occur. In cases where it is possible

to rouse temporarily the patient from his sleep, the loss of consciousness is regarded as partial, and is termed "stupor." When the loss is complete and the patient cannot be roused by any ordinary stimulus (voice, rubbing, slapping), the condition is termed "coma" if it endures longer than a few minutes. In coma there is partial or complete loss of control over the sphincters, superficial reflex action is generally lessened or lost, the muscles are relaxed, and there may be loss of the deep reflexes. The pupils are usually dilated and immobile; sometimes they are contracted (generally in toxic states). The pulse is often slow and irregular in force and frequency; sometimes it is rapid and feeble. Respiration is diminished in frequency and force. The relaxation of the palate permits it to vibrate with the air-current and gives rise to stertorous breathing. Frequently the respiration is irregular, and sometimes it presents the rhythmical changes in depth known as "Cheyne-Stokes respiration."

In stupor, on the other hand, the reflexes, both superficial and deep, are retained, the pharyngeal reflex may be excited, and the pupils react to light, but there is usually some loss of sphincter control.

Loss of consciousness may be produced by almost any one of the pathological processes to which the brain is subject. In every case the impairment of consciousness probably depends on interference with the highest functions of the cortex, and such interference may be due either to disease of the cortex itself, or to the inhibitory action of lesions elsewhere in the brain.

Chronic processes impair consciousness either through the involvement of a considerable area of the brain cortex, or by increasing greatly or rapidly the intracranial pressure. Lesions of sudden onset (hemorrhage or soften-

ing) on the contrary, may cause loss of consciousness in whatever region of the brain they may be situated, probably by their irritative inhibitory effect on the cortex. When the loss of consciousness is suddenly produced, as it usually is in acute lesions, it is termed "apoplexy." Cerebral hemorrhage [1] is by far the most frequent cause of apoplexy, and on this account the word is frequently used synonymously with "hemorrhage into the brain." Apoplexy may also result from the arrest of blood supply which leads to cerebral softening, and may be caused by embolism or thrombosis of a cerebral artery. Sometimes it results from simple concussion (often slight) of the brain, and in rare cases it can be ascribed, on reasonable grounds, to congestion. In old persons apoplexy occasionally occurs without the existence of any cerebral changes to which it can be attributed. In dementia paralytica also, apoplectic attacks occur which cannot be satisfactorily accounted for. Temporary loss of consciousness, of sudden onset, occurs in epilepsy and constitutes an important feature of the paroxysms. Of the causes outside the nervous system that may give rise to loss of consciousness those that depend, like uræmia, diabetes, and cholæmia, on the production of toxic substances in the organism (auto-intoxication), and those that depend on the introduction of poisons from without (alcohol, opium, chloral), are the most important. The loss of consciousness of cardiac syncope in anæmia depends on a temporary deficiency in the cerebral circulation, and should be classed with the functional varieties of such loss.

The diagnosis of the cause of coma is often a matter of the greatest moment on account of its bearing on prog-

[1] Depressed fracture of the skull is also a frequent cause of loss of consciousness, but only pathological causes are considered here.

nosis and treatment, but is often attended with considerable difficulty. It frequently happens that a patient is brought to a hospital in coma, and that the medical attendant is, for the time at least, without any knowledge of the previous history of the case. The correct diagnosis in such a case rests on its objective features, and every case of coma requires an exceedingly careful examination. Even then it is frequently impossible. The points of distinction between the different organic diseases (hemorrhage, thrombosis, embolism,) which may give rise to coma are described in Chapter V., but the main differences between coma of organic cerebral origin and the loss of consciousness that is due to functional disease of the brain, or to toxic influences, will be briefly mentioned. In every instance *focal symptoms* of brain disease are to be looked for. Hemiplegia may be detected during coma, if there is some restlessness, as the movements are most marked or confined to one side, but if the coma be very deep, evidence of paralysis may be entirely wanting. The presence of hemiplegia must then be inferred from other indications, and of these the most important are the condition of the reflexes, the unequal movements of the superior intercostals on the two sides, and the presence of Babinski's sign. The unilateral loss of the abdominal, plantar, or tendon reflex (knee-jerk) makes the existence of a one-sided cerebral lesion probable, and this probability is considerably increased if two or more of these reflexes are absent. On the other hand, if no distinct abnormality in the skin or deep reflexes can be detected, and there are no decided indications of hemiplegia or other focal symptom, it is improbable that there is a cerebral lesion. Conjugate deviation of the eyes is usually evidence of a unilateral organic cerebral lesion, the deviation being usually to the side of the lesion. If there is no evidence of an organic lesion the loss of con-

sciousness is probably due to functional disease of the brain, or to some toxic condition,—as alcoholic, diabetic, or uræmic intoxication. Of the functional states that cause coma the chief are epilepsy and hysteria.

Post-Epileptic Stupor is usually of short duration (one to two hours at most, even after a series of seizures); the patient can be easily roused; one-sided symptoms are absent, except in hemiplegic (old) epilepsy; the temperature is normal or slightly elevated, and the loss of consciousness is followed by temporary headache and often by a confused state of mind. An epileptic also usually bears physical stigma of his disease—bromic acne, bruises, etc.

Hysterical Coma occurs in patients of the sex and age that suggest hysteria, is generally preceded by other symptoms of hysteria (especially convulsion or headache), and is characterized, even in cases of apparently profound unconsciousness, by the preservation of the power of swallowing. There are no distinctive objective symptoms, but varying rigidity of the muscles and twitching of the eyelids are very frequently observed. If headache has been present it ceases when the coma comes on.

Uræmic Coma may closely resemble apoplexy from an acute cerebral lesion. In both conditions there may be albuminuria and convulsions. The absence of albumen in the urine is strongly against uræmia, but not absolutely so; its presence has not much significance, except in persons under thirty-five years of age. Repeated convulsions occurring at short intervals favor uræmia; single or few convulsive seizures make it improbable. The convulsions of uræmia may have a local commencement, and may remain local (unilateral); usually they are general. The presence of neuro-retinitis of the albuminuric type strongly suggests, but does not prove, that the coma depends on uræmia, and its absence does not exclude the

uræmic origin of the coma. Continuous subnormal temperature favors uræmia. If there is a history of sudden complete blindness before the coma this makes its uræmic origin practically certain. The pupils are usually dilated in uræmia coma.

Alcoholic Coma may be exceedingly difficult to distinguish from the coma of hemorrhage or softening. Its duration is short, there are no unilateral symptoms, and there is usually evidence of drinking. The presence of alcohol in the breath is of course no proof that the coma is due to alcohol. It may be necessary to wait a few hours before coming to a positive conclusion.

Diabetic Coma may be preceded by other cerebral symptoms, especially headache, and sometimes convulsions. The diagnosis is based on the detection of glucose and acetone in the urine, both of which are probably present in all cases of diabetic coma. Acetone is, however, present in many conditions, especially gastro-intestinal disorders, other than diabetes. The breath in diabetic coma often smells of acetone.

Delirium.—The word delirium is employed to express very different ideas. As used here it is intended to designate illogical and incoherent mental processes, as expressed in word or act. The perversion of mental activity that occurs in delirium is distinguished from the similar alteration that constitutes insanity by its acute course and temporary duration, and by its dependence on pyrexia, toxæmia, or organic disease of the brain. In what is termed "quiet" delirium there are sensory images without corresponding sensory impressions—"hallucinations,"—or actual peripheral impressions give rise to false sensory images—"illusions." There are false ideas or delusions, the nature of which is frequently determined by the character of the hallucinations or illusions. The patient generally talks incoherently in a low tone, and

often this muttering speech is almost continuous. He lies quietly in bed or exhibits only slight restlessness. In "active" delirium, on the contrary, the patient is restless and may attempt to leave his bed. He attempts to act according to his delusions, and in these attempts he may commit acts which threaten his life or that of his attendant. The delusions of delirium change rapidly in character and never present the systemized and elaborate character of those observed in some forms of insanity. There is rarely much emotional depression.

Delirium is much more often dependent on pyrexia, toxæmia, etc., than upon organic disease of the nervous system. The cerebral processes on which it depends most often are meningitis, tumor, extensive softening, and cerebritis. The occurrence of delirium in the course of spinal-cord and peripheral-nerve disease (myelitis, multiple neuritis) depends on the pyrexia which accompanies these processes, and not on their situation or nature. The character of the delirium gives no clue in determining whether it is due to pyrexia, its most frequent cause, or to organic disease of the brain. If, in a given case, delirium is the only symptom referable to the nervous system, it can be attributed to organic disease of the brain only if the body temperature is below 101 ° F., because the existence of a temperature greater than this makes it probable that the delirium is referable to the pyrexia. In persons who habitually use alcohol in excess, in those who are in debilitated health, and in the aged, a slighter degree of fever may give rise to delirium, and this must be borne in mind in forming an opinion of its significance. But the coexistence of delirium and a temperature above 101 ° is indicative of the non-organic origin of the delirium only in cases where there are no symptoms suggestive of encephalic disease, and such symptoms should be sought for with the greatest care

before a conclusion is reached. The occurrence of muscular rigidity, convulsions, diplopia, or strabismus, or of repeated vomiting, should excite grave suspicion of organic mischief. The coexistence of optic neuritis makes organic disease practically certain, for, although optic neuritis sometimes occurs after acute infectious diseases, it does not in these cases make its appearance until after the height of the disease has been passed. The association of delirium and headache is exceedingly important. If the headache is *severe* it should excite suspicion of organic disease. The headache of fever usually lessens or ceases when delirium is developed. Hence the persistence of slight headache is an equivocal symptom.

Simple Mental Deterioration is of frequent occurrence both in cases of nutritional and organic brain disease. It is manifested chiefly by defective memory—amnesia. Memory is subserved, in all probability, by the same physical elements that subserve intellectual processes generally, and of these the structures that make up the cerebral cortex and bring into association its constituent cell-elements are the most important. Loss of memory may result from any condition of disease which impairs the nutrition of the brain generally or that of the cortex. It is observed after acute infectious diseases, after severe mental shock (often such as occurs from the fright experienced when an injury is received), in hysteria, in epilepsy, and in neurasthenia. It results also from various organic processes, particularly from those that involve the cortex or the subjacent white matter—meningitis, hemorrhage, softening, new growths, and degenerative processes (dementia paralytica, dementia senilis). Inability to concentrate the attention is often associated with amnesia, and may be its cause, the loss of memory being apparent rather than real.

Defects in the Moral Sense are very apt to be associated with mental failure. Often the change is slight and difficult of estimation by the physician. In given cases the degree of defect can be measured only by comparison with the previous moral status of the individual. It is exceedingly important to take into account the significance of slight defects in the moral sense of which the patient is unconscious, and which contrast with the patient's previous sense of propriety. Such defects are always of serious import. They are often the first indications of dementia paralytica, making their appearance before the unsystematized and characteristic delusions of this disease. A rude disregard of the comfort or interests of others in one who has been habitually thoughtful in these respects, or the telling of unnecessary and usually blundering falsehoods by one who has always been strictly truthful, should in adults always excite some suspicion of degenerative brain disease. This is particularly true if the moral defects are associated with conspicuous loss of memory or defective power of attention. The refusal to swallow food when there is no pharyngeal paralysis is usually an indication of a considerable degree of mental failure. Indecent exposure of the body, or the passage of urine and fæces in the bed when there is no sphincteric paralysis, are evidences, except in children, of considerable mental or moral change. The passage of urine and fæces in the bed, under these conditions, is frequent in some forms of organic cerebral disease—softening, dementia paralytica, etc.

Disturbances of Speech.—Disturbances of speech[1]

[1] The word speech is here used in its broadest sense—that is, to denote the ability of one individual to communicate thought to another by means of symbols, and to recognize such symbols when made by others.

are of frequent occurrence in brain disease, and often give indications as to the position of the disease which gives rise to them. Hence they are of considerable diagnostic importance and deserve careful study. The symptoms of speech affection are, however, numerous and highly intricate in their relations, and their study is rendered difficult by the circumstance that accurate information as to the effects of local lesions in producing these symptoms is still scanty. Often, too, the same facts are susceptible of different interpretations.

Two distinct mechanisms exist for the expression of articulate speech. One of these, situated in the cortex of the brain, subserves the arrangement of the various elements which enter into speech. This may be termed the higher speech mechanism. The other, consisting of nerve-cells in the medulla and pons, is related to the form and not to the arrangement of the speech elements—that is, it is simply concerned with the articulation of words. This is the lower speech mechanism. It is brought into use by stimuli which descend to it from the higher mechanism. Disease of the lower speech mechanism gives rise to defects or loss of articulation. Disease of the higher speech mechanism gives rise to defects in the use and arrangement of words.

Defects of Articulation (dysarthria, anarthria) may result from any lesion involving the lower speech mechanism (nuclei of hypoglossal and facial nerves) or the peripheral paths from it (that is, the lower segment of the motor path to the muscles concerned in speech). Whenever there is considerable defect in articulation there is appreciable paralysis of the muscles concerned in articulate speech. The defect may be due to unilateral or bilateral palsy. Defective articulation may arise also from disease in the upper segment of the motor path, either in the

cortex or in the path from it. When the disease is unilateral, the speech defect is usually transient, because compensation takes place from the other hemisphere ; but if the disease is bilateral—that is, involves the structures concerned in articulation in both hemispheres,—the defect is permanent.

The changes in articulation that depend on disease of the lower segment of the motor path vary according to the muscles that are involved in the palsy. Palsy of the tongue causes imperfect articulation of the linguals *l* and *t*. Paralysis of the lips causes their imperfect approximation and consequent defect in the pronunciation of labial consonants. *P*, *b*, and *n* are replaced by *f* and *v*. Paralysis of the palate gives the voice a nasal and monotonous quality. It also causes imperfect pronunciation of the consonants *p* and *b* ("explosive" labials), because non-closure of the nares hinders the compression of air within the mouth necessary for their production. Separation of syllables, changes in rhythm, and sudden explosive utterance of words in what is termed "scanning speech" (also "staccato" speech), is a form of disturbance frequently observed in multiple sclerosis, of which it is rather characteristic, and occasionally in chorea and in affections causing imperfect transmission of impulses from partial damage to nerve-roots. "Confluent" articulation is one of the manifestations of slighter disease of the bulbar mechanism, and consists in the running together of terminal consonants and syllables with those that follow.

Speech Defects Dependent on Disease of the Cerebral Hemisphere and expressed, not as defects in articulation, but as errors in the arrangement or nature of the speech elements, are designated by the word *aphasia*. Cerebral processes of speech are either motor or sensory in char-

acter, and the leading forms of speech defects, *motor aphasia* and *sensory aphasia*, are due to derangement of the corresponding processes of speech. The motor speech processes are those by which language is uttered. They are effected in the posterior part of the third frontal convolution, and possibly also in the neighboring part of the anterior central convolution. In the lower part of the anterior central convolution of either side is the centre for the muscles concerned in *articulation*, which is interfered with by the bilateral involvement of these cortical centres, as already mentioned, and to some extent by their unilateral involvement. The motor speech centre (Broca's centre), in which the arrangements for the expression of word processes is effected, must be carefully distinguished from the articulatory centre just mentioned. The two are adjacent and perhaps some structures are common to both, but articulation may be impaired without impairment of the higher motor processes, and the latter may be impaired without any defect in the power of articulation, though frequently both are impaired together. From the centre for the movement of the lips and tongue, fibres pass through the internal capsule to the lower mechanism of articulation.

A lesion destroying the left motor speech centre usually gives rise to total loss of voluntary speech (motor aphasia). The loss is of *voluntary* speech only, because emotional and automatic speech is subserved by both hemispheres, and is continued by the speech centres of the right hemispheres when those of the left are damaged. In persons who are right-handed the speech centre for voluntary speech is in the left hemisphere, the corresponding centre on the right side being related only to involuntary and automatic utterance, although it is capable of being educated to supplement the action of the struc-

tures on the left side. In those who are left-handed, although they may use the right hand for writing, voluntary speech is subserved by the centre in the right hemisphere.

Not only may voluntary speech be lost in consequence of extensive destruction of the motor speech centre, but there may be impairment of expression by means of gestures. The patient may for a time make with his head the sign of negation when he wishes to express assent. Very soon there is some recovery of the ability to say simple words like "yes" and "no." These words may be employed in two ways—as propositions, to express the ideas of assent and dissent (propositional use), and as interjections, for the expression of emotion, and they may be regained and employed as interjections before they are again acquired as propositions. Many other words possess these two forms of use, and the propositional use is always regained the later. Thus many patients can swear or sing songs who could not deliberately and propositionally make use of the words of an oath or of a song. In some cases a considerable degree of speech loss persists for a long period of time. In the majority of cases there is slow recovery of speech after the lapse of a few months, so that eventually recovery of language occurs. When the lesion in the speech region is destructive in character, this ultimate recovery of language is unquestionably due to compensation through education of the right hemisphere (speech centre) for higher processes than it originally subserved (voluntary speech). The extent to which this supplemental action of the undamaged speech area is possible varies greatly even in adults. In children it occurs to an astonishing extent. It is exceedingly rare to see motor aphasia in a child in consequence of disease in the left hemisphere.

That this is because of extensive compensation by the right speech region is shown by the fact that a second lesion in this situation has been known to cause permanent loss of speech. In non-destructive lesions of the left centre, or in lesions of limited extent, some recovery of function is possible in the nerve-elements of this side, and it is often difficult to distinguish between the recovery which is due to compensation in the opposite hemisphere, and that due to recovery on the side of the lesion.

In the speech that remains after an attack of aphasia, or in that which is regained, there is frequently error either in form or in use. The error in form is in the elements of the word. Thus, one patient said "tinors" for scissors; another, "lant" for lance; and another, "trayling to dool" for trying to do. In these cases the error lies in the elements of the word used; there is not usually any defect in articulation associated with the error in form. This erroneous formation of words, which often results from disease of the motor speech region, has been termed *ataxic aphasia*, but as a very similar condition may occur from disease elsewhere in the brain (see word-deafness) the term motor aphasia is the preferable designation for all effects of disease of the motor speech area. Frequently there is an erroneous use of words without error in form. At first even the simplest words, as "yes," "no," may be erroneously employed. Where there remains considerable speech, wrong words, which may or may not resemble the ones intended, may be used repeatedly.

Agraphia, or loss of power of writing, is practically always the consequence of motor aphasia. In order that a word may be written, the motor speech processes corresponding to it must be correctly arranged in the motor speech area, from which they pass from a subcortical tract of fibres to the motor centre for the hand in the

middle of the ascending frontal convolution of either side. From these centres the motor impulses are conducted by the pyramidal tracts to the cord, and, through it and the peripheral path to the writing hand. It is important to note that the ability to write does not depend on paralysis of the movements of the arm and hand, for, even when these movements are perfect, a lesion in the motor speech centre causes agraphia. Agraphia is also caused by a small lesion involving the subcortical tract from the speech centre to the hand centre. The loss of the power of writing, like motor aphasia, may be complete or partial. When the loss is partial, the errors in writing are similar in character to those which occur in speaking, and frequently they are greater. In these cases the power of copying is preserved.

Motor aphasia is not always the consequence of disease of the cortex. It may follow interruption of the conducting tract to the hand centre of the same side, which lies just beneath the cortex, or a lesion in the internal capsule. If the lesion is in the internal capsule the loss of speech is transient; if beneath the cortex it may be permanent. No disturbance of intellect is necessarily associated with motor aphasia.

Motor aphasia may be produced by any pathological process which involves the speech region or the subjacent white matter. It is most frequently produced by cerebral softening. The motor speech area lies within the province of the first branch of the middle cerebral artery (see Fig. 57), and hence must suffer if this is occluded by embolism or thrombosis. Aphasia from hemorrhage, though less frequent than that from softening, is also frequent. Tumor is not an infrequent cause. In children tubercular meningitis is frequently operative in producing

motor aphasia, for the irritation of meningitis seems competent to inhibit the speech centre as it does the motor centres.

Sensory speech processes subserve the reception and revival of language. It is chiefly through the senses of hearing and sight that these processes are brought into activity, and there is consequently a close connection between these senses and the disturbances of speech which depend on the arrangement of the sensory processes, auditory and visual, of speech. Such disturbances of speech are designated *sensory aphasia*, the leading varieties of which are word-deafness and word-blindness.

In *Word-Deafness* words that are spoken by another are not understood. This does not depend on deafness in the ordinary sense; the patient hears the spoken words, but they are meaningless to him. Words of his own language sound strange; there is failure to associate these words with the ideas they represent. Word-deafness depends on disease of the posterior half of the first temporal convolution, usually of the left side. This is, therefore, the situation of the structures that subserve the auditory perception of words, but it is also the situation of the auditory centre itself. Whether the two centres, the auditory perceptive centre and the auditory centre, are identical it is impossible to say. They are at present indistinguishable. When this region of the cortex is diseased there is usually, in addition to word-deafness, transient deafness in the ear opposite the lesion.

When word-deafness exists there is always distinct derangement of speech. This is because the revival of the memory of words in the mind, before their arrangement in the motor speech centre, previous to utterance, occurs mainly in the auditory perceptive centre of the cortex. Without this ability to revive past auditory impressions

the patient's memory for words depends on his faculty for reviving the motor processes for words which have left behind changes in the cortical cells (residual states) which facilitate this revival—that is, he has to fall back on what may be called his motor memory. This is imperfect, and errors in the form and use of words occur even when the word-deafness is only partial.

Word-deafness is rarely absolute, because the auditory perceptive centre is habitually aided in the automatic revival of words by corresponding centres in the right hemisphere. Considerable recovery of the ability to understand spoken words and of the voluntary revival of words usually occurs in the course of time, and this is due to the education of the latent powers of the right-sided centre.

In word-deafness the patient is not conscious of the errors of speech which he makes. In this respect there is a striking contrast with motor aphasia, in which the patient not only appreciates his errors but tries to correct them.

Word-Blindness is the inability to comprehend the written symbols of language. There is usually no loss of sight ; the patient sees letters and words, but he fails to recall to mind the ideas they represent. The position of the lesion that gives rise to word-blindness cannot at present be definitely located. The symptom may be produced by a lesion in the inferior parietal lobule, and perhaps also by lesions involving the angular gyrus or the occipital lobe of the left side. In word-blindness there is usually some error in the use and form of words, but in some cases this is very slight. Word-blindness and word-deafness often coexist. Usually the power of speaking is retained.

Mind-Blindness (" soul-blindness ") is a rare condition in which, without mental defect, there is entire loss of visual memory of every kind. The patient no longer

recognizes familiar faces or objects. There is also loss of recognition of visual word symbols—that is, the loss includes word-blindness, which may be regarded as partial mind-blindness. The lesion in cases of mind-blindness is probably in the posterior part of the parietal lobes and in the occipital lobes, anterior to the half vision centres (p. 50). Probably permanent mind-blindness is due to bilateral involvement of these areas. Sometimes hemianopsia is associated with mind-blindness.

Mind-Deafness is a rare condition in which, without mental defect, there is loss of auditory memory. This symptom is especially striking in musicians, who may lose the faculty of appreciating music or following melodies, or of playing or following notes.

The word *Apraxia* is used as a general term to designate an inability to recognize the meaning or use of an object. Hence there are as many varieties of apraxia as there are modes of special sensation. Both mind-blindness and mind-deafness are varieties of apraxia.

Alexia, or inability to read *understandingly*, is of course an essential feature of word-blindness. It is important, however, to recognize the fact that alexia may result from a pure motor aphasia. In many persons, particularly those who are imperfectly educated, motor speech processes are essential to the comprehension of written speech symbols ; a kind of internal speech goes on while these persons are reading, the external evidence of which is the movement of the lips which is often seen in these persons. When these persons are deprived of their motor word-processes by disease, alexia results. The alexia gradually lessens, however, because some motor word processes are regained, and serve to reinforce the visual centre. When the alexia is due to word-blindness the inability to comprehend visual word

symbols is absolute, and the motor speech processes are generally unimpaired.

Dyslexia is a term applied to a rare symptom consisting in a peculiar condition of mental fatigue from reading, which makes its appearance a few minutes after the patient begins to read, and makes it impossible to read more than a few words consecutively. There is no motor speech disturbance. This condition is not associated with any visual defect, and is unaccompanied by ocular pain. It is thought to depend on a subcortical lesion beneath the angular gyrus.

Amnesic Aphasia, or difficulty in voluntarily recalling words, particularly those which, like nouns, are special in character, may result either from partial word-deafness or from pure motor aphasia. It is claimed that in cases where amnesic aphasia is the only form of speech defect from the beginning it constitutes a distinct variety of aphasia, and depends on a distinct pathological condition. This condition is supposed to be a lesion which interrupts the conducting path between the motor and auditory centres, and thus prevents that action of the auditory on the motor speech centre which is essential to the perfect revival of motor word-processes. For this reason it has been termed, somewhat unfortunately, "conduction aphasia" ("Leitungs Aphasie"). It cannot be said that the evidence on the strength of which amnesic aphasia is considered a distinct variety of aphasia is as satisfactory as could be desired.

Speech disturbances are not always due to organic disease of the brain. They occur not infrequently as an expression of functional derangement of the cerebral centres. Thus, transient motor aphasia often occurs after right-sided convulsions from any cause, probably from the exhaustion of the motor speech centre which results

from the discharge of nerve force. In migraine slight motor or sensory aphasia is not very uncommon during the attacks, especially in right-handed persons, when there is some sensory disturbance (tingling, numbness) in the right limbs, particularly the arm. Partial motor aphasia occasionally occurs during emotion or excitement, in persons (women) of hysterical character. In severe cases of typhoid fever motor aphasia, usually transient, has many times been noted. In some of these cases it was unquestionably of organic origin ; in others it has occurred under conditions which make probable its dependence on defective cortical nutrition from the morbid blood state.

Congenital aphasia, motor or sensory, is exceedingly rare, and when it exists probably depends on traumatic meningeal hemorrhage during labor.

The ability of an aphasic patient to make a will may constitute an important practical question. In order that a valid will may be made, it is necessary that the testator should be able (1) to understand the proposition that is put before him, and (2) that he should be able to express assent or dissent in an unmistakable manner. If there is word-deafness it is impossible to say with certainty that the patient understands what is said to him, and hence a valid will cannot be made if what is put before the testator is spoken. If, however, the patient understands written words, and propositions are communicated in writing only, word-deafness is no obstacle to the making of a will. The coexistence of word-deafness and word-blindness of course destroys all testamentary capacity. Motor aphasia, even absolute in degree, does not destroy testamentary capacity if spoken words are understood and if the testator can indicate assent and dissent by means of signs.

General Symptoms.— *Temperature.*—Elevation of temperature is of frequent occurrence in nervous disease. The fever may be simply an associated condition—that is, the morbid process in the nervous system and the elevation of temperature are referable to a common cause. This is particularly the case with processes that are due to infection—for example, some forms of multiple neuritis, poliomyelitis, cerebral embolism, etc. In some conditions it is difficult to say to what extent the fever is due to the general condition or the morbid process in the nervous system. This doubt arises, for instance, when tubercular meningitis and general tuberculosis occur together. The fever may be dependent chiefly on the character of the process without particular reference to its situation in the nervous system. Acute inflammatory processes in any position are attended with fever, which is generally to be regarded as consecutive to the inflammation. In the case of intracranial disease an important peculiarity is frequently observed: There may be only slight rise of temperature or even no rise of temperature in the course of inflammatory processes of considerable or great severity. This is true, for example, in some cases of meningitis. This peculiarity probably depends on the inhibitory influence of irritative processes on the heat centres of the medulla. It is important to remember that the absence of fever or its slight degree does not indicate the absence of inflammation within the cranium.

Sometimes the pyrexia that results from central nervous disease depends, not on the nature of the pathological process, but upon its direct action on the heat centres, or the apparatus which controls them. This is particularly the case with acute lesions (hemorrhage, softening) in the upper part of the cervical cord, medulla, pons, or optic

thalami. In these cases there is often a rapid and great rise in temperature—the fever may rise to 103° or to 105°, and in the case of medullary and pontine lesions even higher (106°–108°). Death usually follows such an elevation of temperature within a short time. In some cases of sunstroke very high temperatures are seen (108°–110°), and there is often a great premortal rise in cases of meningitis. In the case of sunstroke the hyperpyrexia does not necessarily foreshadow a fatal termination.

Sometimes the rise of temperature is the secondary effect of nervous disease, particularly disease of the brain. Any condition—for example, tetanus, epilepsy—which gives rise to many successive attacks of convulsions is followed by a rise, often considerable, in temperature. This is due apparently to the excessive muscular action, aided possibly by the accumulated and retained products of extraordinary tissue destruction, caused by the convulsions. During the "status epilepticus"[1] (p. 576) the temperature may rise to 106°–109°. There is not uncommonly a post-mortem rise in temperature in status of epilepsy, general paresis, uræmia, and in many other states terminated by a series of convulsions.

A subnormal temperature sometimes occurs in brain disease, rarely in disease of other parts ot the nervous system. The process which gives rise to it is generally hemorrhage, the depression of temperature occurring usually at its onset. If the extravasation of blood be great, the temperature may fall to 95°, 93°, or even 90° (rectal). In such cases there are the usual signs of collapse. A slight initial fall may be succeeded by a rise in temperature.

Pulse.—Most forms of organic nervous disease have no

[1] In this state the paroxysms recur at short intervals for a considerable time (often many hours or days).

special effect on the rapidity, force, or rhythm of the pulse. Inflammatory intracranial disease may inhibit the pulse rate even when there is a considerable elevation of temperature, and it is not uncommon in meningitis to meet with a temperature of 103° or 104° and a pulse rate which is normal or even less than normal. A higher temperature from intracranial disease is generally associated with a soft and rapid pulse. Infrequency of the pulse rate (50–40–30 per minute) is sometimes observed at the onset of acute lesions (hemorrhage, softening). A habitually rapid pulse rate (90–120 per minute), without fever, is not uncommon in neurasthenia and some forms of hysteria. Marked irregularity of pulse is often an important early sign of irritative intracranial disease, such as tumor.

Respiration is markedly influenced by organic disease of the nervous system chiefly when this is extensive (hemorrhage, softening), or when it involves the parts in the neighborhood of the respiratory centre in the medulla. In such cases respiration is often exceedingly irregular, both as regards rhythm and rate, and may eventually cease from exhaustion of the respiratory centre. Sudden cessation of respiration occurs from a sudden lesion (hemorrhage, thrombosis, embolism) in the respiratory centre of one side or both sides, and may occur from inhibition of this centre from acute lesions adjacent to the centre. When the lesion is external to but near the respiratory centre, the depression of respiration may continue many hours, and then disappear spontaneously. Irregularity in the rhythm and force of the respiratory movements is often observed in inflammatory cerebral lesions. It is especially frequent in acute meningitis, and is so common and so decided in most cases of tubercular meningitis as to be rather characteristic of this disease.

The paradoxical respiratory sign (page 90) is to be found in all capsular lesions of hemiplegia.

Cheyne-Stokes respiration (peculiar rhythmical changes in breathing, characterized chiefly by the alternation of periods during which there is a gradual and extreme diminution in the force and frequency of the respiration, with periods of increasing depth of breathing, these periods being separated by a variable interval of time—5–40 seconds) is sometimes observed toward the close of cerebral diseases (hemorrhage, meningitis, tumor, and status epilepticus). The patient is usually comatose and rarely recovers when this form of breathing has begun. Cheyne-Stokes breathing occurs in many diseases external to the nervous system (heart disease, pneumonia, nephritis, diphtheria, etc.), and under these circumstances is of less grave significance than when due to brain disease. Its explanation may rest upon the bilateral withdrawal of cerebral inhibitory influence upon the medullary centres.

Mucus accumulates in the bronchi and trachea in states in which the cerebral functions are in abeyance, for example, in the course of prolonged coma. The bronchial and tracheal râles which result must not be mistaken for bronchitis. Passive congestion of the lungs and hypostatic pneumonia are of common occurrence under the same circumstances, and constitute a frequent cause of death in central nervous disease.

Of the symptoms referable to the *Digestive Organs*, *Vomiting* is by far the most important. As an effect of organic nervous disease vomiting is chiefly observed in intracranial disease, although it is not uncommon as an effect of acute processes in the upper cervical cord, and occurs paroxysmally in some chronic general diseases of the spinal cord (locomotor ataxia).

Vomiting occurs as a symptom of organic disease of the brain in many different conditions—in meningitis, tumor, abscess, and not infrequently in cerebral hemorrhage—

that is, in irritative conditions. Sometimes it occurs repeatedly in the course of endarteritis of vessels of the base, before complete obstruction takes place. Generally speaking, any condition giving rise to considerable increase in intracranial pressure may occasion vomiting. The situation of the lesion does not seem to exert any important influence in determining the symptom, excepting in the case of cerebellar lesions, which are particularly apt to give rise to it.

Vomiting of cerebral origin cannot be said to possess any features that are to be regarded as highly distinctive. Still, certain characters, chiefly negative, of such vomiting are sufficiently frequent to be suggestive. These are: (1) The rejection of food soon after its ingestion; (2) the absence of discomfort after eating; (3) the absence of nausea; and (4) the absence of other gastric symptoms. In general the vomiting of functional nervous disease (hysteria) possesses the same negative characteristics. Hence it is that the diagnostic significance of vomiting depends mainly on its associations. The organic processes which give rise to vomiting are exceedingly apt to cause persistent headache, or optic neuritis, or both. The association, therefore, of either of these symptoms and, *a fortiori*, of both of them, with frequent vomiting, makes intracranial disease probable.

The vomiting of intracranial disease depends on an increased excitability of the gastric centre in the medulla. When this exaltation of excitability is great, vomiting may occur spontaneously—that is, without any traceable gastric cause. Before this high degree of irritability is reached, slight peripheral causes, such as dietetic indiscretion, may cause vomiting more readily than would be the case in the absence of this increased irritability. Hence it is easy to understand the vomiting which may be the first indication of organic disease. Every patient,

and particularly every child, who vomits without experiencing the indisposition that generally attends vomiting from gastric cause, should be carefully watched. The vomiting which is associated with vertigo has already been mentioned.

Constipation usually accompanies cerebral and spinal-cord diseases which are attended with elevation of temperature, but has little diagnostic significance. Obstinate constipation is almost a constant symptom of destructive disease of the cord above the lumbar enlargement.

The pathological changes that occur in *The Urine* as the result of nervous disease are few, and, generally speaking, of little help in diagnosis. Transient albuminuria and glycosuria are occasionally observed in disease of various parts of the brain, and particularly in lesions of the medulla and pons. Much more frequently albuminuria is associated with diseases of the cerebral vessels (endarteritis, hemorrhage, atheroma, miliary aneurisms) which result in cerebral hemorrhage or softening. Other urinary changes (oxaluria, excessive or insufficient excretion of uric acid, increase in indican, etc.) are common in nutritional diseases of the nervous system, as neurasthenia, some forms of headache, etc. These changes are, as yet, not sufficiently understood to be of material aid in diagnosis, but it is likely that in the future they will prove of considerable help in the diagnosis of the nature of nutritional diseases of the nervous system.

Affections of the *Sphincters of the Bladder and Anus* are of frequent occurrence in organic disease of the nervous system, but are not common from disease of the peripheral nerves external to the spinal column. Interruption of the voluntary path (probably the pyramidal tract) from the brain cortex to the reflex centres for the bladder and rectum in the lumbar enlargement of the cord (lowest lumbar and sacral segments) results in the loss of volun-

tary control of the functions of these viscera. Their action becomes purely reflex. As soon as a sufficient amount of urine accumulates in the bladder, the viscus is emptied by the reflex contraction of the detrusor urinæ and synchronous relaxation, also reflex, of the sphincter; when fæces accumulate in the rectum and irritate its mucous membrane, they are expelled by an analogous mechanism. The incontinence of urine is called "intermittent" or "reflex incontinence." It is important to understand that there is no paralysis of the sphincters in this form of incontinence. The proof of this is (in the case of the rectum) that the introduction of the finger into the rectum is followed, after slight relaxation, by firm and tonic contraction of the sphincter. The interruption of the voluntary path which results in this loss of cerebral control may result from different conditions; generally from

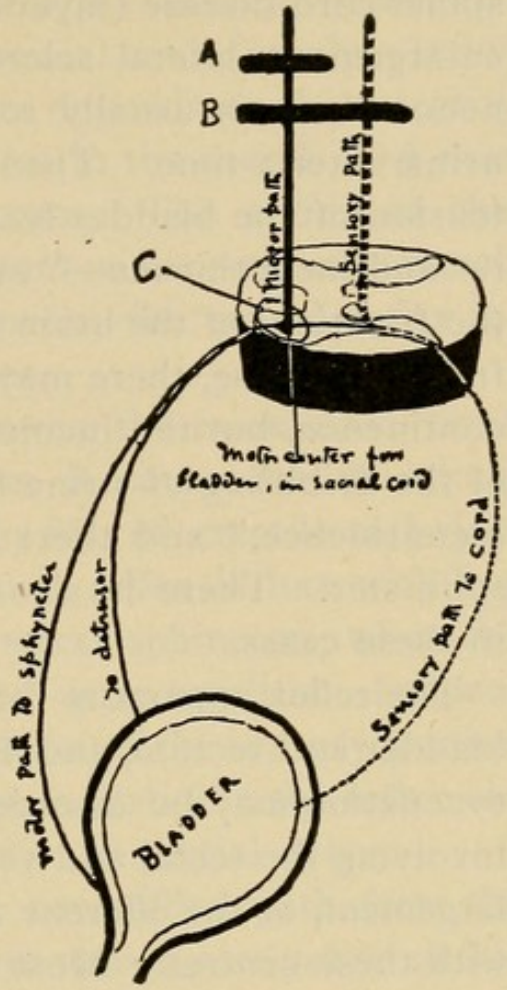


FIG. 70.

Diagram illustrating the innervation of the bladder and the effect of lesions in various parts of the spinal cord upon the function of micturition. A lesion, A, which interrupts the voluntary path to the bladder centre in the sacral cord, causes incontinence of urine; when a sufficient quantity of urine accumulates in the bladder there occurs a reflex contraction of the detrusor and relaxation of the sphincter. The sensory path from the cord to the brain being uninvolved, the patient is conscious of the process, but cannot exercise voluntary control over it. With a lesion, B, which involves also the sensory path, the patient is unconscious of the filling and reflex emptying of the bladder. A lesion, C, which causes destruction of the sacral reflex centre for the bladder causes continuous dribbling of urine, and not its automatic expulsion at intervals.

spinal-cord disease (myelitis or injury above the lumbar enlargement, lateral sclerosis, etc.). In reflex incontinence there is usually some weakness of the detrusor urinæ after a time. Then retention occurs, and the distension of the bladder leads to relaxation of the sphincter and incontinence—"overflow incontinence." When the functions of the brain are in abeyance (stupor, coma) from any cause, there may be retention of urine and incontinence, but the incontinence consists in these cases of the dribbling of urine from a full bladder, "overflow incontinence," and there is no actual weakness of the sphincter. There is also involuntary passage of fæces in these cases.

The reflex apparatus for expelling the contents of the bladder and rectum, and for preventing their continuous evacuation may be disordered in its function by disease involving the rectal and vesical centres in the lumbar enlargement, or the efferent and afferent nerves connected with these centres. Most frequently the disturbance of function depends on destructive disease of the lumbar centres. In this condition fæces or urine are passed as soon as they enter the rectum or bladder. The urine dribbles away continuously; the condition of the rectal sphincter can be determined by the introduction of the finger, which shows it to be persistently relaxed. When there is partial damage to the lumbar centres, of gradual development, there is often difficulty in exciting the action of the detrusor (patient "cannot start his urine"), and this may result in retention. Damage to the cauda equina sometimes produces disturbances like those that result from partial disease of the lumbar centres for the bladder and rectum.

Disorders of the Sexual Functions are of frequent occurrence in nervous diseases, but do not possess much diagnostic importance, and will be only briefly touched

upon. They are more common as the result of functional than of organic disease. The sexual act is largely a reflex act, and its perfect performance depends on the integrity of a reflex arc whose centre lies in the sacral cord near that for the bladder and rectum. The action of this centre is, however, to some extent under cerebral control, and in this, as in other respects, the sexual reflex resembles closely the skin reflexes.

Disease of the sexual centre in the sacral cord, or of the nerves leading to or from it, causes loss of sexual power, as shown in the male by loss of erectile and ejaculatory power. Thus the sexual reflex is lost with loss of control over the anal and vesical sphincters, when the lumbo-sacral cord is destroyed, as in myelitis. In locomotor ataxia it is lost from disease of the sensory portion of the arc. When the damage is partial in the reflex arc the corresponding loss of function is partial only.

Disease which cuts off the controlling path from the brain to the reflex sexual centre causes *over-action* of this centre with resulting priapism. This is seen in dorsal myelitis, compression and crush of the cord, etc. In women various menstrual disorders may accompany organic disease, especially cord disease. Periods of great sexual excitement occur in some women with locomotor ataxia (so-called "clitoris crises"). A great variety of sexual disturbances, including increased and diminished sexual desire, spermatorrhœa, and priapism, are observed in functional disorders (hysteria, neurasthenia, traumatic neuroses). In some cases of hysteria deep pressure over the ovarian region, especially the left, causes severe and characteristic pain, often with nausea and vomiting, and perhaps excites an hysterical paroxysm.

CHAPTER III.

THE DIAGNOSIS OF THE POSITION OF THE LESION—LOCALIZATION.

It is convenient to think of all morbid processes affecting the nervous system as divisible into two great groups —those which consist of changes in the cell-elements, that are demonstrable by present methods of investigation (organic diseases), and those that consist of alterations of variable degree in the nutrition of these elements, but are unassociated with demonstrable structural changes (nutritional or "functional" diseases). These two groups merge into one another insensibly, both in their pathological and clinical characters, but for practical purposes their separation is desirable, and the general principles on which the clinical distinction of organic and functional disease is based are elsewhere presented (p. 556).

This distinction, which occasionally constitutes a problem of the greatest difficulty, ordinarily is made with ease, and having been made, at least provisionally, should be followed by a systematic endeavor to obtain more accurate knowledge of the morbid process. In general, an effort should be made—first, to locate as accurately as possible the seat of the morbid process within the nervous system (local diagnosis); and, secondly, to determine the pathological nature of this process (pathological diagnosis).

In the case of organic disease on the nervous system it is of great practical importance to locate accurately, *i.e.*, to "localize" the lesion (which includes an estimate of its extent), because this information not only is an important guide to prognosis, but may furnish the most important indications for treatment.[1]

Fortunately the data now at command enable a local diagnosis to be made with precision in a large majority of all cases of organic nervous disease.

In the case of functional disease of the nervous system, the localization of the morbid process (nutritional), though desirable, is much less important, since the prognosis and treatment depend on other indications. It is also much less practicable, and is at best uncertain, since the localization depends on inferences based on what is known of the effects of organic lesions.

It is inferred, for example, that certain hysterical paralyses depend on disease (nutritional) of the motor cortex, because it is known that organic disease of the motor cortex gives rise to symptoms which present a close resemblance (segmental distribution of palsy, contractures, normal electrical reaction) to the characters observed in functional palsy. The following review of the chief manifestations produced by disease in various parts of the nervous system relates exclusively to the effects of organic processes. The chief symptoms of nervous disease, and the parts concerned in their production, have already been considered, and it is necessary now to look at these facts from another point of view—to consider the symptoms from a regional standpoint. It is convenient to consider, first, the chief symptoms that result from lesions in various parts of the brain; secondly, the symptoms produced by disease in differ-

[1] This relates especially to operative treatment.

ent regions of the spinal cord, and in the peripheral nerves.

a. **Cortex Cerebri.**—1. Destructive disease of the *Central and Motor Area* of the brain (anterior and posterior central convolutions and paracentral lobule, anterior half of superior parietal lobule) gives rise to hemiplegia on the *opposite* side of the body.

The hemiplegia is usually incomplete, because the lesion more often involves a small than a large part of the central area. Partial hemiplegia (monoplegia) occurs much more commonly from disease of the cortex than from disease more deeply seated in the motor path. Hence, a partial paralysis, if due to brain disease, points to the existence of a cortical lesion.

The parts that suffer from the palsy vary according to the position of the lesion. The leg suffers alone chiefly when the lesion involves the paracentral lobule, or the central convolutions close to the longitudinal fissure. If the lesion is confined to that part of the leg centre which lies in the upper part of the anterior central convolution, the paralysis probably affects mainly the foot. If the lesion lies between the longitudinal fissure and the junction of the middle with the lower third of the central convolutions, there is paralysis of the arm, and if the lesion involves the middle third only of these convolutions, paralysis of the arm alone. A lesion in the upper part of the arm centre paralyzes the shoulder more than the rest of the arm. A lesion in the very lowest part of the anterior central convolution gives rise to paralysis of the tongue and lips. Any slight defect in articulation that may result from a unilateral lesion in this situation is transient, being soon compensated by the corresponding centre in the opposite hemisphere.

In cortical disease the lesion is not usually confined to

one of the functional areas mentioned (area for the arm, or leg, or face), but involves adjacent areas. Hence it is that paralysis of the arm and leg, or of the face and arm, is more often encountered as a result of cortical disease than paralysis of one of these parts alone. If a destructive lesion is not confined strictly to the cortex (and it usually is not), it may involve subjacent white tracts from other regions of the cortex which are not the seat of disease, and thus give rise to symptoms not referable to the area which is the seat of the cortical lesion.

In addition to motor paralysis, lesions of the cortex give rise very often to convulsions. This is the case with lesions that are purely irritative in character and with those that are destructive and stationary but in which the destruction is partial only. The convulsion begins locally in the leg, arm, or face, according to the position of the disease which causes it, in or adjacent to the area whose destruction gives rise to motor paralysis. Such local commencement is highly suggestive of cortical disease. If there is reason to regard the lesion as stationary—that is, no longer active and incapable of further structural recovery, the recurrence of local convulsions makes the existence of cortical disease certain. Their recurrence under these conditions, however, does not necessarily mean that the region of the cortex corresponding to the locality of the initial spasm is the part most diseased. A lesion, for example, situated on the leg centre may give rise to convulsions beginning in the face or arm centre, because the disease is capable of starting discharges from adjoining areas which are less diseased but more excitable.

Localized convulsion, therefore, is to be regarded as a less accurate indication of the exact seat of the lesion than localized motor paralysis. This an important point.

Disease of the central area of the brain very often gives rise to loss of sensation in the extremity of the part (or parts) paralyzed. The loss of sensibility is generally slight in degree. It involves chiefly tactile sensibility, chiefly marked at the extremity of the affected limb, but there may be some loss of pain and temperature sense. Complete hemianæsthesia probably never results from lesions confined to the motor area. The sense of posture of the limbs paralyzed is sometimes impaired in some degree by lesions of the motor cortex (especially its posterior part), but there is sometimes a similar loss from disease of the motor path between the cortex and the lower part of the internal capsule. Lesions of the posterior superior parietal lobule also cause impairment of the sense of posture.

2. Lesions of the *Frontal Lobe* of the brain, anterior to the motor area, give rise, with one exception, to no distinctive focal symptoms. When there is disease of the posterior part of the left third frontal convolution in right-handed persons there is motor aphasia. A lesion in the corresponding position in the right frontal lobe produces the same effect in left-handed persons. Extensive disease of the cortex usually, but not invariably, causes considerable mental impairment, as shown by loss of memory and attention, and sometimes by emotional disturbance. Similar mental defect may result from extensive cortical disease in other parts of the brain, but a lesion of equal extent probably causes less impairment than is produced by disease of the frontal lobe. Disease in the posterior part of the second frontal convolution perhaps gives rise to conjugate deviation of the eyes and head (page 109). Disease involving the frontal lobes in such a way as to damage the olfactory bulb or tract causes anosmia on the side of the lesion.

3. The precise effects of lesions of the *Parietal Lobe*

posterior to the motor area are still undetermined. Lesions of the posterior portion of the superior parietal lobule give rise to loss of the sense of posture in the limbs of the opposite side. Disease of the inferior parietal lobule probably causes ptosis on the opposite side, but the exact area on which this depends is not known. It is questionable whether lesions of the angular gyrus which are limited to the cortex are competent to cause crossed amblyopia, but it is probable that the destruction of this region on the left side causes word-blindness (p. 217). Destructive disease of the entire parietal lobe generally impairs, though it does not abolish, sensibility of the opposite side of the body (trunk and limbs). Lesions involving both the parietal lobe and the motor area give rise to more complete hemianæsthesia than is produced by a lesion limited to either region.

4. Lesions of the cuneus of the *Occipital Lobe* cause homonymous hemianopsia. Lesions involving the upper and lower half of the cuneus, respectively, are probably capable of producing partial hemianopsia—that is, quadrantic homonymous defects (p. 165). It is possible that disease just anterior to the half-vision centre (cuneus) or in its anterior part may give rise to color hemianopsia.

Disease of the external surface of the occipital lobe seems capable of causing mind-blindness, which is often associated with hemianopsia from involvement of the subjacent visual tract.

5. Lesions involving the first and second temporal convolutions of the *Temporo-Sphenoidal Lobe* of the left side give rise to word-deafness. Disease of the posterior part of the first temporal convolution probably gives rise to transient deafness in the opposite ear, and an irritative lesion in this situation may give rise to an auditory aura.

The auditory centre in man cannot at present be very accurately located.

A lesion in the uncinate convolution, or involving the apex of the temporo-sphenoidal lobe may cause marked disturbance of the sense of smell. Irritative disease in this situation may determine an olfactory aura.

6. Disease of the *Island of Reil* has many times been associated with hemiplegia, and disease extending through the insula of the left side has been considered effective in producing aphasia by interruption of the path between the motor and auditory speech centres. It is doubtful whether hemiplegia is ever the effect of a lesion limited to the cortex of the insula, although disease of the branches of the middle cerebral artery, as they pass over the insula, may undoubtedly lead to lesions elsewhere (motor cortex and subjacent white substance) that produce motor paralysis. It also remains to be shown that simple interruption of the path beneath the insula, above mentioned, is competent by itself to produce amnesic aphasia.

b. **White Substance and Centrum Ovale.**—Disease of the white substance of the hemisphere and centrum ovale gives rise to symptoms referable, on the one hand, to the interruption of the various association tracts which connect different gyri and functional areas of the cortex with one another, and, on the other, to interruption of tracts which have a peripheral destination (projection tracts). Lesions of the association tracts, if extensive, probably cause mental symptoms chiefly, loss of memory and attention, defective will-control, etc. It is doubtful whether any lesions of this kind give rise to symptoms that possess localizing value. Lesions of the tracts which connect the cortex with peripheral nervous mechanisms (retina, motor apparatus, etc.) produce symp-

toms which resemble those that result from disease of the region of the cortex with which the particular tracts are connected. Thus, a lesion of the white substance of the occipital lobe, involving the visual path to the cuneus, gives rise to lateral hemianopsia; a lesion interrupting the fibres that converge from the central area of the cortex to the internal capsule gives rise to hemiplegia. The various tracts mentioned all converge from different areas of the cortex, widely separated, to the internal capsule. Hence, generally speaking, lesions *just beneath* the cortex produce symptoms like those dependent on disease of the overlying cortical region, while those that approach the internal capsule produce effects like those of capsular disease. General convulsions are not common in disease of the white substance of the hemisphere, even when such disease is irritative in character, unless it be placed just beneath the cortex. When in this position (particularly when the lesion is beneath the motor cortex) an irritative lesion (as tumor) often gives rise to the local spasm (Jacksonian spasm) which is so characteristic of cortical disease. The motor aphasia which follows interruption of the path from the motor speech region (third frontal gyrus) to the internal capsule is temporary, except when the interruption is just beneath the cortical centre. Conjugate deviation of the eyes is common from subcortical lesions.

c. **Corpus Callosum.**—Lesions are so rarely limited to the corpus callosum that the diagnostic indications of its damage cannot as yet be formulated. It is evident, however, from its structure that it associates the action of the two hemispheres. Bilateral paresis of the extremities has been noted in many cases where the callosum was damaged, but this is doubtless referable to pressure upon or extension into the motor tract in the cerebral hemispheres. There is some reason to believe that

destruction of the anterior extremity of the callosum gives rise to mental dulness, loss of memory, and other evidences of impaired mental power, somewhat similar to that noted in lesions of the frontal lobes.

d. **Corpus Striatum.**—Disease of the corpus striatum (caudate or lenticular nucleus) causes no symptoms that can be referred to damage of its gray substance. Acute lesions limited to either nucleus may produce transient hemiplegia, but this symptom is doubtless due to irritation of the adjacent fibres of the internal capsule. Chronic disease of the nuclei produces no symptoms, unless the motor or sensory fibres of the internal capsule be damaged also.

e. **Optic Thalamus.**—Most of the symptoms observed in lesions of the optic thalamus are referable to involvement of the contiguous internal capsule and not to the direct effect of the thalamic disease. Thus, the hemiplegia which often follows disease of the middle third of the thalamus is due to implication, often slight, of the motor fibres of the capsule, which lie just external to this part of the ganglion. Similarly, the hemianæsthesia which is observed to accompany disease of the posterior extremity of the thalamus (pulvinar) depends not on the thalamic lesion, but on damage to the sensory part of the capsule. Disease of the middle and posterior thirds of the thalamus seems to be directly effective in producing inco-ordination and hemiathetosis of the opposite extremities, but it cannot be denied that these symptoms may be due, at least in part, to irritation of the neighboring motor tract. It is highly probable that lateral hemianopsia may be produced by a destructive lesion limited to the pulvinar of the thalamus, and that the symptom is then to be regarded as the direct result of this destruction. The association of hemianopsia with hemianæsthesia indicates a lesion of the posterior part of the thalamus and the adjacent sensory fibres of the internal capsule.

The so-called basal ganglia probably preside over many acts which are commonly performed in a purely automatic manner, such as bodily posture, ordinary gait, facial expression, and probably emotional control.

f. **Internal Capsule.**—Lesions of the internal capsule give rise to symptoms which vary much according to the part of the capsule involved. Disease limited to the *anterior division* of the internal capsule, anterior to the knee (between the caudate and lenticular nuclei), is rare, and does not give rise to characteristic symptoms. Sometimes mental symptoms, similar to those referable to disease of the frontal lobe, are produced by disease of the anterior part of the capsule, but usually it seems to cause no definite effects. Disease involving the *angle* (knee) and the *anterior two thirds of the posterior division* of the capsule causes hemiplegia of the ordinary variety—that is, paralysis of the opposite leg, arm, and face (lower part). If the lesion is on the left side there is frequently motor aphasia, but this is always transient. If the lesion be of acute onset there is often at first conjugate deviation of the eyes, to the hemiplegic side where the symptom is due to irritation. Sometimes there is permanent conjugate deviation. This is usually due to destruction, and the deviation is then to the side of the lesion (*vide* p. 109). Convulsions occasionally attend the onset of hemiplegia from disease in this region. They seldom recur after the onset. Hemiathetosis often follows incomplete hemiplegia, but it is not certain whether the symptom is referable to the capsular disease or to adjacent disease of the thalamus.

The hemiplegia of disease of the internal capsule is usually complete. A very small lesion, limited to the angle of the capsule, may give rise to facial paralysis only, and, similarly, minute lesions in the anterior and middle thirds respectively of the posterior division of the capsule, may produce a brachial monoplegia and a crural

monoplegia. It is exceedingly rare, however, for a paralysis from such lesions to involve exclusively face, arm, or leg, although one of these parts may be chiefly affected.

Probably three fourths of all cases of hemiplegia result from disease of the internal capsule; hence, in the absence of symptoms pointing to another locality as the seat of the lesion, there is always a strong presumption that a hemiplegia depends on damage to the internal capsule.

Disease of the posterior third of the posterior division of the internal capsule, in which lies the sensory tract, gives rise to hemianæsthesia. Taste, hearing, and smell may be lost on the anæsthetic side, as the paths for the special senses are contiguous to the path for common sensibility. The visual path is also involved in such cases and the resulting symptom is lateral hemianopsia.

g. **External Capsule and Claustrum.**—Lesions of the external capsule and claustrum do not give rise to distinctive symptoms and cannot therefore be localized.

h. **Corpora Quadrigemina.**—Lesions limited to the corpora quadrigemina are of exceedingly rare occurrence. Hence there is still considerable doubt as to the precise effect of disease in this situation. Disease of the *anterior pair* has been thought to produce blindness, loss of pupillary reflex and nystagmus. It is certain that there is no adequate reason to suppose that blindness is ever directly referable to disease of the corpora quadrigemina. Irritative disease (tumor) of the quadrigeminal region, like such disease elsewhere, may give rise to optic neuritis and secondary optic atrophy, and by this mechanism to amblyopia, but, except in this manner, visual acuity is not impaired. There has been no impairment of vision even in cases where the corpora quadrigemina were wholly destroyed. The condition of the pupillary reflexes varies so much in different cases of quadrigeminal disease that no general statement can be made regarding it.

It is probable that in some cases of such disease the reaction to light is lost. Nystagmus may occur from irritative disease of the quadrigeminal region as from irritative disease in other regions. It probably bears no particular relation to the anterior pair, and, as elsewhere stated (p. 129), has no localizing value. Disease of the *posterior pair* has many times been associated with inco-ordination of movement (unsteadiness in walking and standing, reeling or staggering gait), and is probably, in some degree, a highly constant symptom of such disease. It must be owned that there is still some doubt as to whether the symptom is really due to the quadrigeminal disease and not to implication of the middle lobe of the cerebellum, but the balance of evidence is much in favor of the former view. Paralysis of the muscles supplied by the oculomotorius nerve is a frequent and important symptom of disease of the quadrigeminal region. It doubtless depends on implication of the subjacent nuclei of the third nerve and not on the quadrigeminal disease itself. Usually the muscles are neither symmetrically nor equally involved in the paralysis, nor are they all implicated.

While it is impossible, therefore, to localize a lesion of one or other pair of corpora quadrigemina, disease of the quadrigeminal region may frequently be diagnosed with accuracy. The symptoms on which such a diagnosis can be made are the coexistence of inco-ordination of movement (of early development usually) and unsymmetrical and incomplete ophthalmoplegia referable to the oculomotor nucleus.

i. **Crus Cerebri.**—Lesions of the crus cerebri (pes) frequently cause a characteristic combination of symptoms—hemiplegia, involving the lower half of the face and the limbs, and paralysis of the third nerve on the side of the lesion—that is to say, on the side opposite the hemi-

plegia (crossed or "alternate" hemiplegia). Usually the paralysis of the third nerve is complete; occasionally it is partial. It is important to bear in mind that if the process which results in this form of crossed hemiplegia is of acute onset, the lesion cannot be referred to the crus unless both hemiplegia and ocular palsy are of simultaneous occurrence. In the case of chronic processes the separate development of the two symptoms leaves a doubt as to whether or not they depend on the same lesion.

Convulsions are rarely observed in cases of disease of the crus. Irritative disease, however, may give rise to rigidity. If the lesion involves the sensory path in the tegmentum of the crus, anæsthesia may be associated with the motor paralysis. Occasionally (chiefly in irritative disease) there is lateral hemianopsia, from implication of the contiguous optic tract. Occasionally, also, there is unilateral inco-ordination.

j. **Pons.**—Lesions of the pons Varolii generally give rise to distinctive symptoms. Unilateral disease commonly involves the motor path to one side of the body and certain of the cranial nerves (fifth, sixth, or seventh) of the other side. Several different varieties of crossed hemiplegia may thus arise from unilateral pontine disease, according to the size and level of the lesion. Thus, the limbs may be paralyzed on one side and the face on the other—that is, on the side of the lesion—by a lesion which involves the facial path before it crosses and the motor path on the same side. In this case the entire half of the face is paralyzed and the muscles give the R D. If the lesion is near the median line and near the middle of the pons, it may be so placed as to involve the facial paths of both sides with the motor path of one side. Both the sensory and motor part of the fifth nerve, or either alone, may be involved. If the sensory part of the fifth is damaged by an irritative lesion there is severe pain on one

side of the face. If the nucleus of the sixth nerve of one side is involved in a destructive lesion there is conjugate deviation of both eyes to the side opposite to the lesion. This implies loss of function of the internal rectus muscle which habitually acts with the external rectus muscle (that is, the internal rectus of the *opposite* eye) which has been paralyzed by destruction of the nucleus of the sixth nerve. In conjugate movements of the eyes this loss of function in the internal rectus exists, although for other purposes (convergence) there may be no loss of power. Where the lesion is in the motor path of the pons at the level at which the sixth nerve traverses the pons in passing from its nucleus to the surface, the nerve-trunk is apt to be involved. Then there is hemiplegia with paralysis of the external rectus on the side of the lesion. The associated internal rectus of the other eye acts normally, and hence there is no conjugate deviation of the eyes.

A lesion may be so placed in the motor path of the pons that there is no coincident affection of any of the cranial nerves. In such cases the lesion is usually above the middle of the pons—that is, in a part of the pons in which the upward paths from the nuclei of the nerves in question have already crossed to the opposite side. The resulting hemiplegia cannot of course be distinguished from that which depends on capsular disease.

Bilateral disease of the pons is not rare and may give rise to a variety of symptomatic combinations. A lesion implicating the motor path of both sides may cause complete paraplegia. The cranial nerves escape or are bilaterally involved according to the size and level of the lesion. Bilateral disease of acute onset usually gives rise to apoplexy and often to rapid death.

Convulsions are uncommon in pontine lesions unless they are of acute onset. Sometimes the convulsions are

peculiar in distribution ; they affect chiefly both arms or both legs. Irritative processes involving both pyramidal tracts may give rise to rigidity of all the extremities. Tonic spasm is very often met with in partial disease of the motor path within the pons, and may correspond in distribution to the motor paralysis. Other motor symptoms referable to irritation (trismus and facial spasm, choreic movements) are rare.

Lesions involving the tegmental region of the pons, especially the formatio reticularis and root of the fifth nerve, give rise to loss of sensibility in the limbs, trunk, and face. A unilateral lesion thus placed in the sensory path in the upper third of the pons may cause complete hemianæsthesia on the side opposite the lesion. If the lesion is below the upper third of the pons and damages the sensory path from the face and limbs, there is "crossed" anæsthesia—that is, anæsthesia of the face on the side of the lesion and of the limbs on the opposite side (see page 145). There may be considerable pain in the anæsthetic side of the face in these cases. Conjunctivitis sometimes exists on the side of the lesion, and there may be unilateral rhinitis. These symptoms are apt to be present in acute processes which give rise to considerable irritation. Crossed anæsthesia is sometimes associated with inco-ordination of one or both lower extremities. Disease involving the sensory tract of both sides gives rise to bilateral anæsthesia, but this is seldom complete.

Unilateral disease limited to the formatio reticularis may cause only anæsthesia of the opposite limbs and trunk.

Disease of the posterior part of the pons may involve several of the cranial nerve nuclei and give rise to symptoms referable chiefly to their implication. A combina-

tion which is not very rare is paralysis of the sixth and seventh without the auditory. This is highly characteristic of pontine disease. Both the motor and sensory path from the limbs may be involved by a large unilateral pontine lesion, but the degree and extent of the anæsthesia and motor palsy are seldom proportioned. This depends on the fact that the two paths are separated from one another by the considerable interval occupied by the deep transverse fibres of the pons (see Fig. 4).

Acute irritating processes (especially hemorrhage), particularly in the upper part of the pons, often cause contraction of the pupils by their indirect effect on the oculomotorius nuclei. Disturbance of respiration, temporary but often grave, may result from acute lesions of the pons in almost any situation. Cardiac disturbance is less frequent. Deafness is uncommon in pontine lesions, and taste is not often affected. Sight may be impaired as a result of optic neuritis, never as a direct consequence of pontine disease. If the lesion encroaches on the middle peduncle of the cerebellum there may be a tendency to rotation to the side of the lesion in walking and severe giddiness at the onset of an acute process. Hyperpyrexia not infrequently follows acute lesions of the pons, and in itself suggests pontine or medullary disease. Lesions of considerable size in the pons sometimes cause much psychical disturbance.

The most frequent characteristic symptoms of pontine disease are the crossed motor paralyses already described. When such a paralysis occurs suddenly it is practically pathognomonic of a lesion of the pons. Crossed anæsthesia is much less frequent than the crossed motor palsies, and may result from disease of the upper part of the medulla, but its occurrence makes a pontine lesion practically certain. In the absence of any of these characteristic

paralyses the diagnosis may be difficult or even impossible. When possible, the diagnosis must be based on the peculiar symptomatic combinations that result from the involvement of neighboring structures which take their origin in the pons.

k. **Medulla Oblongata.**—Lesions of the medulla oblongata, like those of the pons, may give rise to a considerable variety of symptoms, according to the region involved. The most characteristic of these symptoms are those that depend on damage of the cranial nerve nuclei lying beneath the floor of the fourth ventricle. These nuclei are the ninth, tenth, eleventh, and twelfth. The ascending root of the fifth also arises in part from the medulla. Disease of the bulbar nuclei frequently gives rise to a characteristic group of symptoms—paralysis of the tongue, palate, pharynx, and often of the larynx—constituting glosso-labio-laryngeal or bulbar paralysis. The paralysis is commonly bilateral, because it depends usually on a degenerative process in the nuclei ; when it depends on acute disease (hemorrhage or softening) the paralysis is generally bilateral (acute bulbar paralysis), because the nuclei in question lie very near the median line, and are damaged on both sides. The orbicularis oris is generally involved in the bulbar palsy. Acute lesions (especially hemorrhage) of the medulla, unless exceedingly small, lead almost invariably to death, which may occur instantaneously or after the lapse of a few hours. The cause of death is interference with the functions of the cardiac and respiratory nuclei. Small hemorrhages (bilateral) into the vagi nuclei have been known to cause sudden death. Motor paralysis from acute bulbar disease is sometimes slight and limited in distribution, since a lesion sufficiently large to interrupt the entire motor path is usually quickly fatal. Motor symtoms (paralysis and spasm) may

be either bilateral or unilateral. When bilateral, either the upper or lower extremities may be chiefly involved in the paralysis and spasm. A small chronic lesion (tumor) may damage the pyramidal tract above the decussation and the hypoglossal nerve of the same side. The resulting variety of crossed hemiplegia (paralysis of the tongue and limbs on opposite sides) is distinctive of disease of the medulla. The tongue in such a case deviates to the side opposite the limbs palsied. This form of crossed paralysis is much less frequent than the crossed seventh and limb palsy that occurs from pontine disease. The face is never implicated in a lesion of the medulla, but the orbicularis oris may be involved, since it is innervated, not by the seventh nerve, but by the hypoglossal nucleus or cells connected with it.

Lesions of the medulla in some cases cause paralysis of motion, or paralysis of sensation, or both, according to the situation of the lesion in the motor or sensory tract, without giving rise to symptoms that are characteristic of or even suggest medullary disease. Glycosuria and polyuria are occasionally observed in the course of chronic lesions (tumor) involving the floor of the fourth ventricle, but the causation of these symptoms is too obscure and their occurrence too inconstant to make them of diagnostic value.

l. **Cerebellum.**—A destructive lesion of considerable size involving the *Middle Lobe* of the cerebellum gives rise to unsteadiness in standing and walking. In walking the patient may grope about with the feet widely separated, the hands extended, and the body inclined forward. The tendency to oscillation may make progress so irregular that the gait resembles that of a drunken man. This titubation is sufficiently characteristic and frequent to make it suggestive of cerebellar disease. Occasionally

the gait of tabes resembles cerebellar titubation, but in disease of the middle lobe there is not the ataxia that characterizes locomotor ataxia. There is also usually no inco-ordination in the upper extremities, closure of the eyes may not increase the unsteadiness, and there may be no ataxia whatever in the upper or lower extremities when the patient lies supine. Occasionally disease of the middle lobe causes a tendency to fall backwards when the patient attempts to stand.

In irritative disease of the middle lobe of the cerebellum (tumor, hemorrhage, softening), giddiness and vertigo are of frequent occurrence. The giddiness may or may not be associated with titubation or staggering. Vomiting is also a frequent symptom of active cerebellar disease, but both vomiting and giddinesss are often caused by disease in other parts of the brain. Hence, although these symptoms are distinctly more constant in cerebellar disease than in disease elsewhere, they possess only a slight degree of localizing value. Optic neuritis is perhaps somewhat more constant in cerebellar disease than in similar disease elsewhere, and is often an early symptom.

Disease of the lateral lobes or hemispheres of the cerebellum probably gives rise to no symptoms of localizing significance, unless it involves the middle peduncles or exerts pressure on the middle lobe of the cerebellum. In the latter case the symptoms are those referable to disease of the middle lobe.

Irritative lesions of the cerebellum (especially tumor) sometimes inhibit temporarily the spinal reflex centres on which the knee-jerk depends, so that the knee-jerk may be lost or very much diminished. In some instances the knee-jerk is lost for days or weeks and then reappears, to be lost again after a variable period of time. The knee-jerks may be unequal in activity on the two sides.

Sometimes the knee-jerks are diminished for a long period of time. It is not known whether this unique symptom is related to disease of any particular region of the cerebellum. In cases of chronic organic intracranial disease, in which indications of the position of the lesion are absent, the occurrence of this symptom suggests a cerebellar process.

Disease (tumor) of the middle or lateral lobes of the cerebellum may exert pressure on surrounding parts, particularly on the pons. The symptoms referable to such pressure are mainly the symptoms of pressure on one or both pyramidal tracts—slight or considerable weakness of the extremities, increased knee-jerk, etc. Usually the symptoms are most marked on the side opposite the cerebellar lesion; sometimes they are confined to the same side. Sometimes there is also evidence of implication in the sensory paths in the pons, slight anæsthesia, paræsthesia, etc. There is seldom sufficient pressure to cause more than slight and transient sensory disturbance. Sometimes the cranial nerves arising from the pons suffer from the pressure. The sixth nerve is particularly apt, on account of its passage under the prominence of the pons, to be involved. Convulsion is of rare occurrence in cerebellar disease. Rigidity of the limbs and muscles of the back of the neck may occur, as a temporary or as a constant phenomenon. It is probably dependent on pressure exerted upon the pons.

It is important to remember that even extensive lesions of the cerebellar hemisphere may produce no symptoms that suggest intracranial disease, but this is probably the case only in very chronic processes which cause little irritation (for example, osteoma and some cases of massive tubercle).

Active disease of the *Middle Peduncle of the Cerebellum*

(processus ad pontem) almost invariably gives rise to distinctive symptoms. The most important of these is vertigo, which is often intense, and a tendency to rotation of the body on its long axis (forced or compulsory movements), to or from the sides of the lesion, or simply a tendency to lie on one side, or rotate to one side in walking.

A lesion seldom involves exclusively the middle cerebellar peduncle ; it usually encroaches on the side of the pons or on the corresponding hemisphere of the cerebellum. In the former case there may be paralysis of the fifth nerve, and, if the motor tract is compressed, unilateral weakness of the limbs. Stationary disease of the middle peduncle, and lesions which simply exert pressure on it, may cause no characteristic symptoms.

Disease at the **Base of the Brain** (tumor, meningitis) often produces symptoms which are highly characteristic. This depends chiefly on the circumstance that in most instances basal disease damages two or more of the cranial nerves between their superficial origin from the brain and their exit from the skull, thus giving rise to symptom-groups which possess localizing value. It is convenient to consider separately the symptoms of basal disease in the anterior, middle, and posterior fossæ of the skull.

Disease may be limited to the *Anterior Fossa* of the skull, in which case it may produce symptoms referable to damage of the olfactory nerves, or, if the process compresses or invades the frontal lobes, there may be mental symptoms. Frequently morbid processes originating in the anterior fossa extend backwards and involve the optic nerve or chiasm. Implication of the optic nerve is usually unilateral, and causes unilateral amblyopia or amaurosis. Damage to the chiasma may affect

chiefly the decussating fibres, with resulting temporal hemianopsia.

Disease in the *Middle Fossa* causes symptoms referable chiefly to the fifth nerve. The Gasserian ganglion is usually involved, and all three divisions of the nerve are apt to be damaged, though they may suffer singly or unequally. The symptoms may be those of irritation or of paralysis (pain or anæsthesia) of the nerve. When the Gasserian ganglion is damaged there is neuro-paralytic ophthalmia, and the association of this symptom with symptoms of irritation and paralysis of the fifth is highly distinctive of disease of the middle fossa. The symptoms are, of course, unilateral. Sometimes the crus is involved by extension; there may then be hemiplegic weakness. Occasionally the third, fourth, and sixth nerves of one side suffer damage from disease near the sphenoidal fissure.

Disease in the *Posterior Fossa* of the skull causes combinations of symptoms that resemble those produced by disease within the pons and medulla, with similar variations according to the position at which the process is effective. There are two facts, however, which generally serve to distinguish disease originating in the posterior fossa from that which involves the pons and medulla primarily. First, the combinations of the cranial nerves that can be damaged by disease are different in the two cases; and secondly, when there is disease in the fossa the cranial nerves are prone to suffer before there is evidence of damage to the motor path in the pons and medulla.

In basal disease the fifth and sixth nerves readily suffer together, since the sixth in its course passes close to the former. This combination is exceedingly rare in pontine disease. The seventh and eighth readily become

involved together in disease at the base, while the same combination is possible only in very extensive disease of the pons. Similarly, but more rarely, the sixth, seventh and eighth are damaged together. The eleventh and twelfth or the ninth, eleventh, and twelfth may be injured together. In the latter case there is unilateral paralysis of the tongue, palate, and vocal cord—a combination distinctive of disease at the side of the medulla. Disease of the same parts from a central lesion is generally bilateral, and is associated with palsy of the lips. Occasionally, many cranial nerves are damaged by a process (chronic meningitis) in the posterior fossa. Thus, the fifth, sixth, seventh, eighth, ninth, tenth, eleventh, and twelfth have been known to be involved on one side. The number of nerves involved in a basal lesion, and the order and degree of their implication, are subject to great variations. The cranial nerve paralyses from disease of the posterior fossa are rarely strictly unilateral throughout the disease. When the fifth is involved there may be ophthalmitis, but this is less frequent than in disease of the middle fossa. The motor path is often involved unilaterally, with the usual results. Occasionally the middle cerebellar peduncle suffers from pressure. Convulsions may result from pressure on the pons, but these possess no distinctive character from those which result from disease in its substance.

Having passed in review the chief symptoms of brain disease in their relation to the position of the lesion, it becomes necessary to mention certain general facts of diagnosis which are of importance in forming correct conclusions in localization. In any case of brain disease in which the symptoms are of acute onset it is necessary to distinguish the indirect from the direct effects of the lesion. This can be done surely only by waiting until

the indirect symptoms have disappeared or diminished greatly, and this usually occurs in the course of ten days or two weeks. In this way the symptoms that depend on actual destruction of tissue may be certainly distinguished from those that result from irritation, and the distinction is important, not only for accurate localization, but for prognosis. Of course, a prediction can be made soon after the development of the symptoms, as to which symptoms are direct, but at best the truth can only be approximated. In the case of chronic lesions (tumor) which both irritate and destroy, the distinction is often difficult and may make a nice localization impossible. A second point of importance relates to the order of appearance of the different symptoms. It is especially desirable to note this in the case of acute lesions, *since only such symptoms as make their appearance together are to be referred to the same focus of disease.* When all the symptoms are of gradual onset it may be difficult to determine whether they are to be referred to the same or to different lesions. Multiple lesions of the brain are usually difficult to localize accurately, since they are apt to produce combinations of symptoms that are of doubtful significance. If, however, there are two lesions in opposite halves of the brain, it is frequently an easy matter to localize them, especially if they are not simultaneous in their development. In any case it is exceedingly important to determine accurately the mode (whether sudden or gradual) and order of development of the symptoms, before formulating a conclusion as to the locality of the lesion.

Spinal Cord.—The localization of lesions of the spinal cord is in many respects less difficult than the localization of lesions of the brain. This depends chiefly on the fact that even a small focus of disease in the sub-

stance of the cord almost invariably interrupts structures that have important motor or sensory functions, the disturbance of which indicates approximately or accurately the position of the morbid process.

Most diseases of the spinal cord involve a part only of its substance. Some lesions damage a considerable extent of certain structures, white or gray, without invading adjacent structures. The term "system-diseases" has been applied to such lesions. Other lesions are of small vertical extent, and may be confined to particular structures, as the ganglion-cells of the anterior horns or the pyramidal tracts, or may damage all the structures of the cord at a particular level. Lesions which are limited both in vertical and horizontal extent are called "focal" lesions. The problem of localization includes the determination of both the vertical and horizontal extent of the lesion. The indications of the horizontal extent of the lesion depend on the fact that different areas of the white and gray substance at the same level have widely different functions.

1. Lesions involving the *Gray Substance* of the spinal cord produce different symptoms according as the morbid process involves (*a*) the anterior horns, (*b*) the posterior horns, or (*c*) both anterior and posterior horns.

Disease of the *Anterior Horns* gives rise to three distinct results. There is, first, motor paralysis. This depends on the fact that destruction of the ganglion-cells of the anterior horns constitutes an interruption of the motor path from the cerebral cortex to the muscles. In addition to motor paralysis there is degenerative atrophy of the paralyzed muscles with R D. The combination of paralysis with degenerative atrophy always localizes the lesion to some part of the lower segment of the motor path. The third result of disease of the anterior horn

is loss or impairment of reflexes in which the muscles paralyzed are concerned. The clinical type of paralysis resulting from disease of the anterior horns may be termed *atrophic spinal paralysis.*

The distribution of these symptoms depends on the level of the spinal cord at which the ganglion-cells are involved, and on the extent of the damage in a vertical direction. The special symptoms of anterior cornual disease at various levels will be considered later.

If the lesion of the anterior horn is of acute onset (for example, poliomyelitis) the loss of voluntary power is also of rapid development. Degenerative atrophy follows, and the atrophy and altered electrical reactions become apparent in the course of one or two weeks. When the lesion is of gradual development (progressive muscular atrophy) the paralysis and atrophy develop *pari passu.*

Disease limited strictly to the anterior horn does not give rise to sensory symptoms, and the absence of such symptoms seems to distinguish disease of the anterior horns from disease of other parts of the lower segment of the motor path. Paralysis, wasting, and loss of reflex action are symptoms common to disease *in any part of the lower segment of the motor path,* but disease of the peripheral part of the path is usually (not always) associated with sensory symptoms—numbness, tingling, anæsthesia, etc.—because the motor fibres are mixed with sensory fibres, and the former seldom suffer alone.

When an acute lesion of the anterior horns is accompanied with sensory symptoms, these are due to damage, often slight, to adjacent sensory structures. At the onset of an acute lesion of the anterior cornua spasm may be a prominent symptom.

Disease limited to the structures of the *Posterior Horn*

is exceedingly rare, and there is some uncertainty as to the exact character of the symptoms that result from such disease. Irritative disease probably gives rise to pain, and perhaps to hyperalgesia, in a corresponding skin area. Destructive disease probably causes loss of certain forms of sensibility, especially the sensibility to pain and temperature. It is very doubtful whether tactile sensibility or the muscular sense are ever disturbed.

Chronic lesions (chiefly gliomatous infiltration and cavity formation) involving both *Anterior and Posterior* horns of gray matter are occasionally observed. They are characterized by the development of muscular atrophy with R D, gradual loss of voluntary power, and disturbances of sensibility, not for touch, but for pain and temperature. Since these sensory disturbances are not obtrusive they may be overlooked unless they are especially sought for, and the symptoms (atrophy and wasting, etc.) may be referred to the anterior horns (progressive muscular atrophy). The chief condition that gives rise to this characteristic combination of motor and sensory symptoms mentioned is syringomyelia (p. 488).

2. *White Matter.*—Disease of the *Anterior and Lateral White Columns* of the cord gives rise to a characteristic group of symptoms. There is, first, loss of voluntary power below the level of the lesion. This loss of power is not associated with degenerative atrophy of the muscles. Secondly, there is excessive action of the spinal centres (ganglion-cells of anterior gray cornua) below the lesion. This is manifested by the presence of two symptoms, (*a*) excessive reflex action, shown especially by increase in the knee-jerk, Babinski's sign, and often by the presence of an ankle clonus, and (*b*) spasm or rigidity of the muscles below the seat of the disease.

The former symptom may be present without the latter where the excess is not great, but in many cases both expressions of the overaction of the spinal centres are present. The clinical type of paralysis that results from disease of the lateral columns is called *spastic spinal paralysis*. When only the anterior and lateral columns are involved in disease there is, as stated above, no degenerative atrophy. If, however, the ganglion-cells of the anterior horns suffer also, as they may, degenerative atrophy occurs in the muscles connected with them, and with this there is diminution or loss of reflex action, notwithstanding the fact that reflex action may previously have been increased.

The symptoms described as evidence of disease of the anterior and lateral columns depend on the involvement of the pyramidal tracts. We cannot yet say precisely what symptoms are produced by lesions involving the other important conducting tracts of these columns, (direct cerebellar tract, antero-lateral tract).

It is evident from what has gone before that two distinct clinical types of paralysis (atrophic spinal paralysis, spastic spinal paralysis) may result from spinal-cord lesions, according as the morbid process involves the anterior horns or the pyramidal tracts of the anterior and lateral columns. The clinical features of these forms, which are sometimes blended, are elsewhere enumerated (see Chap. V.).

Disease confined to the *Postero-External Column* of the posterior column of the cord gives rise to distinctive symptoms. These are (1) inco-ordination in the extremities below the level of the lesion ; (2) loss of tactile sensibility, varying in distribution (usually chiefly in the legs) according to the vertical extent and position of the lesion ; and (3) diminution or loss of reflex action (loss of knee-jerk if the lesion is in the lumbar cord). These symptoms

depend on the fact that the postero-external columns contain many of the fibres of the posterior nerve-roots which are necessarily interrupted by a morbid process in the former situation. The posterior nerve-roots are often diseased by degenerative processes that involve the postero-external columns, and give rise to similar and sometimes identical symptoms—with the exception that pain is often a prominent feature in nerve-root disease (the path for painful stimuli being contained in these roots), while it is open to question whether disease limited to the postero-external columns causes pain.

Disease limited to the *Postero-Internal Columns* of the cord (columns of Goll) is exceedingly rare, but disease of these columns secondary to disease (usually degenerative) of the postero-external columns is not of infrequent occurrence. There is little clinico-pathological evidence to show what symptoms result from disease in this position, but it is on the whole probable that it gives rise to disturbance of co-ordination or of equilibrium. Ordinarily any disturbance of this nature must be masked by the inco-ordination that results from the disease of the postero-external columns, to which lesions of the posterior median columns are secondary.

A Unilateral Lesion of the spinal cord gives rise to a highly characteristic group of symptoms. First, there is motor paralysis on the side of the lesion and below its level. This depends on the interruption of the fibres of the pyramidal tracts. Often there is slight weakness on the opposite side, usually of the leg. Secondly, there is loss of sensibility on the side *opposite* the lesion. The loss of sensibility does not extend quite as high as the level of the lesion. This is because the sensory path does not decussate immediately at the level of the entrance of the sensory nerves, but a short distance above this. There is

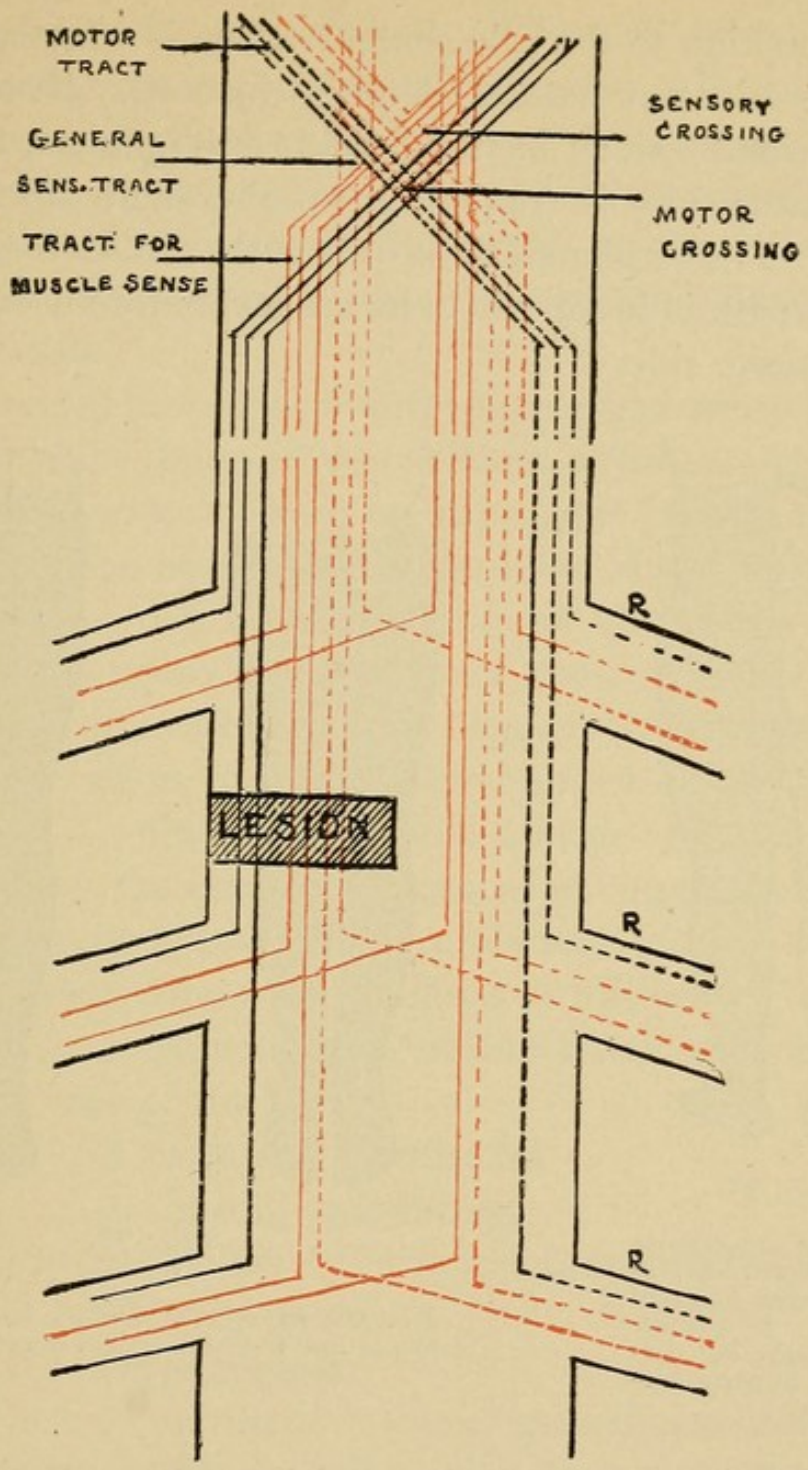


FIG. 71.

Diagram showing the arrangement of the sensory and motor tracts in the cord and medulla and the effects of a unilateral lesion. R, R, R, nerve-roots. The sensory paths are represented in red, the motor paths in black. It is to be noted that the tracts conveying general sensibility cross to the opposite side of the cord soon after entering it, while the tracts conveying muscular sensibility cross in the medulla just above the motor decussation. The nerve-roots are to be considered in the *dorsal* cord, the cervical cord being left out. A unilateral lesion in the dorsal region of the cord would produce the following effects. Paralysis of motion on the same side (leg), loss of muscular sense in the same side in the parts below, loss of sensation on the opposite side (leg) below the lesion.

one exception to the rule that sensation is affected on the side opposite a unilateral lesion of the cord. This relates to one-sided lesions of the lumbar enlargement, in which the sensory loss is frequently on the same side as the lesion. The explanation of this is that in such cases the sensory path is involved (in its ascent) before it crosses to the opposite side.

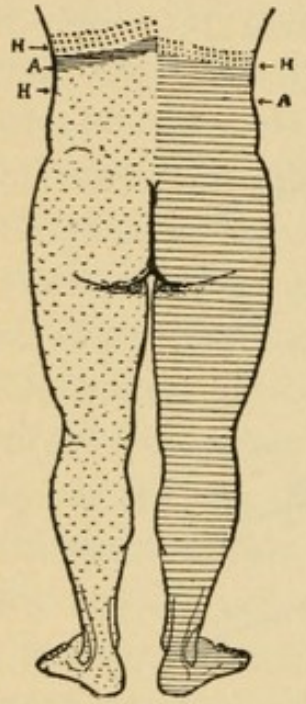


FIG. 72.

Areas of anæsthesia and hyperæsthesia in case of Brown-Séquard paralysis. Lines indicate anæsthesia ; dots indicate hyperæsthesia. (Starr.)

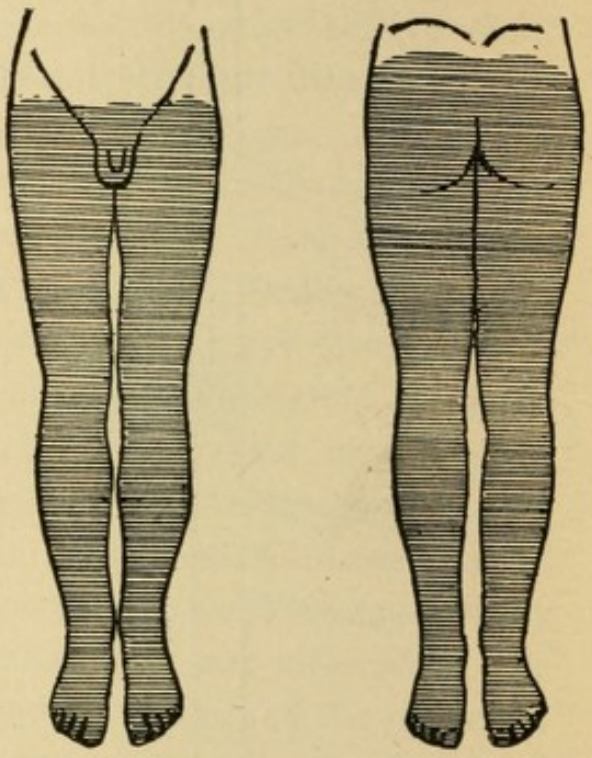


FIG. 73.

Diagram of area of sensory loss in case of crush of first lumbar segment of cord.

In unilateral cord lesions the muscular sense may be impaired or lost in the muscles below the level of the lesion, but it is impaired on the *same side* as the lesion, since, as already stated (p. 134), the path for the muscular sense does not cross in the cord. Of the other forms of sensibility those for pain and temperature are almost constantly impaired, and in the majority of cases tactile

sensibility is also affected. There is often an area of hyperalgesia on the side of the lesion which corresponds in position with the anæsthesia of the opposite side. The reflexes, superficial and deep, are increased below the lesion on the same side, except at the onset of acute lesions, when there is sometimes loss of knee-jerk on the same side.

Besides these motor and sensory phenomena below the level of the lesion there are others at the same level and on the same side. There are (*a*) a zone of hyperalgesia due to irritation of sensory nerve-roots at the upper level of the lesion; (*b*) a narrow zone of anæsthesia just below (*a*), from injury to the sensory roots before they cross; and (*c*) localized unilateral degenerative muscular atrophy in cases where a considerable number of motor cells are damaged. The effects of this damage are best observed in lesions of the cervical or lumbar enlargement.

The typical results of a unilateral lesion [1] of the cord may conveniently be tabulated as follows:

A. At or near the level of the lesion and on the same side of the body—
   - A band of hyperalgesia.
   - A band of anæsthesia.

B. Below the level of the lesion and on the *same* side—
   - Motor paralysis.
   - Hyperalgesia.
   - Impairment or loss of muscular sense (sometimes with ataxia).
   - Early loss of reflexes, including knee-jerk (in acute cases).
   - Subsequent increase of knee-jerk and frequently ankle-clonus.

[1] This characteristic group of motor and sensory symptoms is sometimes denominated Brown-Séquard's paralysis.

Temperature raised. Often the side below the lesion is, for the first few days or weeks, warmer than the other, the difference being usually about a degree Fahrenheit. This difference usually disappears in time, to be replaced by the reverse, the side below the lesion being colder.

C. Below the level of the lesion and on the *opposite* side—

Loss of sensation (pain, temperature, touch).
Temperature same as that above lesion.

A Complete Transverse Lesion of the cord gives rise to loss of motor power and loss of all varieties of sensibility below the level of the lesion. The motor and sensory loss is as complete (so far as voluntary motion and sensation are concerned) as though the entire cord below the lesion were destroyed, since motor and sensory conduction are abolished. In other words, the functions of the cord as a conductor to and from the brain are destroyed as regards parts below the level of the disease. It is otherwise with the functions of the cord as a centre—that is, as a trophic and reflex centre. The ganglion-cells of the anterior horns, being intact below the lower level of the morbid process, still continue to subserve their trophic and reflex functions. Hence there is *no degenerative atrophy* in the muscles supplied by nerves derived from the part of the cord *below* the lesion. The ganglion-cells that subserve the various reflexes, being no longer under cerebral control, overact, and this overaction is shown by increased knee-jerks and often by the existence of ankle-clonus. It must be remembered, however, that the knee-jerks may be abolished for days or weeks in consequence of inhibition from the irritation of the lesion. In such cases overaction eventually sets in, if

the patient survives the damage to the cord sufficiently long.

In addition to these phenomena dependent on a complete transverse lesion of the cord, there may be after a time degenerative atrophy in certain groups of muscles *corresponding to the level of the lesion.* This atrophy depends on the destruction of ganglion-cells of the anterior cornua at the level of the lesion; hence when the lesion is of slight vertical extent the atrophy may be imperceptible, but when the lesion is of considerable vertical extent the atrophy has a correspondingly wide distribution. The atrophy is of course chiefly noticeable when the disease is in the cervical and lumbar enlargements. Just above the level of the anæsthesia there is usually a girdle pain or a band of hyperalgesia. This depends on sensory irritation in the lower end of the upper portion of the normal cord.

We may tabulate the results of a complete transverse lesion as follows :

A. At the level of the lesion—
   - A zone of hyperæsthesia.
   - Muscular atrophy of limited distribution.

B. Below the level of the lesion—
   - Complete motor paralysis.
   - Complete sensory paralysis (all forms).
   - Initial loss of knee-jerk (in acute lesions).
   - Eventual excess of knee-jerk and ankle clonus.

The distribution of these symptoms *vertically* depends on the level of the cord at which the lesion is situated, and this is true also of unilateral lesions of the cord. In every case it is necessary to determine as accurately as possible the upper and lower limits of the lesion. The upper limit of the lesion is indicated by the upper limit of the anæsthesia and motor paralysis. More than

this cannot be learned from the distribution of the motor and sensory paralysis, since this is the same whatever may be the downward extent of the lesion. In order to determine the lower level of the lesion it is necessary to study the condition of the reflexes and the trophic state of the muscles—that is, to interrogate the cord as a central organ. If reflex action is retained, this shows that the reflex or loops on which the reflexes depend are not included in the lesion. On the other hand, loss of reflex action indicates that the lesion has destroyed the integrity of the reflex loops, and presumably that of the ganglion-cells, or nerve-roots, or both, on which the reflex depends. Both the deep and superficial reflexes should be studied with care. The table on page 23 shows the regions of the cord with which the different reflexes are in relation, and with this knowledge it is easy to infer from the state of the reflexes whether particular regions of the cord have retained or lost their integrity.

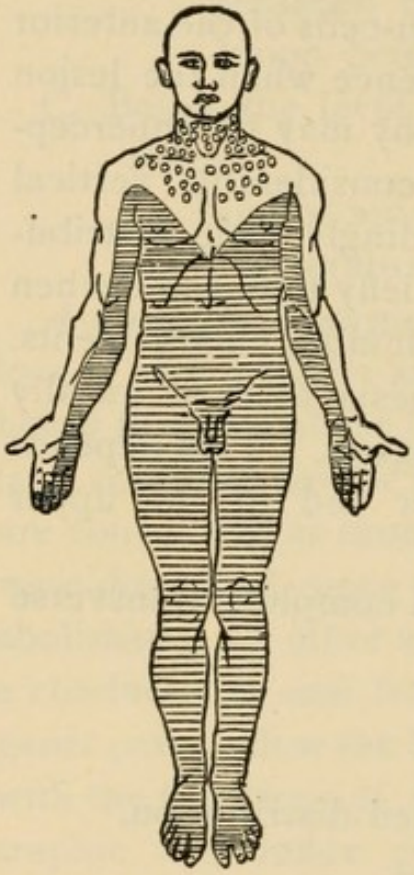


FIG. 74.

Diagram showing area of sensory loss in case of crush of VIIth cervical segment of cord. The distribution of analgesia and anæsthesia in the arms is characteristic of a lesion at this level. Area of hyperalgesia dotted.

The extent downwards of the lesion is indicated also by the state of muscular nutrition and the electrical reactions. The presence of atrophy and of R D shows that the anterior cornua or the corresponding motor roots have been damaged, and the extent of these changes indicates the precise levels thus affected. In order to localize accurately the lower level of the lesion by inference from the

trophic state, it is necessary to bear in mind the relations of the various muscles and muscle-groups to the cord. These relations are given in the table. The indications of the extent of the lesion afforded by the trophic state of the muscles are especially valuable when the disease is in the lumbar and cervical enlargements. When the destructive process is in the dorsal region the effect on muscular nutrition is less noticeable and less valuable as an aid in localizing the lesion, but it is just in this region that the indications obtained from the state of the reflexes are of the greatest aid.

With the help of these facts, and the tables showing the representations of the motor, sensory, and reflex functions of the cord, it is possible, in most cases, to make an accurate diagnosis of the position and extent of the lesion. It may be well, however, to outline briefly the distribution of the motor and sensory loss that results from a complete transverse lesion of the cord at the most important levels of the cord. Individual differences exist in the distribution of symptoms from disease at the same levels, but these differences are seldom considerable, and do not seriously invalidate the following types.

A lesion which destroys only the extremity of the cord (conus medullaris) as high as the fifth sacral nerves, causes paralysis of the bladder and rectum, and a small area of anæsthesia near the tip of the coccyx. Such lesions are very rare. If the lesion destroys the cord at the level of the fourth and fifth sacral segments there is, besides paralysis of bladder and rectum, loss of power in the perineal muscles (erector penis, transversus perinei, accelerator urinæ), with loss of ejaculatory and erectile power. There is also a characteristic anæsthetic area, which probably depends on injury of the fourth sacral segments or roots. There is a saddle-shaped region of

anæsthesia on the back of the thighs and sacrum. The internal surfaces of the thighs high up are also anæsthetic, and the anæsthesia usually extends to the external genitals and perineum. Figure 75 represents the saddle-shaped area in such a case.

In lesions involving the upper sacral segments there is paralysis of the muscles of the leg and foot, and a strip of anæsthesia on the back of the thigh, which extends to the back of the leg and ankle and to the sole and dorsum of the foot. The skin of the inner side of the leg usually escapes.

Damage to the cord as high as the lumbar region causes paralysis of the flexors of the knee and abductors of the thigh. There is also anæsthesia of the anterior and inner aspect of the leg and of the inner and posterior part of the thigh.

When the middle of the lumbar region is destroyed, the motor paralysis includes the extensors of the knee and the abductors of the thigh, and the loss of sensation extends as high as the anterior aspect of the thigh (lower part at least). When the disease affects the upper part of the lumbar enlargement the loss of sensibility includes the upper and outer aspect of the thigh and the skin of the groin and part of the scrotum. The flexors of the thigh and the sartorius are then paralyzed.

As the level of the lesion ascends in the dorsal region the level of the anæsthesia and motor paralysis extends to a correspondingly higher level on the trunk. The abdominal muscles are first paralyzed, then the intercostals. The umbilicus corresponds to the tenth dorsal nerves. When the anæsthesia reaches one inch above the umbilicus the lesion is as high as the ninth dorsal nerves. The epigastrium corresponds to the sixth and seventh dorsal segments, the nipples to the fourth dorsal, and the third rib to the third dorsal segment.

When a lesion is placed as high as the first dorsal segment (that is, the lower part of the cervical enlargement) the first impairment of sensibility and loss of power in the upper extremity are observed. There is loss of power in the intrinsic muscles of the hand and loss of sensibility in part or all of the area of the hand supplied by the ulnar nerve. When the lesion invades the cord at the level of the seventh cervical segment the anæsthesia usually reaches as high as the median line, anteriorly and posteriorly, of the arm, forearm, and hand. Figure 50 shows this characteristic distribution of anæsthesia in a case of total transverse lesion at this level of the cord. The motor loss in such cases is sufficiently indicated by the table.

A lesion destroying the middle of the cervical enlargement (fifth, sixth, seventh) causes loss of sensation in the entire arm and part of the shoulder. The motor paralysis extends to the shoulder muscles and the serratus magnus. There is also sometimes some loss of power in the trapezius and sterno-mastoid when the lesion is placed as high as the fifth cervical segment. When the fourth cervical segment is destroyed the anæsthesia extends to the entire shoulder and lower part of the neck. The action of the diaphragm is impaired and the motor paralysis extends to the few remaining muscles of the shoulder (supra- and infra-spinati). A complete transverse lesion at this level is usually quickly fatal. The accessory muscles of respiration (scaleni, sterno-mastoids) may give the patient enough air to maintain life a few days, but not longer. In the lower part of the cervical cord an acute lesion may inhibit respiration so as to cause death in the course of a few days.

Nerve-Roots.—Lesions of the *Anterior Nerve-Roots* give rise to motor over-action or motor paralysis according as they are irritative or destructive in character. The

distribution of the spasm and paralysis depends on the number and locality of the roots involved. The table (on page 23) shows the representation of the different muscles in the anterior roots, this being practically the same as in the corresponding segments of the cord. In chronic irritative disease long-continued and persistent spasm (from tumor) may produce loss of voluntary power, and in acute irritative lesions (meningitis) irritation symptoms far exceed paralytic phenomena and may even exist alone.

Irritative lesions of the *Posterior Nerve-Roots* cause pain and hyperalgesic skin areas corresponding to the irritated roots. The pain is often severe and persistent. Destructive lesions cause loss of sensibility. When the destruction is partial, tactile sensibility may be chiefly impaired; when it is complete, all forms of sensibility are abolished in the corresponding skin area.

Cauda Equina.—The cauda equina not very infrequently suffers from the effects of pressure. This pressure is exerted most often in consequence of injury to the lumbar spine, especially dislocation. Less frequently the pressure results from tumor or from spina bifida. The resulting symptoms vary considerably according to the position and extent of the lesion. In a lesion which involves the entire cauda equina, or the larger number of its constituent roots, the nerve-roots which take their origin from the lowest part of the cord (that is, lie nearest the median line) are more seriously injured than those above them. If all the roots are originally involved the upper ones may soon recover entirely, while the lower ones continue to suffer seriously. This is a fact of some diagnostic importance. While there is nothing absolutely distinctive about the distribution of the motor and sensory symptoms in cases of injury to the cauda equina, there is considerable uniformity in the

phenomena, and many of the cases conform more or less closely to a certain type. The motor paralysis is especially apt to affect the branches of the sciatic and pudic nerves, and particularly the lower branches of the former. The anterior crural and obturator distribution are also frequently involved, but the paralysis is apt to be less in degree. The R D may or may not be present, according to the severity of the damage. The anæsthesia commonly involves especially the distribution of the great and small sciatic and pudic nerves. When the fourth and fifth sacral roots suffer chiefly, as they sometimes do, the anæsthesia is limited to the perineum, external genitals, and a saddle-shaped area on the buttocks and thighs. Sometimes the anæsthesia is limited to the perineum and genitals and upper part of the inner surfaces of the thighs, even though there is considerable loss of power in the lower extremities.

The condition of the reflexes is different in different cases. The knee-jerk is usually absent, as is also the plantar reflex. Retention of urine, with overflow incontinence, is not common. Such incontinence may exist for several months in consequence of a cauda injury, and yet terminate in recovery of function when the pressure ceases or diminishes. As a rule, the sphincters are only slightly or not at all affected in pure cauda lesions. Trophic disturbances (bed-sores, perforating ulcer) occur in a considerable proportion of cases. The distinction of cauda equina disease from lesions of the lower part of the spinal cord may be exceedingly difficult. If the damage is of traumatic origin the position of the local symptoms (fracture, displacement of vertebræ, etc.) is of much assistance, since the cord itself does not extend below the lower border of the first lumbar vertebra, and any displacement below this level would injure the cauda and not the cord. In the absence of distinct local indica-

tions, the distribution of the motor and sensory phenomena and their mode of development may be of aid. Anæsthesia limited to the buttocks (saddle-shaped areas) or to the buttock and backs of the thighs, or to the buttocks and thighs and the legs (posterior aspect), suggests a dependence on a cauda lesion and not on a cord lesion, but it is not certain that anæsthesia of similar distribution may not result from partial destruction of the conus

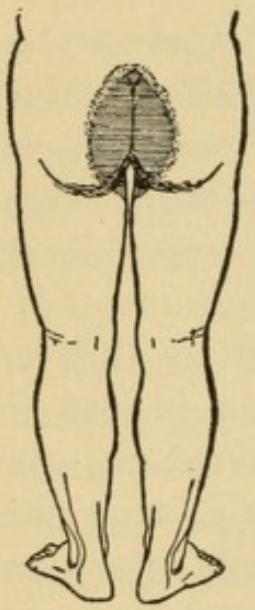


FIG. 75.

Area of anæsthesia in a lesion of the cauda equina, dependent probably on damage to the fourth and fifth sacral roots. (Starr.)

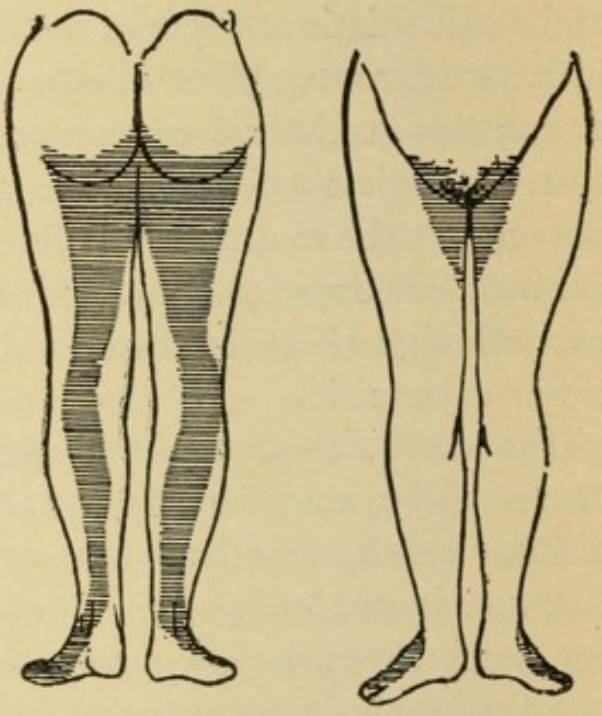


FIG. 76.

Diagram showing the area of anæsthesia in a woman with a lesion of the cauda equina.

medullaris and lumbar cord in cases where there is little or no involvement of the nerve-roots. Considerable sensory loss (as that shown in diagram 75), with slight loss of motor power and slight involvement of the sphincters, also suggests a cauda and not a cord lesion. Pain *in* the area of anæsthesia points to a cauda-equina lesion, pain *above* the area of anæsthesia to a cord lesion. The pain of

cauda lesions is referred to the sacrum rather than the lumbar spine, which is generally the seat of pain in spinal-cord disease. With the aid of these points of distinction a diagnosis can generally be made between damage of the lumbar, or lumbar and sacral cord, and damage of the cauda equina. But when, as is probably not rarely the case, the conus medullaris is involved with the cauda equina, the recognition of this involvement may be impossible. As already stated, it is not certain that damage of the conus and lower end of the cord may not be competent to cause some of the peculiarly disposed anæsthesias which are thought to be characteristic of a cauda lesion.[1] It may be that the involvement of the conus simply intensifies the symptoms of cauda disease. Destruction of the conus medullaris causes complete paralysis of the bladder and rectal sphincters and loss of sexual power. It is not yet known whether such complete paralysis is ever the result of pure lesions of the cauda. There is some reason to think it is not. When, therefore, the symptoms suggest a cauda lesion, but the sphincters are completely paralyzed, there is reason to think that the conus medullaris is also implicated. Further investigation is needed to clear up these points. Asymmetry of the motor and sensory loss suggests a lesion of the cauda rather than of the cord, and extreme chronicity of development (years) has the same significance.

[1] Thus in one case of dislocation of the first lumbar vertebra there was complete paralysis of motion below the knee and anæsthesia of the buttock, back of thigh, leg, and sole of foot, but the autopsy failed to show any damage to the cauda equina. The autopsy did show, however, crushing of the sacral and lowest lumbar segments of the cord and slight damage of the nerve-roots as they lie on the cord at this level.

The distinction of disease or injury of the cauda equina from lesions of the peripheral nerves is usually not difficult. Multiple neuritis is the only disease of the peripheral nerves that can be mistaken for a cauda lesion. The diagnostic points are elsewhere presented (p. 538) Occasionally an injury of the nerves in their extra-spinal course, particularly one involving the lumbo-sacral plexus, gives rise to considerable diagnostic difficulty. The points to be borne in mind in making the distinction are the bilateral symmetry (not always perfect) of the symptoms from cauda injury, and the usual occurrence of local evidences of injury. In rare instances injuries of the cauda have produced chiefly unilateral motor and sensory symptoms, but even in these cases there are usually some symptoms that suggest an intra-spinal nerve lesion (disturbances of bladder and rectum, peculiar distribution of anæsthesia, etc.).

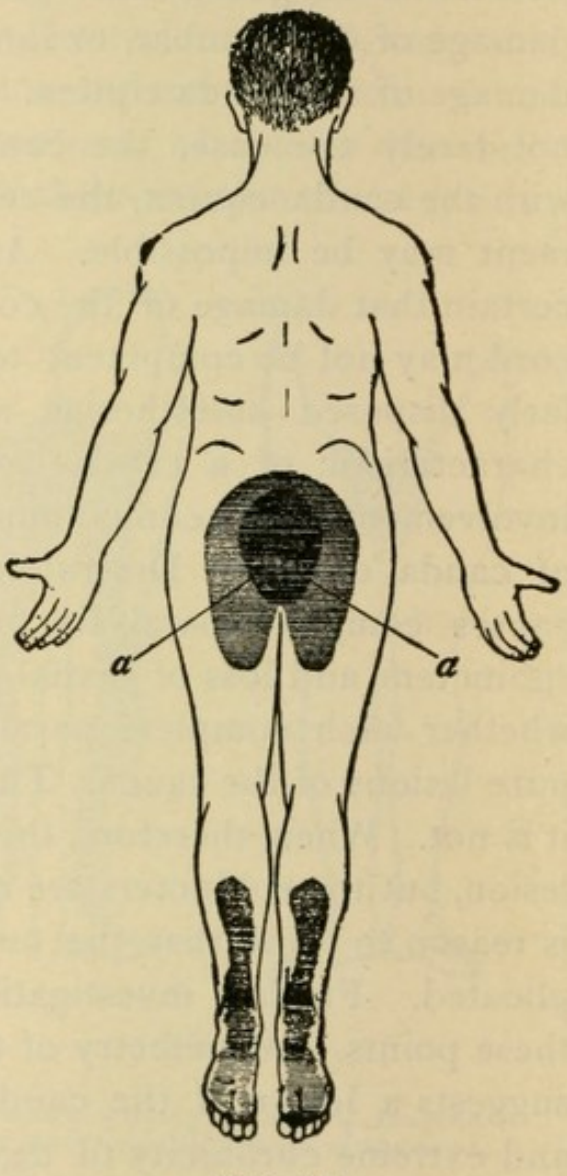


FIG. 77.

Diagram showing the area of sensory loss in a case of crush of the cauda equina and conus medullaris, *a*, *a*, area of anæsthesia and analgesia. The lightly shaded areas on the buttocks and legs represent a slighter degree of sensory loss.

Peripheral Nerves.—The localization of lesions involving the nerves in their extra-cranial and extra-spinal course is, generally speaking, less difficult than the localization of lesions of the brain and spinal cord. The data for an accurate local diagnosis are easier to obtain and are less equivocal in significance than in the case of central lesions; local signs play a more important rôle and the diagnosis is less distinctly inferential in character. An accurate knowledge of the course and distribution of the nerves and of the anomalies that occur, must be the foundation on which every diagnosis rests. The possibilities of collateral innervation must also be borne in mind in determining the effect of a lesion in a given situation.

The general symptomatic characters of peripheral-nerve lesions may conveniently be considered in the chapter on clinical types. The special symptomatic characters of such lesions vary with the special functions and distributions of the nerve or nerves implicated. Certain symptomatic combinations are sufficiently frequent and important to require special notice, and are mentioned as clinical types of disease.

CHAPTER IV.

THE DIAGNOSIS OF THE NATURE OF THE LESION.

HAVING established the situation of the lesion in a given case of organic disease of the nervous system, there remains for determination the nature of the morbid process which constitutes the lesion. Generally speaking, this problem presents greater difficulties than the local diagnosis. In many instances, it is true, an exact diagnosis of the nature of the lesion is readily reached, but the cases are quite as numerous in which an approximate diagnosis only can be made. Sometimes the best that can be done is to narrow the diagnosis to one of two possibilities, and occasionally it is impossible to do better than to make a guess.

Notwithstanding the obstacles to an exact diagnosis of the character of the pathological process, the problem is one of the highest practical importance, and demands for its solution the most painstaking care in balancing the available diagnostic indications. In the case of lesions of the central nervous system the pathological diagnosis is usually of distinctly greater importance to the patient than the exact local diagnosis, for on it depends, in great degree, both the prognosis and the treatment.

Before studying the more important indications that guide us in determining the nature of the lesion it is useful to consider briefly the various morbid processes which are encountered in practice and constitute the great

majority of the cases of nervous disease. These processes are few if we consider only the ultimate processes to which the symptoms of disease are due. They are hemorrhage, necrotic softening, inflammation, abscess, tumor, and degeneration. To this list may conveniently be added congestion and anæmia, although these are not, strictly speaking, organic conditions.

Hyperæmia.—The subject of *Congestion of the Brain* is one of considerable obscurity. It cannot be said that we possess any exact knowledge in regard to it.

The results of post-mortem examination are to a considerable extent negative—that is, there are no constant or characteristic changes in cases which, from their symptoms, seem referable to cerebral congestion. This fact, however, does not in itself make the possibility of the occurrence of congestion impossible, since general pathology teaches that distinct active congestion of other parts may leave no trace after death. Indeed cases occasionally occur in which the symptoms can reasonably be referred to cerebral congestion alone. The hyperæmia may be active or passsive. In the former case there is an excess of blood in the arteries, in the latter an excess in the veins. In both instances the capillaries are over-distended, and it is to this over-distension that the symptoms of congestion are due. In both instances the brain suffers from a deficiency of oxygen, in consequence of the stasis, but in passive congestion there is also an excessive accumulation of carbonic acid in the capillaries. It is probable that prolonged passive congestion during life may cause a permanent increase in the size of the capillaries. Mechanical venous congestion may result a short time after death from gravitation of blood to dependent portions of the brain. When ante-mortem passive congestion has existed in such cases, its effects cannot be distinguished in

the presence of the more obtrusive results of gravitation. It is important to note that even the most intense passive congestion, such as occurs in asphyxia, may leave no trace after death.

What has been said of our imperfect knowledge of cerebral congestion may be said with equal truth of congestion of the cord. It is probable that active primary congestion of the cord occurring independently of the initial stage of myelitis is an exceedingly rare condition. A so-called secondary congestion of the cord is not an infrequent consequence of excessive physiological activity of the nerve elements of the cord, especially of the motor elements. Passive congestion of the cord is of exceedingly rare occurrence. The influence of gravitation in producing mechanical congestion of the cord after death is probably as great as in the case of the brain. The same influence is operative in the cord during life, but to a far slighter degree.

Of congestion of the membranes of the brain and spinal cord, as conditions distinct from the first stage of inflammation, nothing is known. Likewise nothing is known of simple congestion of the peripheral nerves.

Anæmia.—*Anæmia of the Brain* may result from general anæmia, in which the quantity or quality of the entire amount of blood is defective. This is seen in cases of simple anæmia, hemorrhage, chlorosis, etc. The blood supplied to the brain may also be diminished in the absence of any systemic causes for such diminution, as from cardiac weakness. Localized cerebral anæmia may result from obstruction to the flow of blood through the cerebral vessels. Permanent obstruction of this kind often leads to another pathological process—that is, softening. The various causes of anæmia of the brain may be suddenly or gradually operative, and the symp-

toms vary accordingly. There is some reason for believing that angiospasm (arterial spasm) is capable of causing anæmia of greater or less areas of brain substance, sometimes with resulting temporary hemiplegic symptoms.

Anæmia of the Spinal Cord, as a persistent condition, dependent on arterial obstruction, is unknown, but it is certain that such mechanical obstruction to the blood-supply of the cord may result in local anæmic necrosis and subsequent inflammation. It is uncertain whether the symptoms that suggest transient anæmia of the cord depend primarily on arterial spasm, or are due to primary functional derangement of the nerve elements, causing arterio-spasm. In certain blood states, as simple anæmia, chlorosis, and anæmia from blood loss, the nutrition of the entire body suffers, and with this the nutrition and functional activity of the spinal cord. Here, as in the brain, the essential pathological factor is the defective quantity or quality of the blood. Concerning anæmia of the peripheral nerves nothing is known.

Hemorrhage.—*Hemorrhage within the Cranial Cavity* may invade the brain substance itself or the meninges. In either case the hemorrhage is due to rupture of a vessel. Meningeal hemorrhage is almost invariably[1] due to injury; hemorrhage into the substance of the brain (including pons, medulla, and cerebellum) generally occurs spontaneously—that is, as the result exclusively of internal pathological conditions. The hemorrhage is usually from an artery. But occasionally it is from a vein or from capillaries. Excepting from traumatic influences, an artery ruptures only when its walls are the seat of disease. The local factor in producing hemorrhage may

[1] In children extensive subarachnoid hemorrhage sometimes occurs in the course of acute infectious diseases.

be the sole appreciable cause of the rupture. Frequently, however, there is a local increase in blood pressure, and such an increase causes the diseased and weakened arterial wall to give way more readily than it would otherwise do.

When the walls of an artery become weakened by disease they gradually yield before the blood pressure. This gradual yielding leads to local thinning of the coats of the vessel, which eventually become so thin and weak that they may rupture. As the walls yield before the pressure of blood, a bulging occurs, and this bulging constitutes an aneurism. Aneurisms occur in two different situations—in the larger arteries at the base of the brain and over the convexity, and in the small arteries that lie imbedded within the brain substance. Aneurisms of the former class are of uncommon occurrence. They usually attain considerable size and are few in number or single. They may give rise to symptoms due to pressure, and this is especially liable to occur when they are situated on vessels at the base of the brain. These aneurisms (intracranial aneurism proper) may eventually rupture, but, until they do so, their symptoms, if symptoms exist, are those of intracranial tumor. The aneurisms within the substance of the brain, which form on the small arterial branches, are of small size ($\frac{1}{100}$ to $\frac{1}{20}$ inch) and are hence termed miliary aneurisms. These miliary aneurisms are always numerous. They are found most frequently on the vessels to the central ganglia, probably because these small vessels are given off directly from a vessel (middle cerebral) of much greater calibre, in consequence of which the arterial pressure is considerably higher in them than in vessels of the same size which branch more gradually. The fact that the internal capsule (motor part) is supplied by vessels that are often the

seat of miliary aneurisms is of great practical importance, since hemorrhage in this situation is a frequent cause of hemiplegia.

The bursting of miliary aneurisms is the predominant cause of spontaneous cerebral hemorrhage, but it is certainly not the sole cause. Inflammation and degeneration (chronic endarteritis, obliterating endarteritis, atheroma, hyaline degeneration) may weaken the coats of the arteries in which they occur to such a degree that these vessels rupture without the previous development of miliary aneurisms.

Miliary aneurisms seem to depend mainly on a primary degenerative process in the arterial wall, affecting chiefly the contractile and elastic elements. The muscular coat of the vessel disappears and the intima and adventitia come in contact. The most important known factors in the production of this degeneration are the blood pressure to which the arteries are subjected, and the degenerative tendency that exists in the second half of life and that is associated with atheroma. Cerebral hemorrhage may occur without pre-existing changes, as atheroma, in the vessel walls visible to the naked eye, but it is an important fact that atheroma of the larger vessels is present in a large proportion of cases of cerebral hemorrhage, even when the hemorrhage is due to bursting of miliary aneurisms. Certain blood states, notably purpura, scurvy, pernicious anæmia, and leucocythemia, may lead to cerebral hemorrhage, or rather to cerebral hemorrhages, since the extravasations are generally multiple. In many of these cases it is impossible to demonstrate pathological changes in the vessels.

Cerebral hemorrhage may occur in any region of the brain, but is especially frequent in and about the basal ganglia and in the internal capsule. Hemorrhage into the cortex is of rare occurrence.

Hemorrhage gives rise to symptoms by destroying nerve elements, and by compressing neighboring structures and irritating them. This irritation of the adjacent tissue elements is chiefly operative during and immediately after the onset, and, in conjunction with pressure, is responsible for many indirect symptoms.

Hemorrhage into the Spinal Cord, from any cause whatever, is an exceedingly rare occurrence, and its rarity contrasts strikingly with the frequency of cerebral hemorrhage. Primary hemorrhage into the cord is so uncommon that its occurrence has been doubted, and it is certainly true that the conditions which, in the brain, further its production seem to be absent in the cord. The vessels to the cord are long and tortuous and the pressure within them is never high. Miliary aneurisms probably do not occur. Secondary hemorrhage into the cord, during inflammation of the cord, occasionally occurs and may readily be mistaken for primary hemorrhage. Secondary hemorrhage may also occur from certain highly vascular tumors of the cord, especially certain gliomata. Traumatic hemorrhage is more common than secondary hemorrhage and is especially apt to follow falls which severely concuss the spine. Not infrequently there is hemorrhage into the central canal of the cord. Minute hemorrhages into the substance of the cord often occur in the course of asphyxial and convulsive diseases (tetanus, hydrophobia, etc.), but they cause no symptoms. In all forms of hemorrhage into the spinal cord the gray matter is particularly apt to be involved.

Hemorrhage into the Membranes of the Cord is rare. The hemorrhage may be extra-meningeal or intra-meningeal. The chief cause of either form is injury. The minute secondary extravasations which occasionally occur in the peripheral nerves cause no symptoms.

Softening.—The expression "softening of the brain," when correctly used, relates to an actual diminution of consistence of the brain substance, due to the disintegration of tissue elements. The term is, however, frequently employed by the laity in an entirely different sense—that is, to designate almost any variety of chronic mental failure, but especially dementia paralytica and simple senility.

Literal cerebral softening may be the result of several distinct processes. Of these processes the chief are inflammation and arrest of blood supply. We shall consider here the latter only.

Arrest of blood supply depends on the occlusion of an artery. This occlusion deprives a certain area of the brain of its nutrition, because, in many regions of the brain, the terminal arterioles do not anastomose sufficiently to permit a re-establishment through the collateral circulation of the nutritive processes in the dependent part. The tissue elements, thus deprived of nutrition, lose their vitality and their function, and undergo disintegration—that is, softening,—necrotic softening it is termed. The process is also termed acute softening, owing to the rapidity with which the necrosis occurs.

Two distinct pathological processes may cause obstruction of an artery. These are thrombosis and embolism. Thrombosis is the formation of a blood clot within a vessel at the point of obstruction of the vessel. Obstruction from embolism depends on the lodgment of a plug—usually derived from a distant part of the vascular system—in an artery which is too small to permit its further passage. Thrombosis is sometimes secondary to embolism from coagulation in the distal end of an obstructed artery, and embolism is occasionally secondary to thrombosis, from detachment and subsequent advance of a thrombus,

but the two processes are usually quite distinct and separate.

Thrombosis of an artery generally results from local disease of the arterial walls, but occasionally it is due to morbid blood states in the absence of any changes in the artery. The predominant forms of arterial change are atheroma and syphilitic endarteritis. Both atheroma and syphilitic disease cause changes in the arterial walls, which usually narrow the lumen and roughen the inner surface of the thickened intima. The alteration of the inner surface of the vessel disposes to the formation upon it of a clot. Any morbid blood state, or any condition which weakens the action of the heart, or otherwise leads to the slowing of the blood current through the diseased vessels may facilitate the occurrence of thrombosis.

Atheroma involves chiefly the arteries at the base of the brain, especially the internal carotid, middle cerebral, basilar, and vertebral. Obliteration of the lumen of the middle cerebral or of its branches causes softening in or near the central ganglia or internal capsule. Occlusion of the pontine branches of the basilar causes softening of the pons. It is important to note that the atheroma is often symmetrically distributed.

Atheroma is of frequent occurrence during the second half of life. Occasionally it occurs earlier—at thirty-five or forty years of age. Its early occurrence is facilitated by the existence of chronic diffuse nephritis, an important clinical fact. Atheroma increases in frequency as age advances.

Syphilitic endarteritis leads to a greater narrowing of the calibre of the affected vessel than do other forms of arterial disease, and sometimes the thickened tissue formation may in itself obliterate the lumen. Generally a thrombus forms when the vessel is considerably narrowed.

The vessels involved in the process are chiefly those of the base, but the small arteries are often diseased, and may be diseased when the changes in the basal vessels are slight or wanting. The distribution of the change may be symmetrical, but it is usually irregular.

Syphilitic arterial disease is usually a result of acquired syphilis, and as the arterial changes are commonly developed in the course of four or five years after infection, they are generally operative in producing softening during adult life and especially during early adult life.

In *Embolism* the plug takes its source from some point between the lungs and the artery which is obstructed.. In a large majority of cases the obstructing embolus comes from the heart, being derived either from vegetations on the valves or from a coagulum in the left auricle. The embolus may come either from the mitral or the aortic valve. Generally it comes from the mitral valve, and mitral stenosis is the most frequent valvular cause of embolism. Embolism occurs somewhat less frequently in the course of chronic endocarditis than in recent cases of endocarditis or in the course of fresh exacerbations of endocardial inflammation.

An embolus sometimes takes its origin from the aorta, when this is the seat of atheromatous change. There is some reason to believe that minute emboli of septic material may pass through the pulmonary capillaries and find lodgment in the brain. Only capillary cerebral vessels can be obstructed by emboli so minute that they do not lodge in the lung capillaries. If localized cerebral softening results from the lodgment of septic material this is purulent. There may be multiple foci of septic softening in the brain, and these may coalesce to form abscesses. Whenever the embolus contains septic material derived from any part of the vascular system (usually from

ulcerative endocarditis), there may be septic inflammation about the obstructed vessels, with clinical evidences of septic infection.

Embolism occurs at all periods of life. It is particularly frequent between adolescence and middle life. Its occurrence is usually associated with conditions which are attended with endocarditis—especially with acute articular rheumatism, chorea, and scarlet fever—and occasionally it results from the endocarditis which sometimes develops in the course of acute specific diseases. Conditions which increase the coagulability of the blood may favor the occurrence of embolism by the formation of a clot on diseased valves or in a feebly acting heart, with subsequent detachment. Embolism from this cause is not very uncommon in the puerperal state. The same blood-state may lead to the occurrence of thrombosis under similar circumstances.

Acute Softening of the spinal cord, from loss of blood supply, is unknown as a distinct condition. The possibility of the occurrence of necrotic softening from vascular obstruction as the first stage of inflammation is mentioned elsewhere.

Thrombosis of Sinuses.—Thrombosis of sinuses of the brain may result (1) from any blood state or condition of the circulation that favors coagulation (primary thrombosis), or (2) from disease adjacent to the sinuses (secondary thrombosis).

Primary Thrombosis is usually associated with protracted and exhausting disease—for example, acute specific fevers, tuberculosis, carcinoma, chronic suppuration, and very frequently, in infants, prolonged and exhausting diarrhœa. These different causes are operative in two ways—by weakening the action of the heart, and by increasing the coagulability of

the blood. The superior longitudinal sinus is by far the most frequent seat of sinus thrombosis, probably because the blood current through it is exceedingly slow and feeble. It has been supposed that primary thrombosis occurs in cerebral veins, as distinguished from sinuses, and that a lesion of this nature, in the veins of the convexity, may underlie many cases of infantile hemiplegia. There is at present little evidence that such a process is ever thus operative, but with the present state of our knowledge the possibility that venous thrombosis constitutes the pathological basis of some cases of infantile hemiplegia cannot be denied.

Secondary Sinus Thrombosis is most often due to caries of cranial bones, particularly of the bones of the ear. Occasionally it results from disease external to the cranium, as erysipelas, or carbuncle of the face or neck. Almost any cerebral sinus may be the seat of secondary thrombosis, but the sinus affected is usually the one nearest the local disease. The local process is generally ear disease; hence the superior petrosal or lateral sinus is the usual seat of the thrombosis.

Local disease usually causes thrombosis in one of two ways. The inflammation either passes directly to the wall of the sinus, causing a phlebitis, which results in thrombosis, or a clot passes by extension to the sinus through a vein leading from the seat of the local disease to the sinus. In the former case the wall of the sinus is inflamed (phlebitic thrombosis); in the latter the wall of the sinus is healthy.

Inflammation.—Inflammation of the *Substance of the Brain*, as distinguished from inflammation of the membranes of the brain, is of rare occurrence, and when it occurs is usually secondary in origin and restricted in its limits. Acute cerebritis may follow injury to the head,

or bone disease, but constitutes a condition of little medical interest, unless it is accompanied with meningitis. The cerebritis of injury usually goes on to the formation of abscess. Tumors are often surrounded by a zone of inflammatory softening, and there is usually a zone of inflammation about foci of acute softening. Acute specific diseases, and septic conditions generally, may lead to the formation of small foci of encephalitis containing many micrococci. An acute idiopathic cerebritis (polio-encephalitis) has been considered the probable cause of many cases of infantile hemiplegia. There is as yet no direct evidence that this is the case, but there is some reason to believe that such a process may be effective in a small proportion of cases of sudden cerebral palsy in children.

Meningitis.—*Inflammation of the Membranes of the Brain* is from a medical standpoint by far the most important form of intracranial inflammation. Inflammation of the dura mater (pachymeningitis) is of rare occurrence except after injury. A peculiar form of pachymeningitis is that known as pachymeningitis interna hemorrhagica or hæmatoma of the dura mater. The lesion in these cases consists of delicate newly formed connective tissue, rich in small blood-vessels, and containing in its meshes, or on its surface, the remains of extravasations of blood.

Inflammation of the Pia Mater, or pia-arachnoid (lepto-meningitis), is of very frequent occurrence. The inflammatory process may be diffuse, and affect chiefly the base or the convexity, or it may be localized within narrow limits. Certain forms of meningitis, dependent on the grade or nature of the inflammatory process, may be distinguished. These are simple, purulent, tubercular, and syphilitic meningitis. It is impossible to draw a sharp line between all cases of simple and purulent meningitis. The cases to be classed as purulent meningitis are

those in which a layer of purulent exudate over the membranes inflamed is distinctly visible to the naked eye. All cases of inflammation (except specific forms) in which the amount of exudation or infiltration falls short of producing a distinct purulent layer are to be classed as simple meningitis. In many of these cases of simple meningitis there is merely a slight or considerable opacity and thickening of the pia in certain parts, though the microscope shows the presence of distinct and often considerable infiltration of the membrane with small spheroidal cells. It is difficult to say to which class some cases of meningitis belong. The difference between the two varieties of inflammatory change is probably merely one of degree and not of kind.

Simple or Non-Purulent Meningitis is considerably more common than the purulent form. It is sometimes met with after insolation and, more frequently, in the course of acute specific diseases, or without apparent cause. Focal lesions of the brain, such as tumor, abscess, and occasionally softening or hemorrhage, may excite a localized simple meningitis in their vicinity. Inflammatory adhesions between the dura and pia mater frequently occur in the neighborhood of new growths.

Purulent Meningitis most commonly results from adjacent suppuration. A frequent cause is disease of the middle ear. Another common cause of purulent meningitis is suppuration in distant parts. Thus it is observed in various forms of pyæmia and septicæmia. It also occurs at times in the course of acute infectious diseases. The pathological changes in purulent meningitis may involve the entire extent of the membranes or chiefly the convexity or base. Usually the changes are extensive, and occasionally they extend to the spinal meninges. Purulent meningitis is, of course, always of

bacterial origin. Several different forms of micrococci may give rise to it.

Tubercular Meningitis is much the most common variety of meningitis. Its distinctive character is the occurrence of miliary tubercles in the pia in association with meningitis which generally possesses the appearances of simple meningeal inflammation and is rarely purulent. The membranes at the base are usually chiefly involved in the inflammatory exudation, and the distinctive symptoms of the process are those of unequal damage to the cranial nerve-roots. The products of inflammation are especially abundant about the chiasma, in the interpeduncular space, and on the inferior surface of the cerebellum. The tubercles themselves are usually seen as minute translucent grayish-white granulations, which are most numerous about the smaller arteries of the pia. They are apt to be especially numerous about the vessels at the commencement of the fissure of Sylvius.

Tubercles of the pia may exist without the gross appearances of meningitis, and the development of actual meningitis is probably always preceded by the deposition of miliary tubercles in the pia. There seems to be little relation between the number of the tubercles and the extent or degree of the inflammatory process. Clinically it is impossible to distinguish between cases of miliary tuberculosis of the membranes without inflammation, and tubercular meningitis, since the symptoms of the former condition cannot be distinctly separated from those of the latter. Massive, or solitary tubercle, however, not rarely gives rise to the symptoms of tumor before the development of tubercular meningitis. When such a meningitis is grafted on a pre-existing tuberculous growth the symptoms of the two conditions are complex in character.

Tubercular meningitis probably depends on the deposition of tubercle bacilli in the vessels of the pia. In a very large proportion of cases the tubercular process in the meninges is secondary to a similar process elsewhere. In children it is almost invariably associated with general tuberculosis. Cases occur, however, in which there are no indications of tuberculosis and in which the meningitis appears to be primary. These cases usually occur in young adults.

The primary focus of tuberculosis may be in almost any part of the body that is the seat of such disease. In adults the primary process is usually in the lungs. In children it is often in the bronchial, cervical, or mesenteric glands. Tubercular meningitis is particularly a disease of childhood, but may occur at any period of life.

Syphilitic Meningitis is usually a chronic process of limited extent. Occasionally the inflammatory products involve the meninges of the base or of the convexity; generally they are limited chiefly or entirely to a small region of the base, especially of the posterior fossa of the skull. The pia is usually much thickened with the inflammatory exudation, and may be glued to the dura. In a later stage of the process the inflammatory material undergoes organization, and thick layers of fibrous tissue may surround and compress the nerves at the base. When the meninges of the medulla are the seat of such a chronic inflammatory process, the openings of the fourth ventricle may be obliterated, with consequent internal hydrocephalus. Frequently syphilitic meningitis occurs about a syphilitic new growth (gumma).

Inflammation of the Spinal Cord is a condition of frequent occurrence. The morbid process may begin in the nerve elements themselves, or in the neurogliar elements, and may involve the gray substance or both gray and

white substance. Inflammation causes disintegration of the fibres and cells involved, and the first gross alteration that usually results is a diminution in consistence—that is, softening. In the course of time the inflammatory products may undergo organization into new connective tissue, with perhaps an ultimate increase in the consistence of the cord. When the morbid process runs a chronic course, the cells and fibres undergo slower disintegration, and are gradually replaced by the products of cell proliferation in the non-nervous elements. If the changes are very chronic, the cord may be little or not at all softened in consistence. It may be difficult in such a case to decide whether the process in question is really inflammatory and not degenerative in character, and the decision in a doubtful case will depend much on our conception of inflammation.

The mechanism by which myelitis is produced is exceedingly obscure. In many acute cases the inflammation is probably due to the invasion of the vessels of the cord by micro-organisms. It is certain that an anæmic necrosis or softening of the cord (especially the gray substance), from arrest of blood supply, may go on to inflammation, and it is not improbable that vascular obstruction by thrombosis or embolism is a more important factor in causing myelitis than has been supposed. The elements of the cord, especially of the gray substance, have very little or no power of repair.

Inflammation of the Spinal Meninges may involve chiefly the dura (pachymeningitis), or the pia (leptomeningitis). The process may run an acute or chronic course. Acute inflammation rarely remains limited to the membrane in which it begins ; it extends to the other. Chronic inflammation, on the other hand, may remain limited to the membrane (dura or pia) in which it commences. Hence

it is only in chronic cases that the symptoms of meningitis can be referred to one or the other membrane, according to their character, with any degree of certainty.

For practical purposes it is convenient to divide meningitis into two forms—external meningitis, and internal meningitis.

External Meningitis is that form which begins outside the dura mater. It is probably always secondary to adjacent disease, and of such disease the most frequent cause, by far, is bone disease—caries. The inflammation is usually purulent in character or semi-purulent. Frequently the dura is covered by a thick layer of soft or hard cheesy matter containing tubercle bacilli. These inflammatory materials, if abundant, may seriously compress the cord, and then the symptoms of compression are combined with those of inflammation. The nerve-roots, as they pass through the dura, are either irritated and inflamed or compressed and atrophied. The vertical extent of the inflammation is usually limited.

Internal Meningitis, or inflammation inside the sheath of the dura, usually commences in the pia, but may extend to it from the inner surface of the dura. The inflammatory process may be simple (very rarely), purulent, or tubercular, or, in chronic cases, syphilitic. The causes of acute internal meningitis are often obscure. The disease may follow exposure to cold, trauma, surgical procedures, etc. Sometimes it occurs in the course of acute febrile diseases or septicæmia. Not rarely it results from extension, either from the membranes of the brain or from external meningitis. The symptoms in acute cases are : first, those of irritation of the nerve-roots, later those of pressure. The inflammation is generally of wide extent when it is acute. In both acute and chronic meningitis, it must be noted, the sub-

stance of the cord may suffer from the inflammation (meningo-myelitis), and the symptoms of the two conditions are then mixed. Probably all cases of acute internal meningitis are of bacterial origin.

Inflammation of Nerves—Neuritis—may be acute or chronic, primary or secondary, interstitial or parenchymatous. The interstitial form is that in which the connective tissue which separates the fibres is chiefly involved. In the parenchymatous or degenerative variety the fibres suffer chiefly and primarily. Secondary neuritis may be syphilitic, tubercular, cancerous, or leprous, according to the character of the process to which the inflammation is secondary. Syphilitic and tubercular neuritis affect chiefly the central ends of nerves, which become involved, either from the direct extension of corresponding inflammatory processes in the meninges, or from similar inflammations in adjacent tissues. Syphilitic neuritis frequently involves the cranial nerves.

The term neuritis is ordinarily used to include two different processes. First, it is applied to those changes in nerves which are unquestionably inflammatory in origin, including increased vascularity, small spheroidal-celled infiltration, and the formation of new connective tissue, with increase in the size and consistence of the nerve. Secondly, the word is employed to include destructive changes in the nerve-fibres, that extend down the nerve, beyond the seat of the original inflammation, to the periphery. These changes are, histologically, essentially those that occur in nerve-fibres which have been separated partially or entirely from their ganglion-cells; they are the changes of secondary or Wallerian degeneration (pp. 2 and 6). In many instances these degenerative alterations are not so severe in degree as those which occur, for example, after nerve-section, but they are the

same in kind. Slight evidences of inflammation may extend a short distance down a nerve after a traumatism, but the changes soon become purely degenerative in character (p. 283). Occasionally a neuritis passes up a nerve (neuritis migrans, ascending neuritis), and the changes are then purely inflammatory in nature.

Generally one nerve trunk only is involved in neuritis, but many nerves may suffer at the same time in the condition called multiple neuritis. Multiple neuritis is most commonly a result of alcoholism, but it occurs not very rarely in a variety of constitutional conditions, of which diphtheria, lead poisoning, and typhoid fever are the principal (toxic degenerations). Injury, both slight and severe, is a very common cause of neuritis. Extension from adjacent inflammation is also a frequent mode of origin. When the cranial nerve-roots or the spinal nerve-roots become the seat of inflammation, it is usually by extension.

Abscess.—*Abscess of the Brain* is usually the result of chronic suppurative inflammation of the middle ear, of traumatism with or without injury to the skull, or of suppurative processes at a distance,—as, for example, empyæma, suppurating cavities in the lungs, etc. In rare instances it results from caries of the nasal or orbital bones. Chronic otitis is the most common cause (about 42 %), and traumatism stands next in frequency (about 24 %). Chronic otitis is often associated with caries of the temporal bone or of the mastoid cells. The abscess is usually in the cerebral or cerebellar hemisphere of the side of the otitis,—nearly four times as frequently in the former as in the latter. When the abscess follows disease of the mastoid cells it is apt to be in the cerebellum. Abscess is rare as a result of pyæmia cerebral, and when it occurs the collections of pus are multiple and usually

small. In a considerable proportion of cases of abscess (about one sixth) there has been no discoverable cause.

Abscess may occur in any region of the brain. It is, however, most frequent in the cerebrum or cerebellum, and only occurs rarely in the pons, medulla, or basal ganglia. The collection of pus is generally within the substance of the brain, but sometimes it is on the surface, and in this case the meninges, which are locally inflamed and thickened, form the outer wall of the abscess. As a rule there is one abscess only (about four fifths of all cases). When there are two or more abscesses, they are generally the result of a septic process at a distance. When abscess is due to ear disease, or follows traumatism, it is single in about nine tenths of the cases. In size, abscesses vary between that of a hen's egg and a walnut.

Symptoms of cerebral abscess may be few or absent, in chronic cases, for a considerable period of time, sometimes for years, notwithstanding the lesion is large and in a situation from which focal symptoms might be expected. When acute symptoms eventually commence, they are often dependent on rupture, either into the lateral ventricles, or into the membranes, with subsequent ependymitis in the former case, with meningitis in the latter, if the patient lives long enough. More frequently, acute cerebral symptoms are due to enlargement of the abscess without rupture. All cases of cerebral abscess depend on micro-organic infection. Several different varieties of pyogenic bacteria appear competent to cause cerebral abscess.

Abscess of the Spinal Cord is a condition of extremely rare occurrence. Acute inflammatory affections of the cord seldom go on to abscess formation. Small accumulations of leucocytes sometimes occur in the gray substance of the cord (*e.g.*, in poliomyelitis), but these col-

lections are seldom visible to the naked eye, and can never be said to constitute abscess. In cases of purulent internal meningitis, however, pus may form in large quantity in the substance of the cord, and this probably constitutes the only mode of abscess formation in the spinal cord. The purulent meningitis in these instances is always of septic origin. Thus, in one case, it followed a fetid bronchitis ; in another it was secondary to gonorrhœa. Occasionally abscess is secondary to traumatism (especially fracture of vertebræ).

The practical importance of abscess of the spinal cord is not great, as its distinction from myelitis is usually impossible. Occasionally acute irritative spinal symptoms, associated with a cause of septic suppuration, may point to abscess rather than myelitis. In many instances the acute spinal symptoms of abscess are obscured by the symptoms of the pre-existing purulent meningitis.

Tumor.—*Tumor of the Brain* may be of almost any variety, but certain kinds are much more common than others. Syphilitic and tubercular intracranial growths (so-called diathetic tumors) are more common than other varieties. For clinical and other reasons it is convenient to speak of syphilitic and tubercular growths as tumors. They are not, however, tumors in the strict sense, and it is preferable to regard them as the results of specific forms of inflammation. Next in the order of frequency come the sarcomatous tumors, sarcoma and glioma. Then comes carcinoma, which is always of the soft variety. Other kinds of cerebral tumor (fibroma, osteoma, lipoma, neuroma, echinococcus and cysticercus cysts) are rare, but are occasionally encountered. Intracranial aneurism (of the larger vessels), though it does not constitute a new growth, may be classed clinically with tumors because of its pressure symptoms.

The cause of most intracranial tumors is obscure. Generally speaking they are twice as common in males as in females. During the first six months of life, and in old age, they are rare. The majority of cases occur in childhood and in early adult life. Syphilitic tumors occur chiefly between the twenty-fifth and fiftieth years, and in consequence of acquired syphilis. The time of their development after the initial lesion is commonly between five and ten years, but may be considerably earlier or later. Three quarters of all tubercular tumors of the brain occur before the twentieth year, usually in subjects who have a family history of phthisis or who have signs of pulmonary tuberculosis.

Glioma and sarcoma are most frequent during adult life. Carcinoma is exceedingly rare before fifty. Local injury occasionally operates as an immediate excitant of a new growth, and such an influence has been traced not only in tumors which, like glioma, are supposed to be of purely local origin, but in the case of the diathetic tumors (syphilitic and tubercular).

Tumors may either compress or infiltrate the substance of the brain ; some do both. Growths that spring from the dura or from the bones of the skull usually compress but do not invade the brain tissue. Growths that originate in the brain substance or in the pia may either compress or infiltrate it. The brain tissue is gradually destroyed, in either case, before the advance of the tumor, and there is usually a corresponding loss of function, though it is important to note that the degree of functional loss (expressed as symptoms) may be much less than our knowledge of the functions of the parts and the size of the tumor would lead us to expect. This probably depends on the fact that many of the nerve elements which are compressed by, or included in, the advancing tumor

are not deprived of all functional power. We have already noted the tolerance of nerve elements, especially fibres, to gradual pressure. The development of a tumor generally gives rise to irritation, irritation which results partly from the pressure exerted by the growth, partly from the vascular derangement that is associated with it. This irritation may be considerable in degree, and, in the immediate vicinity of the advancing tumor, may be inflammatory in character. This is the cause of the zone of softening which often surrounds a tumor. The meninges, and particularly the pia, are apt to be inflamed in the neighborhood of the new growth, and some meningeal inflammation may occur even at a distance from a rapidly growing tumor. Many of the most important and distinctive symptoms of tumor are referable to this irritation incidental to their growth, and it is an important fact that the degree and range of these symptoms are often disproportioned to the size of the tumor.

There remain to be mentioned certain individual forms of tumor that possess some diagnostic importance.

Tubercular tumors of the brain are usually in the cerebral substance but near the surface, yet often unconnected with the membranes ; occasionally they spring from the dura. Their most frequent seat is the cerebellum, but they occur almost as frequently in the cerebrum. In 20 % of recorded cases they have been multiple. They compress the brain tissue, and do not usually invade it to any great extent. Sometimes they grow rapidly, sometimes slowly ; often they develop rapidly and then become stationary. A tubercular growth may cause very little irritation.

Syphilomata are usually superficial and connected with the pia ; occasionally they spring from the dura. They compress but usually do not infiltrate the brain. Their

chief seat is the cerebrum, at the base of the brain, and somewhat less frequently they occur in the central cortex, cerebellum, and pons. A syphiloma usually grows rather rapidly and causes considerable and often great irritation.

Gliomata are single nine times out of ten. They develop in the brain substance, usually in that of the cerebrum (one half of the cases), often in the cerebellum (one quarter of the cases). They invade the brain substance without displacing it. They surround and annihilate the nerve elements, and soon compass considerable destruction. The growth of a glioma is usually slow, sometimes very slow. It does not compress the brain as do sarcomata. All gliomata have a marked tendency to undergo fatty degeneration with central liquefaction (cyst formation). The tumor is often highly vascular, and is sometimes the seat of sudden hemorrhage, which may be indicated by an apoplectic seizure.

Sarcomata may grow from the brain substance, from either membrane, or from bone (especially of the base). Usually they do not infiltrate like gliomata, but possess distinct limits, though the softer varieties may have the invasive tendency of glioma. Their growth is usually slow, and they are generally single. Carcinoma may originate from the dura or the brain substance. It is usually secondary to carcinoma in other parts of the body, most frequently of the orbit or the retina. It may occur in any part of the brain, but is most often observed in the cerebral hemisphere. It is usually single, and generally both invades and compresses the brain substance

Tumors of the Spinal Cord.—New growths within the spinal canal may originate external to the dura, or internal to the dura. The tumors that arise outside the dura may take their origin from the membrane itself, from the

extra-dural tissue (fat) or from the adjacent bones. Sarcomata, carcinomata, and enchondromata occasionally spring from the bones or intervertebral substance. Lipomata may originate in the fat between the dura and the bone, but are very rare. Extra-dural growths produce symptoms by compressing the spinal cord, and irritating or compressing the nerve-roots with which they are in contact. Accumulations outside the dura, of inflammatory nature from tubercular bone disease (caries), give rise to effects similar to extra-dural new growths. The former are twice as common as all forms of tumors of the cord and membranes combined; all forms of new growth of the cord are rare. The ratio to tumors of the brain is one to thirteen.

Of the tumors that originate inside the dura, the majority begin in the membrane rather than inside the substance of the cord itself. Such tumors are most often syphilitic; less frequently they are sarcomatous or myxomatous. They compress the cord and its nerve-roots, and are apt to damage the former more seriously than do extra-dural tumors, but they do not often invade the substance of the cord, even if they spring from the pia.

Of the new growths which occur within the substance of the cord itself, syphiloma and glioma are the most common, or, more accurately, the least infrequent. Sarcomata, myxomata, and tubercular tumors are rare. Syphilomata usually spring from the pia, gliomata from the peri-ependymal tissue immediately about the central canal of the cord. Gliomata often have a considerable vertical extent, and, when vascular, are sometimes the seat of hemorrhage, but rarely contain cysts as do those of the brain. Sometimes, also, an extensive infiltration of the spinal cord with gliomatous tissue, constituting gliomatosis, which should not be confounded with gli-

oma, breaks down spontaneously and leads to the formation of cavities in the cord, and which usually connect with the central canal of the cord,—a condition known as syringomyelia.

Tumors growing within the substance of the cord compress and destroy the nerve elements. They are less apt than extra-spinal tumors to compress and irritate the nerve-roots. What has been said of the progress of the different varieties of intracranial tumors applies in general to tumors of the cord. Tumors of the spinal cord are usually single, and small or moderate in size. Sarcomata are sometimes multiple and very numerous. When they invade the substance of the cord, its tracts are frequently the seat of secondary degeneration, ascending and descending. Malignant tumors of the cord are ten times as frequent as benign tumors. Tumors are more commonly situated on the posterior and lateral surface of the cord than upon the anterior aspect.

The *Cauda Equina* is occasionally the seat of a sarcoma, or fibro-sarcoma. A tumor in this region may attain considerable size without producing pressure symptoms, because of the large size of the vertebral canal.

Tumors of the *Peripheral Nerves* are occasionally observed. Any morbid growth on a nerve is called a neuroma, but a distinction is made between true neuromata, which consist of nervous tissue, and false neuromata, which contain no nervous elements. False neuromata are most often hard fibromata, occasionally sarcomata, myxomata, or gliomata. Neuromata generally lie between the nerve bundles, which they push aside, but do not usually involve. They are usually single, but may be very numerous. The nerves of the extremities are most often affected.

Degeneration.—The tissue elements of the brain, and of the spinal cord, frequently undergo a gradual change in structure known as degeneration. This change consists of two distinct pathological processes. One of these is the slow wasting of the nerve elements—the gradual atrophy of the ganglion-cells, or nerve fibres, or both. The other is an overgrowth of the interstitial or connective-tissue (neurogliar) elements. It is customary to distinguish two forms of the degenerative process—that which is confined to structures which possess the same or similar functions, and that which is apparently random in its distribution, and is not limited to structures having the same function. The former type of the degenerative process is well exemplified by the so-called "system diseases" of the spinal cord, in which particular (functional) tracts of fibres are the exclusive seat of the morbid process. Locomotor ataxia, in which the posterior median columns of the cord are degenerated, and primary lateral sclerosis, in which the crossed pyramidal tracts are similarly involved, are illustrations of system-diseases. Other examples are secondary degenerations—that is, degenerations which occur in the brain and in the spinal cord, when certain tracts of nerve-fibres have been separated, from any cause, from the ganglion-cells that constitute their trophic centres. When the fibres have been thus separated from their nutritive centres for a period of two, three, or four weeks, the medullary sheath and the axis-cylinder undergo disintegration, and the tract or tracts in which this change occurs becomes gradually gray in color, somewhat shrunken, and perhaps somewhat harder than normal. In certain tracts these secondary degenerative changes progress in an upward direction (ascending degeneration), in others in a downward direction (descending degeneration); in general, the degeneration progresses in the direction in which the tracts

involved habitually conduct nervous impulses. Thus the motor tracts, the direct pyramidal tract and the crossed pyramidal tract, undergo descending degeneration, whilst the sensory tracts, the posterior median columns, the direct cerebellar tracts, and the antero-lateral ascending tracts (p. 33) degenerate in an upward direction. A transverse lesion of the cord is often the cause of secondary degenerations, both of an ascending and descending character. This is well seen in transverse myelitis and in certain tumors of the cord.

After hemiplegia from any organic disease of the brain there is a descending degeneration of the direct pyramidal tract on the same side and of the crossed pyramidal tract of the opposite side, the degenerative process beginning in the motor path just below the lesion in the brain, whatever may be its position. In the primary degenerative disease of the cortex known as dementia paralytica, there are usually extensive secondary degenerations of the pyramidal tracts of either side, consequent on the changes in the ganglion-cells of the cortex.

It is highly probable that, in all the system degenerations, the changes in the nerve elements are primary and that the interstitial overgrowth is a secondary change. In the second type of degenerative changes, on the other hand, this is not the case. Here the alterations begin, in all probability, in the interstitial elements external to the nervous elements, which are involved secondarily. A good example of this type of degeneration is insular sclerosis, in which patches of degenerative change are irregularly scattered through the central nervous system. Irregular areas of degeneration in the brain and spinal cord are also observed in some cases of the so-called traumatic neuroses, and such changes are probably more frequent after traumatism than has been supposed. These

changes are sometimes associated with extensive hyaline and fatty degeneration of the cerebro-spinal arteries. Some instances of this second type of degeneration are probably intermediate in their pathological characters between true degeneration and chronic inflammation.

The etiology of the primary degenerations of the brain and spinal cord, affecting nerve-fibres or nerve-cells or both, is obscure. What is known concerning it will be alluded to elsewhere in this chapter.

The structural changes which the nerve-fibres of a peripheral nerve undergo after their separation from the cells to which they belong, differ somewhat in character from the changes of secondary degeneration in the central nervous system. The medullary sheaths of the nerve-fibres break up into segments, and these in turn break up into droplets of various size and shape. When these alterations have progressed to a certain point the axis-cylinders usually break up and may be more or less completely destroyed. The nerve-fibre sheaths (sheaths of Schwann) and their nuclei do not usually degenerate. The products which they contain of the disintegration of the medullary sheaths may be eventually absorbed. In a case of complete degeneration of a nerve from injury, the complete segmentation of the myeline sheaths and axis-cylinders is evidenced clinically by a loss of nerve irritability to electrical stimulation. Such a loss of irritability usually occurs at the end of two weeks, but it is not a sudden loss, and is preceded by a fall in irritability which probably corresponds to the partial segmentation of the fibres and nutritional changes in the axis-cylinders. In some cases a slow process of regeneration occurs during the second, third, and fourth month of the injury. This regenerative process consists of a development of new axis-cylinders, which in time develop myeline sheaths, from the central end of the nerve.

The causes of nerve degeneration, which is often a partial process not progressing beyond a moderate degree of structural change, are numerous. We may group them under three heads: first, mechanical injuries, such as incision, over-extension, crushing, or compression; secondly, disease of the special nerve centres (ganglion-cells of anterior horns and the motor cranial nerve nuclei) with which the peripheral nerves are in communication, as poliomyelitis; third, disease of the peripheral nerves themselves, as inflammation.

The process which has been here described as degeneration is regarded by some as inflammatory and not degenerative in character, and the condition has been described as one of parenchymatous inflammation. The ordinary evidences of inflammation are often marked in the neighborhood of the primary lesion, but seldom extend far below the lesion, where the pathological changes may be with propriety considered degenerative.

When a motor nerve undergoes degeneration, changes soon occur in the nutrition of the muscles. After a lapse of ten days or two weeks the muscle fibres become narrower and undergo distinct changes in their histology. If regeneration eventually occurs in the nerve, the muscles are gradually restored to their normal structure and function. If no regeneration takes place, the atrophy of the individual fibres, and of the muscles as a whole, progresses, but the decrease in size from muscular wasting is partially compensated by the growth of connective tissue between the muscular fibres. This new connective tissue gradually contracts, and this contraction may result in permanent shortening of the muscles involved.

Having reviewed the general pathology and etiology of the various morbid processes which occur in the brain,

spinal cord, and peripheral nerves, we may pass to the consideration of the indications that serve to distinguish the several lesions. These indications are of especial importance in establishing the pathological diagnosis in cases of disease of the brain and of the spinal cord, the diagnosis of the nature of peripheral affections being in general much simpler. The following considerations are therefore mainly applicable to disease of the central nervous system.

The indications above referred to as essential to the determination of the pathological diagnosis, are the following : first, *the manner of onset of the symptoms*, which usually makes it easy to distinguish between certain general classes of morbid processes to be hereafter mentioned ; secondly, *the causal indications*, which comprise the various causes of disease that can be traced or excluded ; thirdly, *the position of the morbid process*, taken in conjunction with the special and known liability of certain parts of the brain and cord to particular forms of disease.

I. The Onset of the Symptoms.—The time occupied by the onset of the symptoms—that is, the time that intervenes between the first symptoms of the lesion and their development to a considerable degree of intensity—is an important guide to the nature of the lesion. If the onset occupies only a few minutes or less, it is designated *sudden*, and the lesion which produces the sudden onset is almost always a vascular lesion (provided, of course, that the disease is organic). A vascular lesion in the brain may be either hemorrhage or softening from vascular obstruction ; in the spinal cord such a lesion is practically always hemorrhage. If the onset, instead of being sudden, occupies from an hour or two to a week, it is described as *acute*. An acute onset is usually indicative of an inflammatory process, but vascular lesions are

not always sudden in their development, which may occupy a few hours or days. When, therefore, the lesion develops in a few hours, it may be either vascular or inflammatory in nature. Which it is must be determined by the study of other indications.

When the symptoms attain a considerable degree of intensity only after the lapse of several weeks, the onset is said to be *subacute;* and such an onset usually characterizes the development of the subacute inflammatory processes, some forms of tumor, and in the spinal cord also some pressure lesions not dependent on tumor.

In cases where the development of the symptoms takes a *chronic* course—that is, occupies more than a month—the morbid process is usually a new growth or a degeneration.

Thus a knowledge of the mode of development of the symptoms may enable us at once to roughly classify the lesion in question. For instance, if the lesion is sudden, it must be vascular in character; if it is chronic, it is probably a degeneration or a new growth. As a rule, however, the time of the onset simply enables us to limit the process to one of two lesions. For example, a case which is intermediate between a sudden lesion and an acute lesion, and occupies several hours, may be either vascular or inflammatory in nature. Beyond this point the study of the onset cannot take us; the decision depends on other indications.

II. The Causal Indications.—The causal indications that bear on the diagnosis of the nature of the lesions of the nervous system are numerous and important. There is, however, a wide difference in their relative significance, some having, like age and sex, only a low value; others, like certain kinds of injury and certain forms of infection (syphilis, tuberculosis), possessing

a value, under some circumstances, that is highly significant. While the causes which are to be reviewed will of course be those that bear especially on the organic diseases of the nervous system, it will be well to touch on some of the relations of these causes to the functional forms of nervous disease.

The relations of each etiological condition will be given to each class of lesion (hemorrhage, degeneration, tumor, etc.) in so far as is practicable or desirable, and when profitable the more important relations between these conditions and particular clinical types of disease will be briefly stated.

It is convenient to group the etiological factors to be reviewed, as follows: age, sex, habits, infection, toxic influences, trauma, state of general health, psychic influence, heredity, influence of treatment.

Age.—A knowledge of the age at which the various morbid processes occur is sometimes of considerable value in determining the nature of a lesion. With most processes, however, the occurrence extends over so wide a period, with so nearly equal incidence in several decades, as to deprive the age factor of decided significance. The vascular lesions of the nervous system are met almost exclusively in the brain. Regarded as a group, these vascular lesions (including hemorrhage, embolism, thrombosis) occur at all times of life, but the various members of the group are especially frequent at particular periods. Thus, embolism is rare after fifty and before puberty; syphilitic thrombosis occurs chiefly in the third, fourth, and fifth decades, and both hemorrhage and thrombosis from atheroma are diseases practically limited to the second half of life. The vascular lesions which depend, like extra-dural hemorrhage and spinal meningeal hemorrhage, on injury, occur especially during

early adult life (twenty to forty). The inflammatory processes, like the vascular lesions, occur at every period of life, but are on the whole most common in early adult life and infancy. This is especially true of the acute inflammations. Thus, acute cerebral meningitis and cerebro-spinal meningitis are relatively most common before the twentieth year, and although purulent meningitis is a disease of adult life especially, this is due mainly to the usual traumatic origin of the disease. Acute poliomyelitis, by far the most common inflammatory affection of the substance of the central nervous system, is essentially a disease of infancy and childhood. An acute spinal lesion occurring in infancy or childhood is almost certainly poliomyelitis. Acute myelitis occurs particularly between ten and forty. The various forms of inflammation of the peripheral nerves (multiple neuritis, neuritis), acute and sub-acute, are particularly common in early adult life. More than one half the cases of cerebral abscess occur between ten and thirty (partly due to the influence of trauma). The more chronic inflammatory processes, both of the central nervous system and of the peripheral nerves, probably occur, as a rule, later in life than the acute lesions (for example, chronic pachymeningitis, chronic myelitis, chronic sciatic neuritis).

The best example of the influence of age is found in the case of the degenerative affections, of which probably more than two thirds of the cases commence between the thirtieth and fiftieth years. This group includes locomotor ataxia, primary spastic paraplegia, ataxic paraplegia, general paralysis, progressive muscular atrophy, etc. There are, however, some exceptions to the rule of commencement between the ages of thirty and fifty. Bulbar paralysis often begins after fifty, insular sclerosis most often begins between twenty and thirty-five, and Fried-

reich's ataxia most often between seven and twenty. But these cases form a very small proportion of all the cases of degenerative disease.

As a class the new growths show no striking features in relation to age. They are rare in infancy and after sixty. The most frequent forms (tubercle, glioma, syphiloma) belong particularly to the first half of life, the tubercular and gliomatous forms being most often observed between five and thirty, and the syphilomata between twenty and forty. Carcinomata belong to the second half of life.

The group of so-called functional nervous diseases belongs largely to the adolescent period and early adult life (fifteen to thirty-five). This is conspicuously the case with the most common forms, chorea, epilepsy, hysteria, neuralgia, and migraine. Paralysis agitans is, however, an exception, in that its period of greatest frequency is between forty and sixty. Wry-neck and neuralgia also occur very frequently between thirty-five and fifty, but these cases do not in any way invalidate the truth of the general statement made above.

Sex.—Looking at nervous diseases, in so far as we can, as a class, it is difficult to say in which sex they preponderate. Certain it is, however, that to the male sex belongs considerably more than half of all the cases of organic disease. This special liability on the part of the male appears to depend mainly on the effects of injury, alcohol, and syphilis, to all of which he is more exposed than the female. The functional forms of nervous disease are more frequent, as a class, in females, owing mainly to the preponderance of two common diseases, hysteria and chorea. In paralysis agitans, migraine, and tetanus, the greater number of cases occur in males—in the case of tetanus the large preponderance depending, no doubt, on the greater exposure of the male to injuries favoring the necessary infection.

The greater prevalence of organic disease in males is particularly noticeable in the group of degenerative affections.

In the case of locomotor ataxia the unequal incidence in the sexes is very striking,—being probably at least ten to one in favor of males. An even greater inequality is noticeable in an allied degenerative disease—dementia paralytica. A less striking inequality obtains in ataxic paraplegia and progressive muscular atrophy. Among the inflammatory processes there is a marked preponderance in males of those conditions that depend to a considerable extent, or mainly, on injury,—as acute myelitis, meningitis of the convexity, and abscess of the brain (two to one), on alcohol (multiple neuritis, pachymeningitis), or on syphilis (as syphilitic endarteritis, chronic meningitis, etc.). With the possible exception of embolic softening, the vascular lesions preponderate slightly, and in the case of cerebral hemorrhage, largely, in males.

The indications of the nature of the lesion which are derived from the consideration of age and sex, are, of course, to be regarded as quite subsidiary to the indications obtained from other causal factors. Some of the factors which have been mentioned may, however, be allowed weight when the more important indications, in a given case, appear evenly balanced, and occasionally the facts noted will prove of real service in diagnosis.

Habits.—A variety of influences in the production of nervous disease, which might properly be classed as habits, could be enumerated, but of these there is only a small number that deserve special attention. The excessive use of alcohol and tobacco, and sexual excess, including masturbation, belong to this class. Of these influences, alcoholic excess is by far the most important as regards the causation of organic disease.

Excessive Use of Alcohol.—A history of the excessive use of alcoholic drink serves to clear up the nature of a variety of affections of the nervous system. It is important, however, to distinguish between the acute disturbances from alcoholic intoxication and the different morbid states of the nervous system that develop gradually as a consequence of habitual excess. The most important form of acute disturbance from alcohol is delirium tremens (p. 596) or acute alcoholic delirium; other forms of pathological interest are the various manifestations of alcoholic insanity—maniacal, melancholic, etc. These acute alcoholic insanities are commonly associated with hereditary predispositions to mental derangement. The common variety of acute alcoholic intoxication, known as "intoxication" *par excellence*, does not concern us.

A feature of practical importance in the causation of various forms of nervous disturbances that are slowly induced by habitual excess is that they may occur in persons whose excess has always fallen short of actual intoxication. It may thus become difficult, in a given case, to say what constitutes excess. The daily quantity of alcohol that is borne without detriment varies much with different individuals, and with the same individual at different times and in different conditions of health. Sedentary occupations are particularly unfavorable to the perfect assimilation of considerable quantities of alcohol. Probably most adult males can safely dispose of the equivalent of two ounces of absolute alcohol in a day—many of much more, and some undoubtedly develop disease from the habitual use of smaller quantities. It results chiefly from the stronger forms of alcohol, especially from spirits, but the prolonged and excessive use of beer seems particularly apt to lead to multiple neuritis.

The organic diseases that might be accounted for by a history of alcoholic excess are chiefly subacute or chronic in course, and inflammatory in character. To these inflammatory lesions the peripheral nerves appear especially prone. Thus, by far the most frequent organic affection from alcohol is multiple alcoholic neuritis. Neuralgic pains in the limbs, without atrophy or weakness, are common as a result of alcoholic excess. They depend sometimes on slight acute neuritis (sensory form), sometimes on functional (nutritional) changes. Subacute and chronic myelitis, with or without neuritis, are occasional results.

Of the cerebral affections that may depend on alcohol, slight chronic meningitis (with slight optic neuritis, headache, and mental failure) is the most common, but habitual alcoholic excess favors atheroma of the walls of the cerebral arteries and is then a factor in producing cerebral hemorrhage. Two closely allied degenerative affections of the central nervous system—general paralysis and locomotor ataxia—occur frequently in alcoholics, but it is doubtful whether alcohol is ever the sole cause of either disease. Some cases of chronic alcoholic meningitis closely resemble general paralysis (pseudo general paralysis).[1] Atrophy of the optic nerve probably never results from alcoholic excess alone. Epilepsy is apparently an occasional consequence. Hemianæsthesia (of uncertain nature) is said to depend at times on alcoholism. By far the most common nervous affection that results from the habitual abuse of alcohol is a fine irregular tremor

[1] A special form of psychosis (Korssakow's disease) not infrequently accompanies alcoholic polyneuritis. The disease is marked by disturbance of attention, defective memory, and pronounced fabrications. Its pathological anatomy consists in marked atrophy of the cerebral cortex.

involving the lips, tongue, and hands, and often the legs—alcoholic tremor. Such habitual abuse also strongly favors the development of occupation neuroses and the muscular atrophies from over-use.

The Excessive Use of Tobacco (by smoking or chewing), appears to give rise to but few organic diseases of the nervous system. The principal condition consequent upon such excess is a peculiar form of amblyopia characterized by the existence of central scotomata, especially for the colors green and red. A variety of functional disturbances may be accounted for by the habitual excessive use of tobacco—headache, tremulousness, insomnia, irritability, palpitation, tachycardia, blepharospasm and various digestive disorders.

Sexual Excess is an exceedingly rare cause of organic disease of the nervous system, but a common cause of functional derangements. Repeated sexual acts have been the cause, in males, of hemorrhage into the lumbar enlargement of the cord, followed in some cases by myelitis. In some cases these conditions have resulted in males from coition (not necessarily repeated) in the erect position. A much less unusual effect of repeated sexual intercourse is transient weakness, slight or considerable, of the lower extremities. This depends probably on temporary nutritional derangement of the anterior cornual cells of the lumbar cord; perhaps in some instances on minute extravasations.

The sexual act several times repeated has in rare instances been the exciting cause of hemiplegic attacks in persons with atheromatous cerebral arteries. Similarly, persons with syphilitic arterial disease have been known to become hemiplegic after one or more sexual acts.[1]

[1] In one case of this kind the autopsy showed the presence of acute softening. The patient was a woman, and the paralysis came within

The functional nervous derangements which are the remote consequence of sexual excess are numerous and varied. They include the protean manifestations of neurasthenia and hysteria. Especially common, however, are irritability, slight mental failure, headache and other cephalic sensations than pain, dilated pupils, amblyopia, palpitation, various vasomotor symptoms (flushing, etc.), fibrillary twitchings, mental depression, undue fatigue, oxaluria, phosphaturia, and digestive disorders. These symptoms are of far more frequent occurrence in men than in women. In those who are subject to epilepsy, sexual indulgence occasionally determines the occurrence of paroxysms. Wry-neck sometimes appears to be due solely to sexual excess.

Masturbation.—It is exceedingly doubtful whether masturbation is ever a cause of organic nervous disease, though it may very remotely influence the course and development of such disease. The functional derangements consequent on this habit are, however, numerous, and of frequent occurrence. The derangements resemble in character the neurasthenic symptoms mentioned as dependent on sexual excess, with the general difference that they frequently attain a greater degree of severity.[1] A mental symptom which is some-

a few minutes after the sexual act. It is probable that the circulatory changes in the brain which follow and accompany violent sexual excitement may determine the occurrence of vascular lesions in such cases as that mentioned.

[1] Sexual excitement is probably always followed by an increase of indican in the urine. Normally this substance is present in the urine in very small quantities or is entirely absent. The presence of more than .020 gm. of indican in the twenty-four hours' urine is to be regarded as pathological. Although other causes than sexual excitement may cause an increase of indican in the urine, such increase is sometimes a valuable aid in the detection of the habit of masturbation.

what characteristic of masturbational excess, though it sometimes occurs in cases of ordinary sexual excess, is a peculiar disposition to ramble from one topic of conversation to another in a superficial or incoherent manner. Sometimes, again, there is a morbid tendency to drowsiness during the day, and, in rare cases, young subjects have been in stupor for many days as a consequence of repeated acts of masturbation.

The evil results of masturbation are more equally distributed between the sexes than are those of sexual excess. Though most common among individuals between the age of puberty and the twentieth year, masturbation is often practised by much younger subjects. The habit is not rarely begun in young children by the teaching of lascivious nursery-maids. Not infrequently it originates in young children from genital irritation due to the passage of the irritating urine that commonly results from digestive derangements.

The proportion of persons (of each sex) who practise masturbation at some period of life is probably very large. In the majority of instances the habit is not continued sufficiently long or with sufficient perseverance to give rise to serious functional disturbances.

Infection.—The introduction of pathogenic micro-organisms into the body is the essential condition of infection. After these have obtained mechanical lodgment in some part, local and general conditions, but imperfectly understood, determine their pathogenic influence. An extension (direct or through the blood or lymph channels) of infection from the parts first involved may lead to the implication of the nervous system, and such infection constitutes, perhaps, the most important class of influences in the production of organic disease of the nervous system. Two specific forms of infection, the

tubercular and the syphilitic, concern us especially, since they play a most conspicuous part in the causation of organic disease. The infection which results in suppurative processes in the nervous system (abscess, suppurative meningitis, etc.) is considered, for clinical reasons, under other headings.

Tubercular Infection.—The organic conditions of the nervous system which result from the invasion of tubercle bacilli from primary foci of infection are chiefly inflammatory in character. By far the most frequent of these inflammatory conditions is tubercular meningitis. In infancy and childhood this is much the most common form of cerebral disease. In connection with tubercular meningitis, or occurring quite independently, there may be an agglomeration of tubercles in the substance of the brain, constituting one or more massive or conglomerate tubercles (so-called "solitary" tubercle or "tubercular tumor"). Much less frequently the spinal cord is the seat of solitary tubercle, which produces the symptoms of tumor. The peripheral nerves are involved in a neuritic affection in some cases of advanced tubercular cachexia, and this affection has been regarded as a multiple neuritis of tubercular origin. The correctness of this view is rendered somewhat questionable by the fact that the class of patients in whom the affection occurs (advanced consumptives), is a class in whom alcoholic stimulants are extensively employed in treatment. Conditions are present, therefore, which favor the occurrence of alcoholic neuritis, and it is possible that many of the cases which pass for tubercular multiple neuritis are, in reality, instances of alcoholic neuritis.

The forms of tubercular disease of the nervous system above enumerated, depend, with the exception of the neuritis above mentioned, on the presence of tubercle

bacilli in the tissues of the parts affected. Such local deposition is not usually the case with one of the most frequent consequences of tubercular disease, the so-called compression myelitis. Here, the lesion is a tuberculosis of the vertebræ, which leads to the accumulation of tubercular material *outside* a thickened dura, and consequent compression of the cord. The compression of the cord causes a diminution in its size, and sometimes an actual myelitis. But the myelitis which results from compression is only rarely tubercular in character, and when this is the case the inflammation is derived from a direct extension from the outer surface of the dura to the pia, and from the pia to the substance of the cord. In these cases there is probably always the formation of tubercles in the cord before the actual myelitis develops.

The tubercular affections of the nervous system may be secondary to tubercular processes in a variety of situations. In adults, the primary lesion is usually pulmonary tuberculosis; in children, there is commonly general miliary tuberculosis, and the primary focus may be tubercular bronchial nodes, interstitial tuberculosis, tubercular bone disease (especially knee and hip), or tubercular teno-synovitis. In adults, the genito-urinary system is sometimes the seat of the lesion. In the male, the testicle, prostate, and seminal vesicles may be affected; in the female, the uterus and Fallopian tubes. Thus the infection which terminates in tubercular disease of the nervous system, may make its entrance by way of the respiratory tract, the digestive tract, the genito-urinary tract, or through slight injury to the skin at any point. In some cases the evidences of local tuberculosis are distinct (as in chronic pulmonary tuberculosis, or hip disease), and give most important indications for diagnosis, but it

is essential to note that in many cases the original lesion is so obscure in its symptomatology as to afford no indications whatever of the character of the lesion within the nervous system. Hence it is that the nervous lesions often have the aspect of primary affections. While, therefore, the presence of tuberculosis in some part of the body other than the nervous system may be of the highest aid in determining the nature of the nervous lesion, the absence of indications of such disease should carry very little weight if the symptoms themselves suggest a tubercular process.

Syphilitic Infection.—No form of infection gives rise with such frequency to organic disease of the nervous system as syphilis. Of the organic conditions that arise from syphilitic infection, some, like gummatous growths, are processes which are distinctive of syphilis and never occur as the result of any other cause than syphilis. Others, like certain degenerative processes of the brain and cord (locomotor ataxia, progressive muscular atrophy, etc.), though commonly dependent on syphilis, may arise from other causes, and possess no pathological features that are distinctive of syphilis. It is important, in thinking of the consequences of syphilitic infection, to bear in mind these differences in the relations of its effects.

Both the brain and spinal cord are very frequently the seat of organic disease which is the consequence of syphilitic infection. Of the specific syphilitic processes (gumma, endarteritis), the brain is a much more common seat than the cord.

The chief vascular lesion of the nervous system, of syphilitic origin, is acute softening of the brain from the thrombosis of cerebral vessels that results from syphilitic endarteritis. This is a very important and frequent clinical condition (p. 282). Whether syphilitic endarter-

itis of the vessels of the cord is a cause of focal softening and inflammation of the cord cannot be positively stated, but there is some reason to believe that it may not rarely give rise to these conditions. A much more rare effect of syphilitic vascular disease is intracranial aneurism.

An important class of syphilitic lesions is inflammatory in character. The clinical conditions which belong to this group are characterized by their subacute or subchronic course. This group includes pachymeningitis, cerebral and spinal, and leptomeningitis. Cerebral syphilitic meningitis is usually local. In the cord, inflammation of the pia is seldom local, and is usually associated with pachymeningitis. Inflammation of the substance of the brain and cord, possessing the pathological characters of a syphilitic process, probably does not occur. Whether acute myelitis is ever due to syphilis it is as yet impossible to say. Subacute disseminated myelitis probably depends in some cases on syphilis, but its precise relations to the disease are not fully understood. Whether syphilitic infection ever leads to multiple neuritis must be regarded as uncertain. If it does ever cause multiple neuritis, it must be in very rare instances. It is possible that some of the cases of Landry's paralysis which have been shown to be due to peripheral neuritis, were the result of syphilitic infection.

Syphilitic meningitis, either of the brain or of the cord, is not rarely associated with the formation of gummata. In such cases the symptoms of tumor are added to those of meningitis. Gummata may occur with very little meningitis, and the symptoms of tumor then preponderate. They are much more frequent in the brain than in the cord.

The degenerative diseases of the nervous system stand in an important relation to syphilis. None of them

appear to be invariably due to syphilis, and some of them probably never are due to it, but a considerable number are commonly a late sequel of syphilitic infection. In two important and closely allied conditions, locomotor ataxia and general paralysis, a history of syphilis can be traced in seventy-five or eighty per cent. of the cases, and probably exists in even a larger proportion. The various forms of ophthalmoplegia that are observed in tabes may occur as isolated conditions and depend usually on syphilis. The same may be said of cases of simple atrophy of the optic nerve, and of the rare cases of isolated Argyll-Robertson pupil. Progressive muscular atrophy and primary spastic paraplegia are probably occasional consequences of syphilis. In the case of ataxic paraplegia and insular sclerosis, it is very doubtful whether there is ever any dependence on syphilis.

A recognition of the various possible consequences to the nervous system of syphilitic infection is often of considerable aid in practical diagnosis. Having first determined from the character of the symptoms the location of the lesion, and from their course its probable nature (vascular, inflammatory, degenerative, etc.), we may ask ourselves, in any given case, whether the process that has thus been provisionally diagnosticated is one of those that may be the result of syphilis. If the process in question may be due to syphilis, it becomes very important to know whether the patient has a history of syphilis. In a considerable number of cases there has been syphilitic infection without the knowledge of the patient, and this is especially common in women, in whom the primary lesion is apt to be overlooked, and in whom secondary symptoms are often slight or absent. As a rule, in an intelligent person, who has been infected, it is not difficult to elicit a reasonably straightforward history,

but, even in such cases, slight symptoms may have passed entirely unnoticed and left no objective traces. It is important, therefore, to realize the fact that we can never positively exclude syphilis, unless we can exclude the possibility of infection from impure sexual congress. There are, however, many cases where the possibility of such infection cannot be excluded, in which the probability of infection having ever occurred is exceedingly slight. When, in a given case, there is uncertainty as to past syphilitic infection, to what extent should this fact be permitted to influence the diagnosis? It is probable that too much weight is allowed to this uncertainty. The important principle to bear in mind is that any decided indications, positive or negative, regarding the nature of the lesion, that may be gathered from the nature and course of the symptoms, must be allowed full weight even if they are not supported by what is known or believed regarding a history of syphilis. Thus, if the symptoms justify the belief that we have to deal with a syphilitic process, a process such as rarely depends on any other cause than syphilis, this belief should be maintained, even if no history of syphilis can be elicited, —unless, of course, the possibility of infection can be quite excluded, which is not commonly the case in adults. On the contrary, if the case is one in which the symptoms make a syphilitic lesion unlikely, this likelihood is not distinctly lessened even if it can be shown that the patient has been infected. But if the indications of the nature of the process as regards syphilis are equivocal, we must be guided largely by the facts regarding the history of syphilis, and even if these are clear there may remain a considerable chance for error.

There is no satisfactory proof that syphilis can ever be looked upon as the special cause of any functional

diseases of the nervous system. Even its influence as an indirect factor must be considered uncertain. It is possible, however, that many morbid conditions, both organic and nutritional, may be remotely related to the disease. The manner in which organic changes in the nervous system result from syphilis is not fully understood; in all probability many of them depend on the action of a toxalbumin, the result of the life processes of micro-organisms, which are with reason considered the agents of infection but about whose characters there is still no positive information.

Diphtheritic Infection.—By far the most frequent and important organic disease of the nervous system which depends on the action of the diphtheritic poison is the so-called diphtheritic paralysis. This paralysis, which is peculiar in distribution (p. 520), depends on a lesion inflammatory in character and limited mainly to the lower or spino-muscular segment of the motor path. In some cases both the gray matter of the cord and the peripheral nerves are involved, but in many instances it is probable that the lesion is confined to the peripheral nerves. It is certain that the peripheral nerves are especially liable to suffer from the effects of the diphtheritic poison. An important peculiarity of the diphtheritic affection is the liability of the vagus nerve to be affected. The relation between the local affection in the naso-pharynx and the paralysis is usually obtrusively clear, but it is essential to note that a considerable grade of paralysis may follow a local state so slight that its diphtheritic nature is overlooked. Probably twenty-five per cent. of the cases of diphtheria that occur are affected with some degree of diphtheritic paralysis. A rare result of diphtheritic infection is hemiplegia of sudden onset, due to one or more small hemorrhages into the motor path. Such hemor-

rhages may be so placed as to cause no important symptoms. A consequence of diphtheria which is of not very usual occurrence is hysteria. The hysterical symptoms may develop in association with the diphtheritic paralysis, or separately.

Other Forms of Infectious Disease.—Tubercular, syphilitic and diphtheritic infection are, as we have seen, exceedingly frequent causes of nervous disease. Various other infectious diseases are occasional causes of organic disease, and some of them are frequent causes of functional nervous disease. The pathology of some of these conditions is as yet but imperfectly understood. Among the acute infectious diseases there is a general correspondence in the clinical aspects of some of the diseases that are observed. Thus, in the course of any one of the acute infectious diseases, symptoms of sudden onset (hemiplegia, aphasia), such as indicate a vascular lesion of the brain, are liable to occur. In some cases the lesion is embolism, in others thrombosis of an artery or vein, in others still, hemorrhage. Embolism is especially common after scarlatina and acute rheumatism. These various lesions are a frequent cause of the acute cerebral palsies of childhood. Convulsions are common at the onset of these processes, especially in children, where they are often indicative of cerebral irritation only.

The other lesions dependent on infectious diseases present considerable diversity in character, and will be mentioned in connection with the diseases on which they depend.

Typhoid Fever.—Organic nervous disease is of much less frequent occurrence after typhoid fever than is functional disease. It much more often involves the cord or peripheral nerves than the brain. Paraplegia of rapid or subacute onset is occasionally seen during convalescence

from typhoid. It depends in some cases on slight myelitis, and in others on multiple neuritis.[1] The paralysis is seldom complete. In rare cases the paralysis corresponds to the type of acute ascending paralysis (p. 524). In other cases there is localized atrophic paralysis (generally subacute) from poliomyelitis. Sometimes there is local neuritis of spinal nerves, especially those of the extremities—the ulnar and peroneal being those most often involved. Sometimes there is paralysis of the palate, like that which follows diphtheria. Brain affections are more rare. They include acute meningitis and hemiplegia (very rare), paralysis of cranial nerves, multiple sclerosis, and optic neuritis.

The functional affections dependent on typhoid fever are, many of them, of common occurrence, and seem to be due to the influence of a diffusible poison (toxalbumin) which affects chiefly the brain. Thus there are observed bilateral deafness (very common), loss of speech, not true aphasia (common); mental failure, as shown by loss of memory, change in character, or imbecility; chronic mania; tremor; transient paraplegic weakness (very common and due perhaps to changes in the muscles); neuralgic pains, and various manifestations of hysteria or epilepsy in those predisposed to these diseases.

The relation of these various functional and organic conditions to the preceding typhoid fever is commonly clear enough. There is nothing sufficiently distinctive about any of the conditions that have been enumerated to enable one to refer them to typhoid fever in the absence of a clear history of this disease.

[1] There is but little doubt that the neuritis present after many cases of typhoid is due in no small measure to the indiscriminate administration of alcohol during the course of the disease.

Typhus Fever is only rarely the cause of organic nervous affections. It is occasionally complicated with acute meningitis, and sometimes followed by functional or organic paraplegia, as is the case with typhoid. Very often typhus is followed by optic neuritis, probably not dependent on meningitis. Functional disorders like those following typhoid are not rarely observed after typhus. There is nothing distinctive about any of the nervous sequelæ of typhus.

Scarlet Fever.—Cerebral affections are more common than others from scarlatina. The most frequent condition is hemiplegia, due in some cases to embolism from endocarditis, in others to thrombosis. Acute meningitis occurs with scarlatina, but is rare. Convulsions may occur during the disease, even when there is no evidence of hemiplegia, and may persist in after life as epilepsy. Rarely chorea may occur as a sequela to the disease. Optic neuritis may occur without symptoms of meningitis, and without evidence of nephritis. Acute poliomyelitis occasionally appears to depend on scarlatina.

Measles.—This disease is an occasional cause of hemiplegia (from thrombosis), and of acute meningitis. More often it is followed by spinal cord diseases, of which acute poliomyelitis and acute myelitis are the most important. In rare instances, measles has been succeeded by disseminated myelitis, or by acute ascending paralysis.

Small-pox.—Small-pox is a more frequent cause of acute meningitis than any other eruptive fever. Hemiplegia as the result of encephalitis may occur. It is also a more common cause of spinal cord disease. Myelitis, especially the disseminated form, is perhaps the most frequent nervous lesion. Poliomyelitis occasionally occurs, and sometimes there is multiple or localized degenerative neuritis. Acute ascending paralysis has been

observed. Small-pox has been succeeded by insular sclerosis. The ataxia of the arms and legs which is now and then observed probably depends on multiple neuritis.

Erysipelas.—This disease is very rarely followed by organic nervous disease, and some of the conditions ascribed to it have probably depended on other causes. Facial erysipelas may cause unilateral or bilateral paralysis of the facial nerve, and sometimes results in acute meningitis.

Acute Rheumatism.—The disease of the nervous system which most often results from acute rheumatism is acute cerebral softening from embolism, the result of endocarditis. Spinal cord lesions are rare. Myelitis, however, sometimes occurs, and poliomyelitis has been thought to be frequently a consequence of acute rheumatism, but its occurrence is probably rare. Multiple neuritis, with widespread symptoms, probably occurs in rare instances.

Pneumonia.—Pneumonia is a rare cause of nervous disease, but is capable of giving rise to various manifestations in different cases. The pathology and even the pathological anatomy of most of these morbid conditions is obscure. Nervous complications and sequelæ appear to be most often observed in cases of pneumonia that are distinctly infectious and occur in epidemics. This, as well as other facts, suggests their dependence on a micro-organism. It is probable that some of the nervous lesions enumerated depend on the presence of the Fraenkel-Weichselbaum bacillus, while others depend on the action of a diffusible poison produced by this micro-organism. Thus, purulent meningitis, especially of the convexity, occasionally complicates or succeeds pneumonia, and is probably due in all cases to the lodgment of Fraenkel's bacillus in the pia. Again, epidemic cerebro-spinal meningitis sometimes complicates pneumonia, and is probably

due in these cases to the action of a pneumococcus or bacillus.

Occasionally pneumonia is succeeded by symptoms that indicate a multiple neuritis. These symptoms doubtless depend on the action of a diffusible poison. The same may be said of the rare cases of acute ascending paralysis that follow pneumonia.

Sometimes nervous symptoms suggesting myelitis follow pneumonia; in other cases there are indications of multiple sclerosis. Ataxia of all four extremities has been the chief manifestation, due possibly to multiple neuritis. Local paralyses, with atrophy and altered sensibility, are occasionally seen. In some cases there have been twitching of muscles, rigidity of limbs, neck, and jaw muscles, and painful contractions.

Hemiplegia during pneumonia probably depends on thrombosis, determined often by the existence of atheroma. Occasionally sinus thrombosis may be caused by extreme exhaustion, as in other acute specific diseases.

Influenza—Grippe.—The epidemic disease known as influenza is a far more frequent cause than pneumonia of nervous disorders. These are, however, for the most part functional. It is rare for patients to recover from this disease without some functional nervous derangement. Most often there is a state of neurasthenia and prostration, sometimes slight, but usually well marked, and often persistent and profound. A certain degree of mental disturbance is commonly linked with this neurasthenic state; most often there is slight mental depression, and with this there may be hypochrondriasis. In those who have mental disease all the symptoms are exaggerated by the occurrence of an attack of influenza. Insanity is occasionally caused by influenza. The following nervous symptoms are met with singly, or in combi-

nation, during or after the grippe: frontal headache; dizziness; insomnia; neuro-muscular pains, especially in the back, calves, and thighs; neuralgic pains referred to the eyeballs; paræsthesiæ; loss of sexual power; attacks of syncope; tremor and unsteadiness in gait.

Organic disease appears to be rare as a sequel of influenzá, but cases have been reported of suppurative meningitis and of multiple neuritis. Spinal meningitis is probably sometimes a sequel of the disease. All these conditions, functional and organic, doubtless depend on a diffusible poison or on a pathogenic micro-organism.

In many cases of disease that unquestionably depend on infection, the source and character of the infection are unknown. Thus, many cases of poliomyelitis result from an unknown infection. The same may be said of many cases of infantile cerebral paralyses. There is reason to believe that both these conditions may sometimes depend on the same infection.

Toxic Influences.—We have already passed in review the chief forms of infectious disease that give rise to nervous disease. In so far as these forms of infectious disease produce their effects through the agency of a proteid substance derived from the activity of living micro-organisms, they are to be regarded as *toxic influences*. There remains for consideration, however, a class of toxic influences very different in character from those just mentioned—a class of unorganized poisons. This class includes a variety of organic and inorganic substances, many of which are used as drugs. Of these, the most important in the causation of organic nervous diseases are the heavy metals, lead, arsenic, mercury and silver, which produce their most decided effect on the nervous system when taken into the body in small quantities and during a considerable period of time. Many

other drugs, as quinine, opium, belladonna, strychnine, etc., produce nervous symptoms when taken in excessive doses, but are rarely productive of organic disease, and do not concern us here. Alcohol, which is a most important toxic agent, is considered elsewhere.

Lead.—The entrance of lead into the blood gives rise to a variety of nervous disorders. Chief among these are the clinical forms of disease that depend on the action of lead on the lower segment of the motor path. The principal lesion resulting from lead poisoning involves certain peripheral nerves, and especially the radial nerves. The toxic influence may, however, extend to that portion of the lower segment of the motor path which is located in the cord, and we may thus distinguish between the cases of lead paralysis that depend on a peripheral degeneration, and those that are due to a peripheral degeneration and a degeneration of the ganglion-cells and root-fibres from which the peripheral nerves affected spring. The typical symptoms of neuritis from lead are given elsewhere. (See multiple neuritis—lead paralysis.) A rare form of lead neuritis causes laryngeal palsy.

In a small number of cases lead poisoning gives rise to cerebral symptoms. In some of these cases there are evidences of organic change in the membranes of the brain; in other cases there are no alterations, and the symptoms appear to be due to the direct effect of the poison on the nerve elements, which undergo nutritional changes that fall short of structural alteration. Among the symptoms that arise in this way are delirium, coma, convulsions, and, occasionally, hemiplegic weakness and hemianæsthesia. Optic neuritis is an occasional but not rare consequence of lead poisoning. It is probably one expression of the direct action of lead in the blood on the peripheral nervous system, but it may sometimes depend on descend-

ing inflammation from the meninges. Sometimes there are the symptoms of an axial neuritis—that is, scotomata.

Less acute cerebral symptoms may be due to lead. Thus, general epileptiform seizures may occur at intervals for a long period after lead has ceased to enter the system. Sometimes there is general mental failure, associated with general muscular weakness. There may be melancholia, with delusions, and in some cases there are symptoms that resemble those of general paralysis of the insane.

Severe abdominal pain ("lead colic") with constipation are common occurrences when the lead has entered the system through the intestines. Pains in the limbs referred to the muscles or joints are common and may occur alone or in connection with paralytic symptoms. The pains sometimes suggest a neuritic process. Tremor is an occasional occurrence. It varies considerably in character in different cases and may be associated with paralysis or occur alone.

An important effect of lead is that upon general nutrition. A considerable degree of true anæmia is often rapidly induced by the entrance of the poison into the circulation. There is also a diminution of uric acid in the urine and an increased liability to the development of gout. With the liability to the development of gout there is the strong liability to the occurrence of kidney disease observed in gouty subjects (chronic diffuse nephritis, granular form). Indeed, lead poisoning may produce kidney disease without inducing gout. By its coincident effect on the heart and vessels lead-poisoning may become a cause of cerebral hemorrhage—a fact of considerable practical importance.

The extensive use of lead in the industrial arts makes certain artisans particularly liable to lead poisoning.

Plumbers, painters, compositors, and type-founders suffer most often. Painters constitute a large proportion of cases of lead poisoning. The practice of "flatting," or working with lead and turpentine within doors, is particularly detrimental. Those who grind flint glass and glaze pottery sometimes suffer. In many cases lead enters the system through what may be called accidental sources, especially through drinking-water that has been contaminated by lead pipes or cisterns lined with lead. An important source of poisoning is from beer which has stood in lead-lined receptacles. Snuff wrapped in lead paper has been contaminated, shot for cleaning demijohns has been dissolved in wine, etc., etc. Lead poisoning has resulted from the use of lead-containing cosmetics and hair-dyes, and rarely from medicinal preparations, as "Goulard's Extract." It is hardly necessary to insist on the practical importance in diagnosis of a knowledge of the possible sources of poisoning when symptoms are observed which may possibly be due to lead.

The way by which lead most frequently enters the system is the alimentary canal, but it may pass through the skin, by way of the nose (as in poisoning from snuff containing lead) and probably by the lungs. It has been thought that when lead enters the system by the skin it may cause purely local effects (local saturnism) by purely local action. It is probable that local anæsthesias are sometimes so produced, but there is no evidence that there is ever muscular paralysis from this cause.

The descendants of those who have had lead poisoning (like the descendants of alcoholics) are especially liable to develop functional nervous disease (migraine, epilepsy, neuralgia, neurasthenia, chorea, tic convulsif, etc.).

Arsenic.—The nervous symptoms produced by arsenical poisoning bear a close resemblance to those resulting

from alcoholic excess, and are referable, as in alcoholic poisoning, to a multiple neuritis, or, more rarely, to myelitis. As in neuritis from alcohol, there are two chief types of arsenical neuritis,—that in which there is paralysis of the extremities, and especially of the extensors of the hands and feet, and that in which the chief symptom is general ataxia, associated with diminished sensibility. A special variety of neuritis, namely that resulting in herpes zoster, is occasionally a result of arsenical poisoning.

Unlike poisoning by lead, arsenical poisoning is rarely dependent on occupation, but results, as a rule, from the use of pigments containing arsenic. It may thus occur from the arsenic contained in wall-papers, in book leaves, in artificial flowers, and in various paints, and enters the system by the lungs. Sometimes it results from the medicinal administration of arsenic in some form, particularly in chorea of children. The quantity required to produce toxic effects varies much in different individuals. Arsenical neuritis may result from the entrance of small quantities of the metal for long periods of time, or as a sequel of acute poisoning, or it may follow the acute symptoms that sometimes result from the slow accumulation of the metal in the system.

Mercury.—The nervous symptoms that result from mercurial poisoning appear to be chiefly functional in character and of cerebral origin. The most important symptom of mercurial poisoning is a tremor, beginning in the face and tongue, and extending, in time, to the arms and legs,—"mercurial tremor," or, in vulgar parlance, the "trembles." After a time the tremor (*vide* page 88) may be associated with some loss of muscular power. Other derangements may follow mercurial poisoning. Of these the chief are as follows: neuralgias (especially facial), formication, local partial analgesias, mental irrita-

bility and slight mental failure, insomnia, hallucination headache, irregular heart action, maniacal excitement. In rare cases there is aphasia and transient hemiplegic weakness. Mercurial poisoning is met with chiefly in those who work in the metal. It is thus often observed in quicksilver miners, in those who make certain physical instruments (barometers, thermometers), and in those who silver mirrors. Less often it is seen in furriers, hatters, in bronzers, etc. The metal generally enters the system by the lungs, for it is somewhat volatile even at ordinary temperatures. Rarely it enters by the skin or by the alimentary canal. Those who are in depressed health are much more susceptible than those who are robust. It is an important practical point that the symptoms of mercurial poisoning may not appear until some time after the metal has ceased to be absorbed.

Silver.—Poisoning by silver has been rare since the practice of treating certain nervous diseases with salts of silver has fallen into merited disuse. It almost never results except from the medicinal use of the metal. The chief symptom of poisoning is a bluish discoloration of the skin due to the local deposit of silver. There is often a black line on the gums, resembling the "lead-line." In very rare instances there has been paralysis of the limbs (especially the extensors), dependent, no doubt, on neuritis. The discoloration of the skin is said to begin when about an ounce of the nitrate of silver has been administered,—no matter during what period of time or in what doses.

It is an important fact that small doses of a poison which by themselves would give rise to no toxic effects, are capable of producing toxic symptoms if these doses act in the presence of another toxic influence. Thus it has been observed that those who work in the heavy

metals (especially lead, arsenic, mercury, and silver) are liable to develop typical alcoholic neuritis from very moderate indulgence in alcohol.

Trauma.—Traumatic influences play an exceedingly important rôle in the production both of organic and functional nervous disease. In most of the functional diseases related to trauma, the influence of injury is accessory and exciting, and operates on a predisposition. In many forms of organic disease injury is the sole factor. The character of the injury varies as greatly in different cases as does the resulting form of damage. The forms of injury most often encountered are blows and falls upon the bony structures enclosing the cerebro-spinal axis, wounds or contusions or pressure of nerve-trunks and general concussion. Upon the nervous structures these traumatic influences operate in various ways : by direct pressure on nerve elements, by pressure from displaced bone, by pressure from hemorrhage, by direct or indirect destruction of nerve tissues, by causing large or punctate hemorrhages in or external to the nervous organs, by causing changes in the nutrition of vessels and later in nervous elements, by exciting simple inflammation, by affording entrance to pathogenic micro-organisms, and by causing severe mental shock. Some injuries act by several of these mechanisms.

Some injuries give rise to immediate, or almost immediate, symptoms ; others produce nervous symptoms only after the lapse of time (days, weeks, or months). Of the conditions that are the immediate consequence of trauma, some are organic, others functional. The chief organic states that are produced immediately through trauma are as follows : extra-dural hemorrhage (from middle meningeal artery), with hemiplegia, hemispasm, ptosis, etc., generally but not always the conse-

quence of fracture of the skull; subdural or subarachnoid hemorrhage, with or without fracture; laceration of brain substance, with or without fracture (often opposite seat of blow); punctate hemorrhages in the subcortical white substance (generally from severe concussion); subdural spinal hemorrhage, with or without fracture; hematomyelia; complete or partial crush of cord or cauda equina, from fracture-dislocation; peripheral nerve paralysis from pressure, laceration, contusion, or section.

The functional disturbances of the nervous system immediately consequent upon trauma are for the most part what may be called indirect effects. Thus, when an hysterical paralysis is the immediate result of a slight injury to the part paralyzed, the trauma operates through the medium of the cerebrum, and not on the part simply, although the injury determines, at least in a measure, the part involved in the paralysis. In most of the cases of this character there exists a predisposition (neurotic heredity or state) without which the trauma would have been quite inoperative. Moreover, there is good reason to believe that in many cases of functional paralysis the symptoms are dependent largely on the influence upon the brain of accompanying emotion. Hence, the precise rôle of trauma varies considerably in these cases and is not always easy to define.

In a smaller class of cases the influence of injury is direct, as when sciatica follows directly upon pressure from the edge of a seat. But here again there is often a predisposition without which the slight trauma would not have become operative. Some cases of this kind are, moreover, organic, but there is no doubt that many of them are dependent on nutritional changes solely.

The remote consequences of injury—that is, those which appear after the lapse of days, weeks, or months,

are very numerous, and arise in a variety of ways. We may enumerate first the more important organic states that belong to this category. It is proper to include here the large class of inflammatory lesions that depend on injury: meningitis, cerebral or spinal, cerebritis, myelitis, abscess, neuritis, arteritis, etc. The development of new growths is influenced by trauma, but some forms of tumor (using this word in its general, not in its strictly pathological sense), as glioma and tubercle, are more often stimulated to activity through injury than the others. Cerebral tumors are more often related to injury than those which are spinal or neural. As a class the degenerative lesions do not appear to be greatly influenced by trauma, but some forms, as tabes, are not rarely a sequel of injury. We may inquire a little more carefully into the nature of the relation between these various classes of lesions and injuries.

Trauma appears to give rise to inflammatory conditions of the nervous system in several ways. First, the injury may act on the nerve elements in such a way as to cause their partial or entire destruction, with consequent inflammatory infiltration into and about the damaged parts. In this way there may be set up a pachymeningitis, or a neuritis, or a variety of other conditions. By this mechanism injury rarely gives rise to a suppurative process, but there is little doubt that it thus causes some cases of acute myelitis, and some of cerebral abscess. It is possible that, in these cases, it produces its effects by the mechanism of severe concussion, which may cause extensive interstital laceration, followed by the local accumulation of immense numbers of small spheroidal cells. Whether or not in such cases the lodgment of micro-organisms is what actually determines the occurrence of suppuration after such

lacerations, has never been proven, but it is in accordance with the concepts of general pathology to believe that supupration here, as elsewhere, depends on the presence of micro-organisms. Without their presence it should seem that even the most severe injury would be followed by an effort at repair. But the most important mechanism by which injury leads to inflammation is of a very different character from those mentioned, and certainly depends on the invasion of micro-organisms. Injury in these cases affords to certain pathogenic organisms an ingress to the membranes, or to the nervous substance itself, or to peripheral nerves. Thus after fracture of the base of the skull there may be a communication between the membranes and the Eustachian tube by the tympanum, through which the pathogenic organisms that reside in the mouth (pneumococcus, etc.) may pass and excite suppurative inflammation. Cerebral abscess is often caused by injuries to the head, which open the way to pyogenic micro-organisms. Sometimes injury acts by giving rise to bone disease, which in turn offers the opportunities for the development of inflammatory processes. This is the case with otitis following injury.

In its relations to new growths in the nervous system, trauma appears to act merely as an excitant, and certain abnormal conditions must have pre-existed, in a given case, to make trauma operative, probably by inducing changes both in the structure and nutrition of the parts, favorable to the development of the variety of new growth in question. In no other way is it possible to explain the influence of injury in causing the development of infective granulomata (syphiloma, solitary tubercle) within the nervous system.

The precise relations between injury and degenerative disease of the nervous system are but imperfectly under-

stood. In some instances there is a predisposition to such disease, as shown by heredity or acquired conditions, such as syphilis ; in other cases, as in tabes, no predisposition may be recognizable.

An important class of remote effects of injury is that which includes the conditions known as "traumatic neuroses" (p. 538). The injuries in these cases vary in character, but are often such as would ordinarily be deemed insignificant. There is, however, almost invariably present at the time of injury, and consequent upon it, a considerable degree of mental shock, which doubtless plays an important rôle in the production of the symptoms. The traumatic neuroses are commonly regarded as dependent on functional cerebral derangements, but it is probable that in some of the cases, at least, the symptoms have been due to slight but distinct organic changes.

Injury to the head is a frequent cause of epilepsy. Very often in such cases it causes local changes in the pia and dura.

One of the remote effects of injury (wounds) to which it is necessary merely to allude here, is tetanus. This condition, like rabies, is dependent on the infection of a wound, and is considered elsewhere. Sometimes an injury to the head appears to determine the locality in which convulsions, caused by a general condition such as uræmia, repeatedly take their origin. The influence of trauma upon certain parts of the nervous system is distinctly increased by certain pre-existing toxic influences. Thus, pressure upon peripheral nerves is more effective in causing paralysis in those who drink habitually to excess, or have been poisoned by a metallic poison, or are recovering from an infectious disease, than in those who have not been so exposed. The same

deleterious influences render persons more liable to the development of the traumatic neuroses from slight injuries. It has also been noticed that alcoholics and convalescents from infectious diseases are liable to develop the symptoms of severe concussion of the brain in consequence of slight head injuries.

Reflex Influences.—The influence of reflex irritation in causing nervous disease has been variously estimated. It has been thought that many organic diseases may at times depend on reflex irritation. Thus poliomyelitis has been considered a consequence of dental irritation in children, and myelitis of slight injury to a peripheral nerve or of irritation from phimosis. There is no evidence that organic disease ever depends on such reflex influences, and their effect in causing functional disturbances has certainly been much exaggerated. In almost all cases in which peripheral irritation appears to cause functional diseases it acts merely as an excitant, operating on a recognizable predisposition, hereditary or acquired. Thus, when attacks of migraine are determined by strain of the ocular muscles, the underlying cause will be found in disturbances of nutrition and assimilation. In a similar manner ocular strain may result in chorea or epilepsy, but it is probable that some conditions, as transient functional headache, may depend solely on such strain. Of the very numerous states that have been attributed to genital irritation (paraplegia, hemiplegia, epilepsy, chorea, insanity, etc.), none has been due solely to this cause. The same may be said of the effects of scars, wounds, intestinal worms, conditions of the ear, nasal defects, uterine disease, etc. It is probable that the symptom vertigo is often due largely or solely to the misleading sensory equilibrating influences which arise from the disturbance of certain special

sense-organs (especially those of the internal ear), but this case is exceptional and does not belong strictly in the category of which we are speaking.

In every case of disease in which the question of causation by reflex irritation arises, such an origin should not be admitted until all the more intelligible causal influences have been excluded.

Increased Atmospheric Pressure gives rise to one important disease of the nervous system—namely, caisson paralysis (see p. 418). The relation of this influence to the paralysis in question is usually obtrusively evident.

Exposure to Cold.—The influence of no etiological factor has been so overrated as that of exposure to cold. Scarcely a form of organic nervous disease exists which has not at some time been regarded as dependent on cold. Yet there is no evidence that exposure to cold ever acts except as an excitant, and there is good reason to think that the conditions in which it acts in this way are usually due to some form of infection. Thus, though poliomyelitis has been considered due to cold, it bears the stamp of an infectious disease, and it is difficult to see how cold can do more than determine, at times, the occasion of its development. The "rheumatic" ophthalmoplegia of bygone days has been shown to be due almost invariably to syphilis. The most common form of facial paralysis is the so-called "rheumatic" form, which develops after exposure, often slight, to cold. Even in this condition it is possible that infection is the actual cause of the inflammation, and cold merely the excitant. And so it is with many other states,—myelitis, meningitis, neuritis, etc. Belief in this dependence on cold has been gradually shaken by the discovery of more intelligible causes. Many cases of musculospinal paralysis were once considered "rheumatic"; they are now known to depend on pressure.

When cold acts as an excitant of disease it is probably by lowering the vitality of the parts concerned. The locus minoris resistentiæ may then become open to influences which were before innocuous. Such a lessening of vitality, general or local, would explain the increase in the gravity of symptoms of organic disease which sometimes occurs after an exposure to cold.

Exposure to Heat.—Prolonged exposure to heat, in addition to giving rise to heat-exhaustion or heat-stroke, appears capable of determining the development of inflammatory states of the central nervous system. Thus, subacute poliomyelitis has developed in adults from unusual exposure to heat. The exposure may be to the rays of the sun or to the heat of an engine-room or kitchen. Sunstroke has been followed by acute meningitis. It seems to be capable also of inducing the symptoms of chronic meningitis. Sunstroke has been followed by symptoms resembling closely those of general paralysis of the insane. Less grave conditions, such as neuralgias, irritable heart and various neurasthenic symptoms, are a common sequel of insolation.

Over-Fatigue from Excessive Exercise.—Over-fatigue appears to act as a cause of nervous diseases chiefly through its temporary debilitating effects. In this manner it often determines attacks of neuralgia or of migraine where a predisposition exists. Rarely it appears to determine the development of paralysis agitans. Some cases of locomotor ataxia, and others of acute internal spinal meningitis, appear to have been related in their development to over-fatigue.

The Excessive Use of Particular Muscles, or groups of muscles, is a much more important and definite cause of nervous disease than general over-fatigue. An important class of functional disorders, the occupation neuroses,

depends on the habitual excessive use of particular groups of muscles. But it is doubtful whether even great excesses of this nature are competent to bring on these neuroses in the absence of a central (cerebral) state of instability, shown not rarely by a hereditary tendency to functional nervous disorders.

Temporary paralysis of a part may result from excessive use, and probably depends on temporary nutritional changes in the anterior cornual cells of the spinal cord. Thus, cases are observed of paralysis of the lower extremities from excessive walking. Violent and excessive use of muscles may lead to more serious conditions. Thus, spinal meningeal hemorrhage may occur at the level of the centres concerned in the violent muscular contractions, and, more rarely, hematomyelia results in the same way. In those who have miliary aneurisms, cerebral hemorrhage sometimes follows a violent muscular effort, such as occurs in lifting, straining at stool, vomiting and coughing. Excessive use of the muscles of the legs appears to have determined the occurrence of some cases of acute myelitis, and others of acute poliomyelitis. Occasionally the symptoms of locomotor ataxia date from the excessive use of the arms and legs, but it is probable that in these cases important predisposing conditions have been present.

An important fact in its diagnostic bearings is the occurrence of muscular atrophy from the excessive use of particular muscles. In engravers, and those who do the work of "illuminating," atrophy of the thenar eminence or of the first dorsal interosseus (abductor indicis) is sometimes observed. The degree of atrophy may be considerable, but remains limited to the muscles over-used. Such atrophy probably never leads to progressive muscular atrophy.

Mental Shock—Fright.—Of the forms of mental shock that influence the development of nervous disease, the emotion of fright is the most potent. Almost any form of nutritional disease of the nervous system may follow a severe fright, but especially distinct is the relation between fright and chorea, epilepsy, and paralysis agitans. When excited by fright the conditions named follow almost immediately on the event. Violent neuralgia has developed from fright and also from grief.

It is a most noteworthy fact that violent fright appears competent in rare cases to excite the development of certain forms of organic disease of the brain in those who have appeared, and probably have been, quite free from nervous disease. Thus, organic vascular changes have been found not only in the brain, but in the cord, in cases of the so-called "traumatic neurosis" (p. 538). The influence of violent emotion, especially anger, in determining the occurrence of cerebral hemorrhage is well known. Occasionally emotion determines the occurrence of cerebral embolism in the subjects of heart disease.

Influence of Abnormal Conditions Affecting the General Health—Cardiac Disease.—Organic disease of the heart is of importance chiefly in its relation to one process—acute cerebral softening from embolism. The presence of valvular disease of the heart in a person presenting the symptoms of acute cerebral softening (p. 553) is most important evidence of the nature of the lesion, provided the other causes of acute softening can be excluded. A considerable proportion of cases of chorea are preceded by valvular disease of the heart, but the nature of the relation between these conditions is unknown.

Feeble heart-action is a powerful cause of thrombosis of cerebral vessels, and many of the general causes of thrombosis (p. 265) probably operate chiefly through the

cardiac depression they cause. Overaction of the heart favors the occurrence of cerebral hemorrhage in those who have miliary aneurisms of the cerebral vessels.

Disease of the Vessels.—Of the forms of arterial disease that bear an important etiological relation to disease of the nervous system, only one form—atheroma—is recognizable during life. Indications of possible degenerative disease of the cerebral vessels is afforded by the presence of atheroma of the accessible arteries (radial, temporal, etc.) and by atheroma of the retinal vessels (recognizable by means of the ophthalmoscope). Atheroma stands in causal relationship to two conditions which, though very different pathologically, are nearly allied in their clinical features—cerebral hemorrhage and acute softening from thrombosis.

Kidney Disease.—Chronic disease of the kidney, primary or secondary to heart disease and associated with general arterio-sclerosis (granular kidney), is an important cause both of cerebral hemorrhage and of acute softening from the thrombosis which occurs in atheroma of the cerebral vessels. The presence, therefore, of kidney disease, does not materially aid us in the distinction of these two forms of disease.

Among the nervous conditions of importance that are met with as a consequence of uræmia, are headache, insomnia, stupor and convulsions. The convulsions are commonly general, but it is an important fact that they may have a strictly local commencement, and may be limited to one side of the body ; that is, they sometimes have the character of the convulsions so suggestive of a focal brain lesion. Optic neuritis and neuro-retinitis are not rarely observed in the course of chronic kidney disease.

Disorders of Digestion and Assimilation are frequent and potent causes of functional nervous derangements, such

as headache, neurasthenia, mental depression, insomnia, etc. Much remains to be learned respecting the exact nature of these nutritional disturbances, and their relations to special conditions and symptoms. Organic disease of the nervous system probably never results directly from such disorders of digestion and assimilation.

Diabetes.—Diabetes is often the cause of continued and severe headache. A more important consequence, from a diagnostic standpoint, are the convulsive seizures that are occasionally observed in the course of the disease. These are ordinarily general, but may, like the seizures of uræmia, be of local commencement. In rare instances diabetes gives rise to a multiple neuritis which may bear some resemblance in its symptoms to locomotor ataxia.

Rickets, in young children, is not rarely associated with hydrocephalus. Rickety children often show retardation in the development of speech. The muscular weakness which is present in these cases leads to late standing and walking, and may, in extreme cases, be mistaken for paralysis. It is not very rare to see brief attacks of loss of consciousness, with peculiar reflex contraction of the limbs, in rickety young children.

Morbid Blood-States.—Certain morbid conditions of the blood, such as occur in acute diseases, in the terminal period of cancer and tuberculosis, in gout, occasionally in chlorosis, and in various conditions of malnutrition, favor coagulation of the blood within the vessels, and thus become an important cause of thrombosis of cerebral vessels. The influence of these various blood-states in causing thrombosis is greatest in the old and in the young, and in those who have arterial disease. The thrombi of typhoid fever are due to agglutination rather than coagulation. The morbid blood-state in purpura

hemorrhagica may lead to punctate sub-cortical hemorrhages, but these rarely give rise to symptoms.

Pregnancy.—The blood-state in the latter part of pregnancy is such as favors coagulation of the blood within the cerebral vessels, and hence it is that, soon after delivery, the symptoms of thrombosis of a cerebral artery or vein are sometimes observed (hemiplegia of sudden onset, hemianopsia, aphasia, etc). Exertion soon after delivery seems to favor the occurrence of this condition. Of the functional states that may arise from pregnancy, the chief are chorea, which usually develops early (third month), and certain psychoses (mania, melancholia).

Influence of Treatment by Drugs.—The only form of treatment that may afford an important indication of the nature of an organic disease of the nervous system is that known as antisyphilitic. If, under the influence of antisyphilitic treatment (mercury, potassium iodide, singly or together), there is a rapid and considerable subsidence of the symptoms due to a lesion such as may have been caused by syphilis (*vide* page 300), there is a considerable probability that the lesion was syphilitic in character, and this probability may amount to a practical certainty. If, on the other hand, the symptoms do not show considerable improvement after the administration of maximum doses of iodide and mercury for a considerable period of time, there is a considerable probability that the lesion is not a true syphilitic lesion. It is very important to note, in this connection, that a lesion may be of syphilitic origin and yet not be, strictly speaking, a syphilitic process. For example, acute cerebral softening occurs from syphilitic endarteritis, *but there is nothing syphilitic about the necrotic process.* The softened area has the same pathological characters as soft-

ened areas from any other cause of acute softening (atheroma, embolism). The fact, therefore, that the softening was due to syphilitic disease of an artery, renders the necrotic area no more amenable to antisyphilitic treatment than if it had been due to embolism or thrombosis from atheroma.

Heredity.—Conditions of abnormal heredity underlie many morbid states of the nervous system. In the case of some of these morbid states the relation to heredity is uncertain; in others it is more definite, but, in all, the ultimate nature of the relation is but imperfectly understood. For practical purposes it is convenient to consider separately the influence of heredity on mental diseases, on neuroses and on organic diseases.

A neurotic heredity (shown by a disposition to functional nervous disease in the ancestors) may be traced in the majority of all cases of insanity but not in all. The graver chronic forms of insanity, especially paranoia, and allied conditions, always develop on an hereditary degenerative basis, but the simple psychoses, mania and melancholia, may develop in persons without known hereditary taint. Very often the heredity of insanity is dissimilar (heterologous)—that is, dissimilar types of disease develop in the progenitors and the offspring; less frequently the type is the same, as when paranoiac progenitors have paranoiac offspring or descendants. The milder forms of mental disease often show merely a general neurotic heredity. Usually the heredity is direct—that is, derived from parents and grandparents; less often it comes from the collateral ancestors (collateral heredity).

Among the neuroses direct heredity is the rule and similar heredity is common. Thus, in about two thirds of the cases of epilepsy in which an inherited tendency

to the disease can be traced, there has been a family history of epilepsy. In the remaining third, the inherited tendency was derived from some form of insanity. But a direct inherited tendency is shown in only about one third of all cases of epilepsy. In more than half the cases of migraine a morbid nervous heredity can be shown. As a general rule the inheritance is direct and similar—that is, the disease is derived from a parent who has migraine. In hysteria the influence of heredity is much less strong than in the case either of epilepsy or of migraine ; still it is considerable. The hereditary influence is usually direct and may be similar or dissimilar. Hereditary influence is traceable in a small proportion of cases (one sixth) but is very marked in some of them. The heredity is usually direct and is often similar, but may be dissimilar (insanity, epilepsy). In Huntington's chorea the heredity is direct and similar ; but it is doubtful whether this disease belongs in the category of neuroses. In paralysis agitans, an hereditary tendency, usually dissimilar, and either direct or indirect, may be traced in a small proportion (about one sixth) of cases. In exophthalmic goitre, in infantile eclampsia (non-symptomatic), in tetany, spasm of the glottis and writers' cramp, a neuropathic heredity can in many instances be traced. But here, as in the case of the other neuroses, and the psychoses, the facts of heredity do not afford us material aid in diagnosis.

Heredity does not appear to play a very extensive rôle in the production of organic disease of the nervous system. Yet, in some cases, its effects are strikingly evident. An important class of affections, the primary myopathies (p. 453), show usually a direct and similar inheritance,—the transmission being most often through the mother. Often this influence extends through sev-

eral generations. This is also the case with Thomsen's disease. In pseudo-hypertrophic paralysis there is often a direct and similar heredity, but many isolated cases occur and the inheritance may be collateral. It is, however, almost invariably through the mother.

Most of the other organic diseases related to a morbid heredity belong to the degenerative type of nervous diseases. It is certain that some of these conditions, as locomotor ataxia and general paralysis, may depend on hereditary syphilis, and it is not unlikely that some other degenerative diseases, as Friedreich's disease, may be, in some instances at least, the consequence of syphilitic taint in a parent—though at present we have no evidence that this is so. The one organic disease of the brain, general paralysis, which shows a hereditary influence, appears to be related to some form of insanity, or some neurosis (epilepsy) in the parents, in about 37 % of all cases. The hereditary influence is probably never direct and similar—that is, general paralysis in the parent probably never leads to general paralysis in the offspring.

A considerable number of degenerative diseases (especially those of the cord) seem to depend on the early or late appearance of morbid structural characters, the basis of which is congenital or embryonic. This is true of syringomyelia, hereditary ataxia, some cases of lateral sclerosis and cases of arrested cerebral development with mental defect, imperfect power, inco-ordination and increased knee-jerks. The character of the morbid influences in progenitors that lead to these congenital states is quite unknown, but the possible bearing of diathetic conditions, gout, diabetes, syphilis, tuberculosis and chronic alcoholism, should be borne in mind.

With the evident lack of accurate knowledge respect-

ing the nature of hereditary influences in the production of nervous disease, and the consequent inability to make generalizations, it is not strange that the diagnostic indications derived from heredity should be, as a class, unimportant. The only exception to this is in the case of the myopathies and Thomsen's disease, in which the usual family history is of great aid in fixing the nature of the affection.

III. The Position of the Morbid Process, taken in connection with the special and known liability of certain parts of the nervous system to particular forms of disease, is sometimes of considerable aid in the diagnosis of the nature of the lesion. In the case of the brain and spinal cord, where these indications are chiefly useful, they should be employed in subordination to the indications derived from the mode of development and the causal factors that have just been reviewed. The most important facts to be borne in mind in this connection are as follows :

In the spinal cord an acute focal lesion of the white substance is certainly inflammatory. A focal lesion of the gray matter is either hemorrhage or inflammation. Lesions which affect definite systems of fibres in the cord are commonly degenerative in character—unless they are of acute or subacute onset, when they may be focal and consist of disseminated patches of inflammation. An irritative lesion of the nerve-roots, if of gradual development and followed by symptoms of compression of nerve-roots, indicates compression of the cord.

In the brain a vascular lesion of the cortex or subcortical region is usually acute softening, seldom hemorrhage. A vascular lesion of the pons or of the medulla, which does not cause death within a few hours, is much

more likely to be acute softening than hemorrhage. Obstruction of the basilar artery is, in the absence of definite indications (as heart disease or a history of syphilis), much more likely to be due to syphilitic thrombosis than to embolic occlusion. This would be true, also, if equivocal indications were present—such as valvular disease and a history of syphilis.

A lesion supposed to be tumor is likely to be a sarcoma if it begins outside the brain substance. A tumor of the cerebral cortex is probably either a glioma or a syphiloma. A tumor of the cerebellum or pons is probably a solitary tubercle or a glioma. A tumor of the crus is probably syphilitic. At the base of the brain a tumor is likely to be a syphiloma, but may be a sarcoma. A basal tumor, situated in the position of a large artery, may be thought to be an aneurism, provided the patient is in the second half of life, has heart disease, and has had syphilis, and whose cerebral symptoms are not influenced by vigorous antisyphilitic treatment.

CHAPTER V.

THE DIAGNOSIS OF CLINICAL TYPES.

THE first step in the diagnosis of any case of nervous disease is to determine, at least provisionally, whether it be of organic or functional origin, and ordinarily this is readily done (see Chapter VI.). If the case appears to be one of organic disease, we endeavor to determine, again provisionally, whether the morbid process affects chiefly the brain, the spinal cord, or the peripheral nerves. The value of such a rough diagnosis, when it is possible, is that it gives direction to subsequent efforts to make a more exact anatomical and pathological diagnosis.

In most instances a distinction between disease of the brain on the one hand, and disease of the spinal cord or peripheral nerves on the other, is a simple matter. The distinction between spinal cord disease and peripheral nerve disease is usually easy, sometimes difficult, and occasionally impossible. The rough separation of organic disease above recommended into that of the brain, cord and nerves is much facilitated by bearing in mind the general characters of disease in these situations. These characters are described in detail elsewhere, but they may be advantageously enumerated here.

The general characters of brain[1] disease are as follows :

[1] Brain is used to include hemispheres, mid-brain, cerebellum, pons and medulla.

1. Motor, sensory, and trophic symptoms are usually unilateral in their distribution. The type of motor paralysis is hemiplegic.

2. Sensory symptoms are usually absent or slight, and involve the distal portion of the extremities only. The sensory changes are segmental, glove, sleeve, or circular in type.

3. Trophic symptoms are slight or absent. When present they are due to non-development rather than atrophy. Vaso-motor symptoms are common and are usually unilateral.

4. Mental symptoms are often prominent, and are rarely entirely absent. Loss of consciousness, convulsions, and disorders of motility accompany or follow cerebral lesion. All disorders of motility, choreiform movements, coarse and fine tremors, etc., cease during sleep.

5. In functional brain affections the disorders of movements are of a unilateral character, as the movements of the parts of one side are more closely related than are the parts of the upper or lower extremities of both sides. The finer muscle movements are first and most affected. Complex and delicate movements are first and most impaired. In convulsive and paralytic diseases of the brain, the arms are most frequently affected. Convulsions originating in the cerebral cortex are clonic in character.

6. The paralyzed muscles never present RD.

7. The knee-jerks are increased on the paralyzed side. Sometimes they are temporarily lost (at onset of acute lesions).

8. The sphincters are almost never paralyzed, but there may be incontinence from mental apathy.[1]

[1] Occasionally the sphincter ani is quite relaxed, as it is in disease of the lumbar enlargement, in cases of sudden and complete hemiplegia. The explanation of this is difficult. Possibly it depends,

The general characters of spinal cord diseases are as follows:

1. Motor and sensory and trophic symptoms are usually bilateral and often symmetrical in their distribution, as analogous parts of the upper or lower extremities of the two sides are closely related in the cord. The type of motor paralysis is usually paraplegic. Not rarely, however, a small cord lesion produces strictly unilateral symptoms, and then groups of muscles which subserve definite acts are involved.

2. Sensory symptoms, *if present*, are usually marked (anæsthesia, analgesia, etc.).

3. Muscular atrophy and other trophic symptoms, *if present*, are usually marked.

4. Atrophied muscles present the RD.

5. The sphincters are usually involved.

6. Mental symptoms are absent.

7. The muscle reflexes may be increased or absent or (occasionally) normal.

The general characters of disease of the peripheral nerves may be enumerated as follows:

1. The distribution of the motor symptoms corresponds not to *segments* of the body, as in brain disease, nor to particular movements, as is the case in spinal cord disease, but to the distribution of particular nerves. Single muscles, or groups of muscles, corresponding to nerve distribution are involved.[1]

like the associated loss of knee-jerk in such cases, on irritative inhibition. Similar relaxation of the sphincter is sometimes observed in crush of the cervical region of the spinal cord.

[1] Occasionally a limb muscle is paralyzed for one kind of movement and not for another. Thus the pectoralis major may be so affected that it can depress and adduct, but can no longer elevate the arm. Such paralysis of one movement only of a muscle probably

2. Sensory symptoms, especially pain, are prominent.

3. Muscular atrophy, if present, is usually considerable.

4. The atrophied muscles usually present an incomplete RD.

5. The sphincters are seldom involved.

There are exceptions, and sometimes important exceptions, to all the rules formulated above. Nevertheless they hold good in a large proportion of cases, and are therefore useful.

Clinical Types of Brain Diseases.

Meningitis.—The symptoms that indicate the existence of *acute meningitis* are headache, vomiting, fever and delirium, all of them of rapid though not of sudden onset. Headache is usually severe, but it varies in intensity at different periods of the disease and may be absent at the beginning. It is ordinarily the most conspicuous and persistent symptom of meningitis. The vomiting of meningitis is often without gastric cause, and may be persistent. It is usually an early symptom and is often absent late in the disease. The absence of any *one* of the four symptoms enumerated—headache, vomiting, fever, delirium—has little negative significance. The mere combination of delirium and fever does *not* suggest meningitis. This is an important point, the neglect of which leads to many errors in diagnosis. Besides these four cardinal symptoms there are other symptoms of less constant occurrence, the presence of one or more of which adds to the certainty of the diagnosis. These are, rigidity or weakness (often slight) of

always depends on disease of the ganglion-cells of the cord or the anterior nerve-roots, never on disease of the peripheral nerves. Unfortunately this point of distinction is only rarely available.

one or more of the extremities, rigidity of the muscles of the back of the neck, paralysis (often slight) of cranial nerves, marked inequality or great contraction of the pupils, strabismus, convulsions (local or general), a slight or considerable degree of optic neuritis and irregular respiration.[1]

The existence of meningitis having been established, or appearing highly probable, there remains to determine, first, the situation of the meningitis, whether of the base or the convexity, and secondly, the nature of the inflammation, whether tubercular, purulent, or simple.

The symptoms which suggest *Meningitis of the Base* are the early involvement (sometimes extensive) of the cranial nerves (strabismus, ptosis, inequality of pupils, etc.), repeated and early vomiting, the relatively late occurrence of delirium, the early appearance of optic neuritis, irregular breathing and rigidity of the muscles of the back of the neck. The most constant and significant of these symptoms of basal meningitis are early palsy of cranial nerves and rigidity of the muscles of the back of the neck.

On the other hand, when delirium is pronounced and early, cranial nerve paralyses are absent or slight, and local convulsions, slight hemiplegia and marked rigidity of the limbs occur, the indications point to *Meningitis of the Convexity*. Infrequent vomiting and the absence of optic neuritis render a basal meningitis unlikely if the other symptoms are doubtful and if there has been time for optic neuritis to develop.

Purulent Meningitis. Purulent cerebral lepto-meningi-

[1] This is a very constant symptom, especially in tubercular meningitis, and should always be looked for in cases of supposed meningitis.

tis of the convexity may be suspected if the symptoms of acute meningitis follow adjacent suppuration or a trauma or septicæmia. The course of the process is generally very acute. All of the four cardinal symptoms of acute meningitis—headache, vomiting, fever, and delirium—are usually well marked. The temperature is commonly high, continuous and rather irregular in its variations. The symptoms vary in intensity in different cases according to the severity of the process, and are sometimes partially latent for a considerable period,—especially in cases secondary to suppuration in other parts. The inflammation commonly involves chiefly the convexity; hence the symptoms are usually those of meningitis of the convexity, but sometimes the process extends to the base and causes basal symptoms.

Epidemic Cerebro-Spinal Meningitis is suggested by the presence of symptoms of cerebral meningitis (basal type) associated with those of spinal meningitis, the former predominating, and by a very rapid and severe onset of the symptoms. It is caused by the diplococcus intracellularis of Weichselbaum. In a typical case there are headache, vomiting, irregular and high fever, delirium, muscular rigidity, general hyperæsthesia of the skin, and retraction of the head. After a time varying from a day to a week, according to the severity of the case, the delirium that occurs in the early stage of the disease is succeeded by stupor deepening into coma. In some cases, both severe and light, herpes labialis or herpes facialis makes its appearance. This eruption is commonly an early symptom, and where present is of much help in making the diagnosis of epidemic cerebro-spinal meningitis. Petechial spots occur in a large proportion of severe cases and may also aid in the diagnosis. A knowledge of the prevalence of the disease in epidemic

form is of great help in distinguishing between this and other forms of meningitis.

Tubercular Meningitis ("basal meningitis") differs somewhat in its symptoms in children and adults. The symptoms which indicate tubercular meningitis in children are: (1) the occurrence for many days and weeks of a prodromal stage, consisting of loss of flesh and strength, and often of slight irregular fever, restlessness, irritability, apathy and slight headache, followed by (2) a period during which these prodromal symptoms continue, with the addition of vomiting without gastric cause or from inadequate cause, and increased headache, followed in turn by (3) the symptoms of a *basal* meningitis. Of the symptoms of basal meningitis (*vide ante*) the most constant and significant in tubercular meningitis are retraction of the head, vomiting, symptoms referable to the cranial nerves (especially strabismus) and irregular respiration. The temperature is irregular, intermittent, of no special type, and seldom high. The pulse is commonly irregular, is infrequent as compared with the respiration and not proportioned to the temperature; perhaps it is not only relatively but absolutely infrequent, being less than in health—60 to 50, or even 40, per minute. This is rather a characteristic symptom. In rare cases tubercular meningitis involves only or chiefly the convexity, and it must be borne in mind that the preponderance of the symptoms of convexity meningitis does not, in itself, exclude the possibility of the process being tubercular, though it certainly creates a presumption against this being the case.

In adults the symptoms of tubercular meningitis do not differ essentially from those in children. General convulsions, strasbismus and retraction of the head are less common than in children. Delirium occurs earlier

and headache is more persistent. In young women hysterical symptoms may attend and obscure the symptoms of the early period of the disease.

Partial or Localized Tubercular Meningitis of the convexity may produce focal symptoms (local paralysis or hemiplegia coming on in the course of a few days or weeks, with local convulsions) much like those of a tubercular tumor (see p. 375). Such symptoms may be combined with basal symptoms.

Certain facts regarding the etiology and occurrence of tubercular meningitis are of aid in diagnosis. First, there is the fact that in most cases there is a family history of phthisis or of infantile tuberculosis. Hence such a history in itself strongly suggests the tubercular nature of the meningitis in the absence of other causes. Secondly, in adults the usual indications of pulmonary tuberculosis are very often present when the meningitis appears and give a clue to its character. Third, during childhood and youth meningitis is far more often tubercular than purulent or simple. Hence the mere circumstance of youth or childhood creates a presumption that the inflammation is tubercular.

Simple, Non-purulent Meningitis (see page 270) may result from trauma, acute specific diseases, adjacent suppuration, or from sunstroke. It rarely or never follows septicæmia, and, with this exception, its etiology is practically that of purulent meningitis. The symptoms of this form of meningitis are essentially those of purulent meningitis, generally with preponderating basal symptoms, but the course of the disease is usually somewhat longer, and the acute may subside into a subacute stage. Simple meningitis is much more common than the purulent form.

The diagnosis of meningitis relates chiefly to cases of

tubercular meningitis since the acute course and causal relations of the other varieties leave little room for error.

Lumbar Puncture in the Diagnosis of Meningitis.—The diagnosis of the nature of an acute or subacute meningitis can often be established with certainty and facility by means of evidence obtained through the practice of lumbar puncture, and this is especially true of epidemic cerebrospinal meningitis and other forms of meningitis in which the spinal as well as the cerebral meninges are the seat of an inflammatory process. The presence of leucocytes in the fluid obtained by lumbar puncture points almost without exception to an inflammation involving the meninges. The absence of a cellular exudate, however, does not exclude meningitis, since, for example, it is usual in tubercular meningitis not to find such an exudate. Much more important is the evidence derived from the microscopic and cultural study of the cerebrospinal fluid. The discovery of the meningococcus (Weichselbaum's diplococcus intracellularis), the pneumococcus, the streptococcus pyogenes, the tubercle bacillus, or other pathogenic micro-organism, as the dominant or sole bacterial form in the cerebrospinal fluid, fixes the nature of the meningitic process. In the case of epidemic cerebrospinal meningitis the microscopical examination is alone sufficient in most instances to establish the nature of the affection. In a very large proportion of cases of this disease the meningococcus is found in the spinal fluid during the first and second weeks of the illness; the failure to obtain the specific organism in the second week is, in the case of children, considerably (though not necessarily absolutely) against the existence of the epidemic form of cerebrospinal meningitis. It must be understood that symptoms indistinguishable from those of the epidemic form of

cerebrospinal meningitis dependent on Weichselbaum's organism are caused not very rarely by other bacterial inciters of disease, such as the pneumococcus, the micrococcus lanceolatus, the streptococcus pyogenes, and the tubercle bacillus.

The failure to detect the tubercle bacillus in the cerebrospinal fluid, either on microscopical or cultural study, does not exclude the possibility of tuberculous meningitis, since the organism may be present in very small numbers, and animal inoculation may be necessary to exhibit its presence.

Some degree of prognostic significance attaches to the appearance of the cellular exudate in the early stages of acute cerebrospinal meningitis due to the specific meningococcus. The presence of degenerated leucocytes containing few micro-organisms, while many cocci are seen to be extracellular, points to feeble powers of resistance on the part of the leucocytes and hence to a bad prognosis. On the other hand, the presence of well preserved leucocytes showing many intracellular diplococci (indicating active phagocytosis) and the absence of many extracellular micro-organisms indicates good powers of resistance and a better prognosis (other conditions being comparable) than in the former case.

Meningitis may be confounded (1) with other diseases of the nervous system, and (2) with diseases outside the nervous system. The following are the conditions external to the nervous system in which the distinction has to be made.

a. Acute General Diseases (typhoid, typhus, etc.).—When a difficulty in diagnosis arises it is commonly in the early stage of these diseases, which are often mistaken for meningitis, though the opposite error also is sometimes committed. The error is of the same character in

all the cases in which an acute general disease is confounded with meningitis,—the early headache, the later delirium and the fever are incorrectly regarded as pointing to cerebral disease. But the delirium of an acute general disease is always accounted for by the fever, and in the absence of other symptoms of meningitis (persistent headache and vomiting) we are not justified in ascribing the delirium to meningitis unless the temperature is below 101°. The headache of acute general diseases and of febrile conditions generally *ceases when delirium comes on;* that of meningitis persists. When the occurrence of headache followed by delirium can be explained by the existence of fever there is no reason to suspect the existence of meningitis. Meningitis should be suspected in such cases only when focal symptoms, such as local spasm or paralysis, or diffuse cerebral symptoms which, like optic neuritis are not accompaniments of general diseases, make their appearance. In some instances a confident diagnosis can be made only after watching the course of the symptoms for several days.

b. Intestinal Disease in Children.—In some intestinal disorders in young children, especially exhausting diarrhœas, there may be cerebral symptoms that suggest meningitis for a time—rigidity, strabismus, convulsive movements, somnolence, and stupor and fever. The diagnosis is based on the absence of focal paralytic symptoms and on the short duration of focal spasmodic symptoms—where these exist,—the history of intestinal derangement, and on the presence of a depressed fontanelle. In rare cases intestinal worms in children give rise to disordered nutrition, irritability, headache, rigidity of extremities and strabismus, and these symptoms may be mistaken for those of tubercular meningitis. The facts

already given regarding tubercular meningitis and the diagnosis of meningitis in general will serve to prevent an error.

The following are the chief diseases of the nervous system that are likely to be confounded with meningitis:

a. Tumor of the Brain.—A tumor of rapid growth (glioma, tubercular tumor) sometimes gives rise to symptoms of rapid onset which may be confounded with those of meningitis. Partial paralysis of the limbs occurs in both conditions, but in tumor it occurs early and gradually, while in meningitis it is of rapid development and does not generally come on early. The presence of optic neuritis does not in itself give any help, as it may occur in both conditions, but a high grade of optic neuritis makes tumor highly probable, as does a neuritis which develops rapidly in intensity. The absence of optic neuritis does not aid in the diagnosis. When no indications can be obtained from the focal symptoms or optic neuritis, the course of the disease will often decide the diagnosis. If the nervous symptoms continue to increase for more than a fortnight after the beginning of the symptoms without the onset of coma the case is one of tumor; if the case is one of meningitis the coma will be of earlier occurrence. When meningitis is the result of tumor, the symptoms of the tumor usually precede those of the meningitis, which latter are distinguishable by their acute onset. Ordinarily cerebral tumors cannot be confounded with acute or subacute meningitis. The gradual onset and development of the symptoms and the absence of fever in the case of tumor make the distinction from meningitis, with its more acute onset and course and its fever, sufficiently easy.

b. Abscess of the Brain.—Purulent meningitis may be mistaken for abscess, since the latter may give rise to

symptoms of rapid onset, and both are often secondary to the same etiological factors, injury and ear disease. The course of abscess may be latent until the acute symptoms develop, and if these are sufficiently acute the distinction from meningitis may be impossible. More often the symptoms of abscess run a subacute or chronic course, and this makes the distinction easy in most instances. Then again the fact that in meningitis the cranial nerves are more extensively and commonly involved than in abscess, helps in making the distinction. The presence of an active, persistent, unilateral ankle clonus suggests abscess. It must be borne in mind that purulent meningitis is often the terminal stage of abscess, and that the two conditions may coexist. In such cases the acute symptoms of meningitis are preceded by the signs of more chronic cerebral disease.

c. Hysteria.—It is not rare for meningitis, especially tuberculous meningitis, to be mistaken for hysteria. This error is especially liable to arise in cases of meningitis occurring in young females who are predisposed to hysteria and in whom hysterical symptoms may appear during the early stage of the disease. Even if there are no actual hysterical symptoms during the invasion of the disease, a knowledge of previous hysterical symptoms in a young girl may lead to the erroneous interpretation of the signs of organic cerebral disease. In cases where doubt arises it may usually be dispelled by due regard to the temperature, the existence of local convulsions and the state of the fundus oculi. Fever, convulsions of local commencement and optic neuritis all point strongly to meningitis. Marked inequality of the pupils has the same significance. Strabismus may occur in hysteria as well as in meningitis, but is always convergent. Divergent strabismus, therefore, indicates meningitis. In general it

may be said that the most important rule to be followed in doubtful cases is to look sedulously for any indications whatsoever of organic disease, and to allow these their full diagnostic significance even in the presence of undoubted hysterical symptoms.

Chronic Meningitis.—The symptoms of chronic meningitis are both focal and diffuse, but the focal symptoms, as paralysis and spasm, usually predominate. Fever and delirium, which are commonly conspicuous in acute meningitis, are absent or slight in degree in the chronic form.

The principal forms of chronic meningitis met with in adults are the alcoholic and the syphilitic. Chronic meningitis in adults not due to one or both of these causes is exceedingly rare. In children there is a chronic form of meningitis which is occasionally related to syphilis, but of which the other causes are unknown.

Chronic Alcoholic Meningitis is a diffuse process involving chiefly the convexity of both hemispheres. The symptoms are for the most part diffuse, and consist of headache, which is constant and may be either moderate or severe, mental dulness or apathy and sometimes delirium. Occasionally there is a slight degree of optic neuritis. The disease runs an exceedingly chronic course.

When the symptoms are well developed the condition is easy of recognition. The marked alcoholic history, the unmistakable alcoholic tremor and the other evidences of alcoholism make plain the nature of the diffuse cerebral symptoms. When other and more obtrusive effects (such as chronic diffuse nephritis, interstitial hepatitis, multiple neuritis) of alcoholism coexist with chronic meningitis, the symptoms of the latter, if slight, are readily overlooked. Its symptoms, like

those of tumor, depend largely on the position of the lesion.

Chronic Syphilitic Meningitis is usually a localized, seldom a diffuse, process; it occasionally exists where there is no distinct new growth. It occurs, as a rule, about a syphilitic growth. It may occur either at the base or on the convexity of the brain, and in either case the symptoms are chiefly focal in character, and result from irritation and compression, especially from irritation. When the meningitis occurs about a syphilitic growth its symptoms are not readily distinguishable from those of the tumor. When the process exists alone, the symptoms still resemble those of a syphilitic tumor, with which they may be confounded. As compared with a syphiloma, the symptoms of chronic syphilitic meningitis are more irritative and less destructive in character. When the process involves the base of the brain a larger number of cranial nerves is involved than is usual in tumor, and the extent of mischief to each nerve from compression is less. The distinction of chronic syphilitic meningitis from other forms of chronic meningitis is based on its limited extent and upon the rapid improvement that occurs on antisyphilitic treatment. The most constant of the diffuse symptoms of chronic syphilitic meningitis is headache.

In Chronic Infantile Meningitis the most constant and distinctive symptom is spasm of the muscles of the back of the neck, causing retraction of the head. The development of this symptom may be rapid or gradual, and is preceded, in some cases, by fever, repeated vomiting, and general convulsions. There may or may not be rigidity of the limbs; paralysis is rare. The symptoms of hydrocephalus are sometimes added to these symptoms. Sometimes retraction of the head and convulsions date from

birth. In these cases the lesion is not meningitis but meningeal hemorrhage.

Chronic infantile meningitis may last many months or several years. Usually it ends in death, but in the cases which depend on syphilis recovery may occur.

Pachymeningitis Interna Hæmorrhagica (Hæmatoma of the dura mater).—This is the only form of pachymeningitis that requires mention. External pachymeningitis is practically always secondary to injury or to neighboring disease, and its symptoms are usually obscured by those of the primary trouble. Purulent internal pachymeningitis, an exceedingly rare condition, gives symptoms indistinguishable from those of purulent leptomeningitis.

Cerebral Hemorrhage.—It is convenient clinically to divide all cases of intracranial hemorrhage into two groups—of which the first includes all cases of hemorrhage into the brain substance—cerebral hemorrhage proper,—while the second includes all cases of hemorrhage from the membranes of the brain—meningeal hemorrhage.[1]

The symptoms of cerebral hemorrhage proper vary considerably according to the position and size of the hemorrhage, but almost all cases have certain features in common—namely: 1. The sudden onset of the symptoms. 2. The occurrence of general and usually transient symptoms, especially loss of consciousness. 3. The occurrence of local and persistent symptoms, especially symptoms of injury to one motor tract, of which hemiplegia is the most constant manifestation.

The most common position, by far, of hemorrhage into the brain substance is the internal capsule, and the

[1] Most cases of hemorrhage from meningeal vessels are dependent on injury to the head, and are therefore treated elsewhere.

symptoms of capsular hemorrhage resemble those of hemorrhage anywhere in the motor tract of the brain, and may be regarded as constituting the type of the disease. The symptoms that point to a hemorrhage in the internal capsule are as follows: 1. Loss of consciousness, of sudden or rapid onset, which is common to all cases in which the hemorrhage is large, without reference to its position, (but which may be absent if the lesion is very small) and which presents the features described on page 184. 2. Hemiplegia of sudden onset—usually complete—*i. e.*, involving face, arm, and leg. 3. Motor aphasia if the hemiplegia is on the right side.

During the period of unconsciousness, which lasts in those that recover from a few minutes to several hours, there may be distinct evidences of a unilateral lesion, as unilateral loss of reflex action, conjugate deviation of the eyes to the side of the lesion, increased movement of the upper part of the thorax on the paralyzed side, defective movement and flaccidity of the limbs of one side, etc.

Some of these symptoms, such as unilateral loss of reflex and conjugate deviation of the eyes, wear away soon after the return of consciousness. The distinctly focal symptoms, hemiplegia, and motor aphasia, if this exists, persist for a variable period, according to the extent of the destruction, for the persistent focal symptoms depend on destruction of tissue, while the more general symptoms depend on pressure and irritation. These persistent focal symptoms depend on the situation of the lesion (see Localization), and constitute the most important symptom of the chronic stage of cerebral hemorrhage. The less important symptoms of the chronic stage are slight headache and some mental change, very rarely convulsions.

The symptoms that indicate hemorrhage in other

parts of the brain than the internal capsule differ somewhat from those of capsular hemorrhage, according to the situation of the lesion, and may be for the most part deduced from the account elsewhere given of cerebral localization (p. 213). Some of these variations in the symptoms, together with certain other facts relating especially to cerebral hemorrhage, may properly be mentioned here.

The symptoms of hemorrhage into the substance of the cortex differ from those of hemorrhage into the internal capsule in the following particulars: 1. The onset is generally attended by local convulsions, or convulsions having a local commencement. 2. If motor paralysis results it is usually confined to one part of a side, face, arm, or leg, after the usual manner of cortical disease.

The symptoms that point to a hemorrhage into the centrum ovale closely resemble those of a cortical hemorrhage, without its irritative symptoms. A hemorrhage into the crus is indicated by crossed hemiplegia (see p. 224) with or without hemianæsthesia, in addition to the diffuse symptoms of hemorrhage. The symptoms that suggest a hemorrhage in the pons are: 1. General convulsions attending the onset, or irregular convulsions confined to the legs. 2. Bilateral motor paralysis. 3. Marked bilateral anæsthesia. 4. Contraction or dilatation of the pupils. 5. Respiratory embarrassment. 6. Repeated vomiting. 7. High temperature. A hemorrhage into the medulla, if of considerable size, is rapidly or instantly fatal, and, even if of small size, is rarely survived. Cerebellar hemorrhage varies much in its symptoms (see Localization), and cannot ordinarily be diagnosed. Primary hemorrhage into the ventricles can rarely be diagnosed. Hemorrhage into the ventricles, which is secondary to hemorrhage in other parts of the brain is

suggested by: (1) the occurrence of a second apoplectic seizure soon after the first, together with (2) the extension of hemiplegic symptoms (especially relaxation of the muscles) from one side *to both sides* of the body.

Cases of hemorrhage into the internal capsule make up more than one half the cases of cerebral hemorrhage; next in frequency is hemorrhage into the centrum ovale, then in succession hemorrhage into the cortex, pons and cerebellum, hemorrhage into the medulla and crus being very rare.

The most important facts regarding the etiology of cerebral hemorrhage are, that the disease is one of the second half of life and that chronic Bright's disease, gout and chronic alcoholism are conditions that predispose strongly to its occurrence.

The diagnosis of cerebral hemorrhage has to be made either during the apoplexy, or after the apoplectic stage has passed. During the apoplectic seizure the diagnosis is often difficult and occasionally impossible, but it is exceedingly important that every effort be made to determine the nature of the case, as the correct treatment and prognosis depend upon it.

When a patient is seen in apoplexy and cerebral hemorrhage is suspected, the first thing to determine is whether there are any evidences—such as hemiplegia, unilateral rigidity or relaxation, unilateral convulsions or unilateral loss of knee-jerk—of a focal brain lesion. If such evidences of a sudden focal lesion are entirely absent, and if there is no history of unilateral numbness or weakness before the loss of consciousness came on, the condition is probably not dependent on a cerebral lesion. If, however, indications of a focal lesion are detected, the dependence of the seizure on a cerebral lesion is quite clear. The question to be decided then

is, is the cerebral lesion hemorrhage or is it one of the varieties of acute softening? The various aspects of this question are discussed under the different forms of acute softening of the brain. (See pages 358, 359, 360.)

The apoplectiform seizures of general paralysis of the insane may be confounded with cerebral hemorrhage. In the former, as in the latter, there may be sudden loss of consciousness, hemiplegia, unilateral or general convulsions and a rise in temperature. But in the apoplectiform attack of general paralysis the hemiplegia seldom lasts more than a few hours, and when the patient comes out of coma the distinctive mental and physical indications of the disease become only too evident.

Sudden apoplectic seizures not due to a cerebral lesion,[1] of short duration and usually without any local symptoms, but sometimes accompanied by slight and transient hemiplegic weakness, are occasionally observed. These seizures occur in elderly persons, and may be mistaken for cerebral hemorrhage. In a first attack the distinction may be impossible. The diagnosis is based on the absence of deep coma and the absence or transient duration of focal symptoms. Sometimes a history of repeated attacks of this character can be obtained, and this is a great aid in the diagnosis. The nature of these seizures is unknown. They have been thought to depend on cerebral "congestion," and it may be that some of the cases are of this nature; but it is probable that in some cases, at least, the symptoms depend on diminished blood-supply to motor structures of the brain, due to transient spasm of cerebral arteries.

Acute Cerebral Softening (Softening from Arterial Occlusion, Necrotic Softening, Encephalomalacia.)—

[1] In numerous cases of this kind the patient has died from other causes and the autopsy has failed to show any lesion.

The symptoms that characterize acute cerebral softening closely resemble, in their general features and in many details, the symptoms that result from cerebral hemorrhage (see p. 348).

As in hemorrhage there are: (1) general and transient symptoms (as loss of consciousness) due to general disturbance of brain function; (2) local and enduring symptoms (especially hemiplegia, and when the lesion is on the left side, motor aphasia) dependent on the situation of the lesion, and due directly to the destruction of tissue caused by it; and (3) these symptoms are usually either of sudden or of rapid onset.

In addition to this general resemblance between acute softening and hemorrhage there is a resemblance in details between certain forms of softening and hemorrhage that would make the two indistinguishable more often than they are, were it not for the diagnostic aid derived from the study of the causal indications.

Two principal forms of acute softening, based on pathological differences (see page 264) must be distinguished. These are: (1) softening from embolism and (2) softening from thrombosis. The latter form includes three sub-varieties: (1) Thrombosis from atheroma, (2) from syphilis, and (3) from blood states, between which it is important to distinguish clinically. These different forms of acute softening from arterial obstruction make up a large and important group of cases; they are, in fact, as common if not more common than cases of cerebral hemorrhage.

Acute Softening from Embolism.—In acute softening from embolism premonitory symptoms are exceedingly rare; the onset is sudden, the loss of consciousness and the accompanying focal symptoms coming on in the course of a few seconds or minutes. In rare cases

the onset is gradual, occupying several hours, and in these cases there may be no loss of consciousness. As a rule, the more acute the onset the greater the likelihood of there being loss of consciousness, but this is of *short duration* in embolism. Convulsions are also frequent in embolism, at the onset ; they may be general or local. When the beginning is local it is generally in the part afterwards most paralyzed. The focal symptoms are like those of hemorrhage and vary similarly with the position of the lesion. Hemiplegia is a very constant symptom. Partial hemiplegia, especially paralysis of the arm, or of the face and arm, is not uncommon. It is much more frequent than in hemorrhage. When the lesion is on the left side aphasia is as common as in hemorrhage.

The etiological facts regarding embolism which it is important to remember are :

a. That there is usually present, at least during the first year after the onset of the symptoms of embolism, distinct indications of valvular disease of the heart (especially mitral stenosis).

b. That the absence of evidences of endocarditis a year or more after the onset is perfectly consistent with the occurrence of embolism, provided the onset succeeded a disease which, like scarlet fever, chorea or rheumatism, is apt to be associated with valvular disease.

c. That embolism occurs most frequently during later childhood and early adult life, though it may occur at any period of life.

Acute Softening from Syphilitic Thrombosis (*i. e.*, from syphilitic endarteritis).—The symptoms of acute softening from syphilitic thrombosis usually differ in several respects from those of embolism. First, premonitory symptoms are the rule. The attack is preceded very

often by well marked cerebral symptoms. Of these headache is the most common and characteristic. The headache is frequently severe and is often aggravated at night. Other less constant premonitory symptoms are giddiness, mental apathy or somnolence, numbness of one or more extremities and slight hemiplegic weakness. Another point of difference is that in syphilitic thrombosis loss of consciousness at the onset is much more common than in embolism, being absent in about half the cases. Initial convulsions are also much more rare. The focal symptoms present no important points of difference.

The etiological facts bearing on the diagnosis of syphilitic softening which it is important to bear in mind are :

a. That there is a history of syphilitic infection in a large number of cases, and that the date of this infection varies from a year to twelve or fifteen years before the onset of the symptoms of softening.

b. That in the cases where a history of syphilis cannot be obtained (as in coma), but where infection cannot be positively excluded, reliance must be placed on the age of the patient and on the absence of other causes (atheroma, blood states) of acute softening. The important fact regarding age is that softening from syphilis is rare before twenty and after fifty and that half the cases occur between the thirtieth and fortieth year.

Acute Softening from Atheroma.—The symptoms of softening from atheroma do not differ essentially from those of softening from syphilitic endarteritis ; as compared with embolism there is the same frequency of premonitory symptoms [1] (especially slight general head-

[1] It is of course very evident why premonitory symptoms are absent in softening from embolism and usually present in softening from atheroma and syphilitic endarteritis. In the case of softening from atheroma or syphilitic endarteritis the arterial disease gradually

ache, numbness, hemiplegic weakness and slight mental change), and a similar though not equally marked immunity from loss of consciousness in some cases. The similarity in symptoms extends to the focal as well as to the diffuse symptoms. The etiological facts relative to the diagnosis are as follows :

a. Softening from atheroma is chiefly a disease of the second half of life. Before the age of forty it is infrequent except in association with chronic Bright's disease (chronic diffuse nephritis). After forty the liability to the disease increases as life advances, and this liability continues to increase until extreme old age. Senility is therefore a most important etiological factor.

b. Chronic alcoholism predisposes to atheroma, and any condition which, like blood disease or cachexia, favors thrombosis, favors softening from atheroma. The occurrence of atheroma of the vessels of the brain is favored by injury to the head.

Acute Softening from Blood-States (Simple Arterial Thrombosis).—Arterial thrombosis from a blood-state alone, without the existence of arterial disease, is a rare cause of acute softening of the brain. It usually results in adults from the state of the blood that exists during

diminishes the lumen of the diseased vessels, and a point is at length reached where the quantity of blood passing through the vessel is so little as to *impair* the nutrition of the parts supplied by it without destroying them. This is the period of premonitory symptoms. The diminished blood-stream favors coagulation, and after a variable period a thrombus forms at the highly diseased region of the vessel. The blood supply of the dependent parts is then suddenly shut off, with resulting softening of these parts and loss of consciousness, hemiplegia, etc. In softening from embolism, on the contrary, the sudden lodgment of the embolus is not preceded by any vascular disturbance, the impairment in nutrition of the brain is sudden and great and the symptoms are of sudden onset and without premonitions.

the puerperal state, and the indications of the cause are always evident and usually obtrusive. The symptoms in such cases resemble those of embolism, the focal symptoms varying similarly with the position of the lesion.

When a blood-state like that which accompanies tuberculosis, cancer, acute diseases, etc., is associated with disease of the cerebral arteries, it has an important influence in favoring the occurrence of thrombosis in them.

The occurrence of softening from an arterial obstruction should be suspected in cases giving a history of a sudden cerebral lesion in the presence of conditions that are recognized as the causes of such arterial obstruction. The diagnosis of the cause of the softening (embolism, syphilitic endarteritis, atheroma, blood-state) *depends on the causal indications*, and in far slighter degree on the character of the symptoms that point to the sudden cerebral lesion. These various indications have been already sufficiently considered. The chief diagnostic problem relating to acute softening is the distinction from cerebral hemorrhage.[1] The form of acute softening which it is most difficult to diagnose from hemorrhage is that from atheroma. The diagnosis between embolic softening and hemorrhage, and between syphilitic softening and hemorrhage, is rendered much simpler by the etiological indications.

1. *The Diagnosis between Softening from Atheroma and Cerebral Hemorrhage* can be made with a high degree of probability in a considerable number of cases, but is impossible in others. In all cases it is a matter of probability, and that is more often low than high. In every case where the question of such a diagnosis

[1] This is an exceedingly important question to decide, on account of its bearing upon treatment, which in the one case is stimulating and in the other is depressing to the heart.

arises a conclusion should be reached only after a careful study of all the symptoms, both general and focal, and of the causal indications. The following are the more important symptomatic and causal indications :

Prodromic symptoms (*vide* atheroma), if present, point to softening, but their absence does not point to hemorrhage.

Little help is to be derived from the degree, duration and occurrence of loss of consciousness, unless there is some indication of the size of the lesion. If the lesion is probably small (as indicated by *partial* hemiplegia), loss of consciousness is decidedly in favor of hemorrhage. If the lesion is probably large, the absence of loss of consciousness points strongly to softening. In the absence of indications relating to the size of the lesion deep coma makes hemorrhage probable, while a slight attack of apoplexy of transient duration is in favor of softening. A general convulsion at the commencement of the attack makes hemorrhage more likely, but initial local spasm points to softening.

If the temperature falls more than one degree at the time of the onset, or if it rises several degrees within a few hours after the onset, this is much in favor of hemorrhage, except in cases where the lesion is situated in the pons, when the rule loses its value.

When the early stage is past the repeated occurrence of convulsions commencing in the paralyzed extremities, and the presence of *athetoid movements* or post-hemiplegic agitation or tremor indicate that the lesion was softening, not hemorrhage. Of the causal indications, the condition of the heart and arteries is the most important. High arterial tension and a bounding pulse at the time of the attack point to hemorrhage if the other indications are balanced, while a feeble and irregular pulse, especially if compressible, suggests softening. As the conditions of

disease observed in association with hemorrhage and softening from atheroma are much the same, their value in this diagnostic problem is limited. A considerable degree of atheroma of the arteries points to softening rather than hemorrhage, but only if the other indications are equal. The reverse is true of an advanced state of chronic diffuse nephritis. A history of a cachexia or an exhausting illness before the onset favors softening. The age of the patient does not help much, but the greater the age above seventy-five the greater are the chances that the process is softening, while under forty-five hemorrhage is more probable.

2. *The Diagnosis between Softening from Syphilitic Endarteritis and Cerebral Hemorrhage* is ordinarily a less difficult problem than the one just described, and in it the causal indications play a more important rôle. The important differential points are the following :

The presence of prodromata indicate softening, but their absence does not aid in the diagnosis. The facts relating to loss of consciousness are the same as in the diagnosis between softening from atheroma and hemorrhage *(vide ante)*. A history of acquired syphilis, if the other indications are balanced, makes softening very probable, but if there is reason to suspect that the lesion is hemorrhage, a specific history does not render this diagnosis improbable, since syphilis may lead to hemorrhage by causing an aneurism. But if there is a history of syphilis in a patient over fifty this has little significance in favor of softening. The presence of atheroma or chronic Bright's disease makes the diagnosis of hemorrhage highly probable, as between hemorrhage and softening from syphilitic disease. The absence of atheroma and chronic Bright's disease, with a *possibility* of syphilitic infection, suggests softening. The age of the

patient generally gives important help in the diagnosis, since before the age of forty the chances are much in favor of syphilitic softening, and after forty-five they are equally in favor of hemorrhage. If the patient is between forty and forty-five years of age the indications derived from age are of no value.

3. *The Diagnosis between Softening from Embolism and Cerebral Hemorrhage* ordinarily presents few difficulties. Deep prolonged coma is strongly in favor of hemorrhage; the absence of loss of consciousness, or a slight and temporary loss of consciousness, suggests embolism. The most important evidences in favor of embolism are the presence of a cause of embolism (heart disease, aneurism) in a patient under forty years of age. If the patient is between forty and fifty embolism is still probable, if there is a cause of embolism, but after this period becomes improbable. It is often stated that the presence of right hemiplegia is a point in favor of embolism, but this is an error.[1] In rare instances the diagnosis of embolism may be made through evidences of an embolic process in another part of the body. Thus, the occurrence of an infarction of the spleen, as shown by tenderness and enlargement coming on acutely, or the occurrence of obstruction of the central artery of the retina, as shown by ophthalmoscopic changes, about the time of a sudden cerebral lesion, make the existence of cerebral embolism in the former case exceedingly probable, and in the latter practically certain.

Meningeal Hemorrhage.—Only two forms of

[1] Right hemiplegia from embolism is certainly rather more common than left hemiplegia (about 6–5), but the difference in the incidence of the hemiplegia is not enough to constitute a diagnostic sign, and would not constitute a valuable sign if the difference were much greater.

meningeal hemorrhage, both of them dependent upon injury, require our notice. Of these two forms, one—infantile meningeal hemorrhage—occurs in the course of delivery, and is the cause of an important form of infantile cerebral palsy (see p. 367). The hemorrhage in these cases is between the dura and pia over the convexity, usually on one, sometimes on both sides of the brain. The symptoms are such as might be expected from the irritation and compression of the cortex by the clot—convulsions (general or unilateral), rigidity, opisthotonos and hemiplegia, sometimes diplegia. The diagnosis presents no difficulties if the occurrence of the condition is remembered, its relation to injury during labor (difficult or protracted) being obtrusively clear.

The recognition of the second form of meningeal hemorrhage—*extra-dural hemorrhage*[1]—though often easy, may be exceedingly difficult. There is in this condition an extravasation of blood between the dura and the skull from the middle meningeal artery. It occurs usually on one side of the head, but sometimes the hemorrhage occurs on both sides. Almost always the injury which causes extra-dural hemorrhage gives rise to fracture of the skull, and generally this is extensive and grave in character (vault and base). The condition always depends on direct injury to the head, usually severe, but sometimes, it is important to note, apparently trivial in character. Very often the injury is from a fall of a serious character.

[1] This condition is of frequent occurrence, and is of great importance, both medically and surgically. It is especially important that it should not be confounded with hemorrhage into the brain. In many fatal cases of fracture of the base, death depends on extra-dural hemorrhage—a condition which can sometimes be successfully relieved by surgical means.

The chief clinical characters of extra-dural hemorrhage are as follows :

A. There is a period of consciousness (lucid interval)[1] between the time of injury and the appearance of symptoms of brain compression. This interval may be of only a few minutes' duration, or it may last several hours ; generally the interval is short but well marked. Such an interval is present in considerably more than half the cases of extra-dural hemorrhage, and its presence is therefore highly suggestive of the condition. Its absence, on the other hand, does not in itself render the condition improbable.

B. The lucid interval is followed by pressure symptoms of rapid or gradual onset. Sometimes these symptoms supervene directly on the injury. They are as follows :

a. Hemiplegia, partial or complete, preceded or accompanied by convulsions, usually unilateral, but sometimes general. The hemiplegic limbs may be rigid or flaccid. The knee-jerk is usually but not always lost on the hemiplegic side. Hemiplegia is a very frequent and characteristic symptom, and in its total absence the diagnosis of extra-dural hemorrhage cannot be made with any degree of certainty.

b. Dilatation and loss of reaction in the pupil on the side *opposite* the hemiplegia, *i. e.*, on the side of the lesion. This symptom is thought to depend on pressure on the third nerve at the base. It is present in a considerable proportion of all the cases of extra-dural hemorrhage and is a highly characteristic symptom.

c. Stupor gradually deepening into coma.

The diagnostic value of these symptoms is greatly increased by their association with evidences of fracture

[1] In rare cases of extra-dural hemorrhage the lucid interval lasts several days.

of the base of the skull, especially bleeding from the ear, escape of the cerebro-spinal fluid and tumefaction about the mastoid process. Other less characteristic but important symptoms of different frequency are repeated vomiting, a slow and hard pulse, embarrassed and slow respiration, aphasia, hemianæsthesia (partial), loss of sphincteric control, contraction of the pupils and moderate fever.

The gradual development of anæsthesia points to a backward extension of the hemorrhage; ophthalmoplegia to extension towards the base, and aphasia to extension anteriorly.

The distinction of extra-dural hemorrhage from medical conditions causing rapid or sudden loss of consciousness and focal symptoms (hemorrhage, acute softening) is usually not difficult, but in the absence of any history of injury, or of evidence of fracture of the skull, the diagnosis may be extremely difficult or impossible. Other conditions, which are liable to be confounded with extra-dural hemorrhage are: (1) laceration of the brain[1]: (2) laceration of the lateral or superior longitudinal sinus, with extravasation into the arachnoid cavity, or between the base and dura; and (3) hemorrhage into the arachnoid cavity, not from a sinus. All of these conditions depend on injury, and any or all of them may be associated with extra-dural hemorrhage. No positive data can be given for the distinction of extra-dural hemorrhage and laceration of the brain, but the following facts may aid in the diagnosis: (*a*) In extra-dural hemorrhage any wound or bruise is over the position of the middle meningeal artery; in laceration of the brain it is often on the side opposite the lesion. (*b*) In

[1] Generally the temporo-sphenoidal, or frontal lobes, often on the side *opposite* the injury.

extra-dural hemorrhage there is usually an interval of lucidity, and the injury causing the condition *may* be slight; in laceration of the brain there is no interval of lucidity, and the injury is always severe. (*c*) Severe and persistent convulsions on one side of the body *suggest* laceration of the brain, especially if this condition be unassociated with hemiplegia. In cases of hemorrhage from a sinus the position of the wound is a sufficient indication of the nature of the condition. It is practically impossible to make a diagnosis between extra-dural hemorrhage and hemorrhage into the arachnoid. The former is much the more frequent and important condition, and fortunately the treatment for it—trephining—is also the proper treatment for the latter condition. Which condition we have to deal with is perfectly evident when the button of bone is removed from the temporal region. In the case of extra-dural hemorrhage the clot lies next the bone; in the case of arachnoid hemorrhage, if this is considerable and has caused symptoms closely resembling a typical extra-dural hemorrhage, the dura will probably be found to bulge from the pressure of the clot beneath it.

In some cases of extensive *Subarachnoid Hemorrhage*[1] the symptoms are highly equivocal. There may be no

[1] Sub-pial hemorrhage often occurs in association with subdural or subarachnoid hemorrhage. Its symptoms, if there are any, are then usually lost in the more obtrusive symptoms of the condition with which the sub-pial hemorrhage is associated. But sub-pial hemorrhage may occur unassociated with hemorrhage elsewhere. It depends in such cases on injury to the head which causes severe concussion but no fracture. The hemorrhage is usually single, is located over the convexity (especially of the frontal or parietal region), and varies in size from half an inch to two inches in diameter. The layer of extravasated blood is usually thin. If located over the motor area the hemorrhage

symptoms for four or five days after the injury except slight headache. Then there may be repeated general convulsions during several days, with fever (101°–103°), delirium and somnolence. These symptoms may pass away entirely for several days, and the patient may appear about to recover, when stupor reappears, deepens into coma, the temperature rises and the case terminates in death. The presence of convulsions, headache, fever and delirium in such a case may lead to a suspicion of meningitis, but the only form of meningitis that is likely to follow immediately on a severe injury to the head is suppurative in character and is distinguished by higher fever and a more rapid and progressive course. It is important to note the fact that a slowly developing subarachnoid hemorrhage (with a long lucid interval) may cause no hemiplegia, although the motor area of the brain is much compressed.

Sinus-Thrombosis.—The principal facts relating to the pathology of sinus-thrombosis have been already considered (p. 267). Clinically it is necessary to keep distinct the two forms of sinus-thrombosis—primary or marantic thrombosis and secondary thrombosis.

a. Primary or Marantic Thrombosis.—The symptoms that indicate this condition are : (1) stupor gradually deepening into coma, (2) general convulsions frequently repeated with or without focal paralysis, associated with (3) an exhausting disease—as phthisis or cancer in its last stages, long-continued suppuration, acute specific disease or, most frequently of all, prolonged diarrhœa

may cause repeated convulsions and slight hemiplegic weakness. A hemorrhage not over the motor region may cause no symptom except stupor, but on the other hand may cause repeated general convulsions. It may be difficult to distinguish between the effects of the hemorrhage and those of concussion.

during infancy, and (4), in some instances, distension of external veins connected with the sinus thrombosed.

In young children (especially infants), in whom a very large percentage of the cases of marantic sinus-thrombosis occur, the general cerebral symptoms that result from the thrombosis are often obscured by the general cerebral symptoms that accompany an exhausting diarrhœa, for stupor, coma and general convulsions may result from the cerebral anæmia which an exhausting diarrhœa frequently induces. The diagnosis of sinus-thrombosis should be suspected in young children only when there are present, in addition to the general cerebral symptoms, symptoms of a focal character (local paralysis, local convulsions, local rigidity) or external tumefaction[1] developing after the other symptoms. The diagnosis is to be considered certain only in the rare cases in which the latter is present.

In adults in whom there is a cause for thrombosis (exhausting disease — phthisis, cancer, in terminal stage), marantic sinus-thrombosis may be suspected if there are the acute cerebral symptoms already mentioned, provided there can be excluded all causes of arterial thrombosis (especially atheroma, heart disease, degenerative time of life). But here, as in children, a positive diagnosis can be made only in the presence of external distension of veins or œdema. It is important to note that in persons over fifty the cerebral symptoms referred to are far more apt to be due to arterial thrombosis than to sinus-thrombosis.

[1] The positions in which the external distension of veins or œdema occurs are: œdema and distension of the veins on the forehead and sides of the head in thrombosis of the superior longitudinal; œdema of the eyelids and temples in thrombosis of the cavernous sinus; œdema over the mastoid process in thrombosis of the lateral sinus.

b. Secondary Thrombosis.—There is nothing characteristic about the symptoms of secondary sinus-thrombosis. The sinus-thrombosis results in most cases from caries of bone, especially from disease of the internal ear, more rarely from meningitis (especially tubercular) and suppuration external to the skull, and such symptoms as are due to the thrombosis are obscured by the more obtrusive evidences of the primary trouble. Speaking generally, the symptoms that attend secondary sinus-thrombosis closely resemble those of meningitis running an acute or subacute course, and they are, indeed, usually due to meningitis. In many cases the breaking down of the clot, which is septic, leads to symptoms of septicæmia. The conditions, therefore, that suggest secondary sinus-thrombosis are : (1) the presence of a cause of secondary thrombosis ; (2) symptoms resembling acute or subacute meningitis, associated with or succeeded by (3) symptoms of septicæmia. But it is only when these symptoms are associated with external œdema or venous distension that a diagnosis of secondary sinus-thrombosis can be made with a high degree of probability.

Infantile Cerebral Paralysis.—The paralyses of cerebral origin that are frequently observed during infancy and childhood are not all dependent upon one pathological state, but may probably result from any one of several distinct conditions—from hemorrhage, arterial or venous thrombosis, embolism, etc. Notwithstanding these different modes of origin of the infantile cerebral paralyses, their symptoms resemble one another so closely, whatever may be their origin, and their pathology is in some instances so obscure, that it is not at present feasible or desirable to classify them according to the nature of the lesions to which they are due. It is preferable to group

them all under the clinical designation above adopted—infantile cerebral paralysis.

The clinical characters of infantile cerebral paralyses, though differing widely in some cases, have enough points of resemblance to constitute a very distinct clinical type. These characters are as follows :

1. Motor paralysis, usually hemiplegic in form, occasionally diplegic and rarely monoplegic. The paralysis, whatever may be its form, has the following features:

a. There is marked rigidity in the muscles paralyzed, and in most cases contractures, which may be extreme.

b. The knee-jerks and reflexes generally are much exaggerated. Usually this increase of reflex is most marked on the side paralyzed if the case be one of diplegia.

c. The paralysis is frequently followed after a time by athetoid, associated, choreiform or other movements.

d. The muscles paralyzed do not present the RD, and electrical changes, when present, are quantitative or slight in degree.

e. The paralysis is usually partial. If considerable at first it rapidly lessens. In hemiplegic cases the face often escapes entirely. The leg recovers more rapidly and perfectly than is the case when hemiplegia occurs in the adult, and the ultimate paralysis may be chiefly in the arm.

f. There are no disturbances of sensibility.

2. In the cases of infantile cerebral paralysis that develop in children who have acquired language, transient motor aphasia is often developed. In cases of hemiplegia in children, aphasia is almost as common when the lesion is on the right side of the brain as when it is on the left side.

3. In nearly all cases of infantile cerebral paralysis there is arrest or retardation of development in the limbs

paralyzed. The paralyzed extremities become shorter and smaller than those not paralyzed. The arrest may be noticeable in the shape of the cranium, and in a diminution in most of the cranial diameters. There are well-marked vaso-motor changes in the paralyzed extremities, and atrophy of the muscles, slight or considerable in degree, is usually present.[1]

4. Nearly all cases of infantile cerebral paralysis are accompanied with some degree of mental impairment. In a small proportion there is idiocy (most often in diplegic and paraplegic cases), in others there is imbecility, and most frequently there is simple feeble-mindedness (most common form in hemiplegia).

5. Nearly one half the cases of infantile cerebral paralysis are followed by epilepsy. In most cases the form is that of general epilepsy (grand mal),with a local onset, but in a considerable number the attacks are partial, incomplete, or Jacksonian in type. The convulsions persist and sometimes survive all traces of the original paralysis.

For clinical purposes it is desirable to distinguish three types of infantile cerebral palsy : (I.) Paralysis of intra-uterine onset ; (II.) palsy at birth ; (III.) the acquired form.

(*I.*) *In Paralysis of Intra-uterine Onset.* In paralysis of intra-uterine onset, which is of comparatively rare occurrence, the morbid lesion usually is parencephaly with large areas of cortical destruction, defective development of the pyramidal tract, and imperfect development of those parts of the brain subserving the higher mental processes.

[1] In rare instances hypertrophy of paralyzed parts occurs owing to athetosis and other morbid movements.

(*II.*) *The Congenital Form of Infantile Cerebral Palsy* (*Cerebral Birth-Palsy, Infantile Meningeal Hemorrhage*). —The congenital form of infantile cerebral palsy

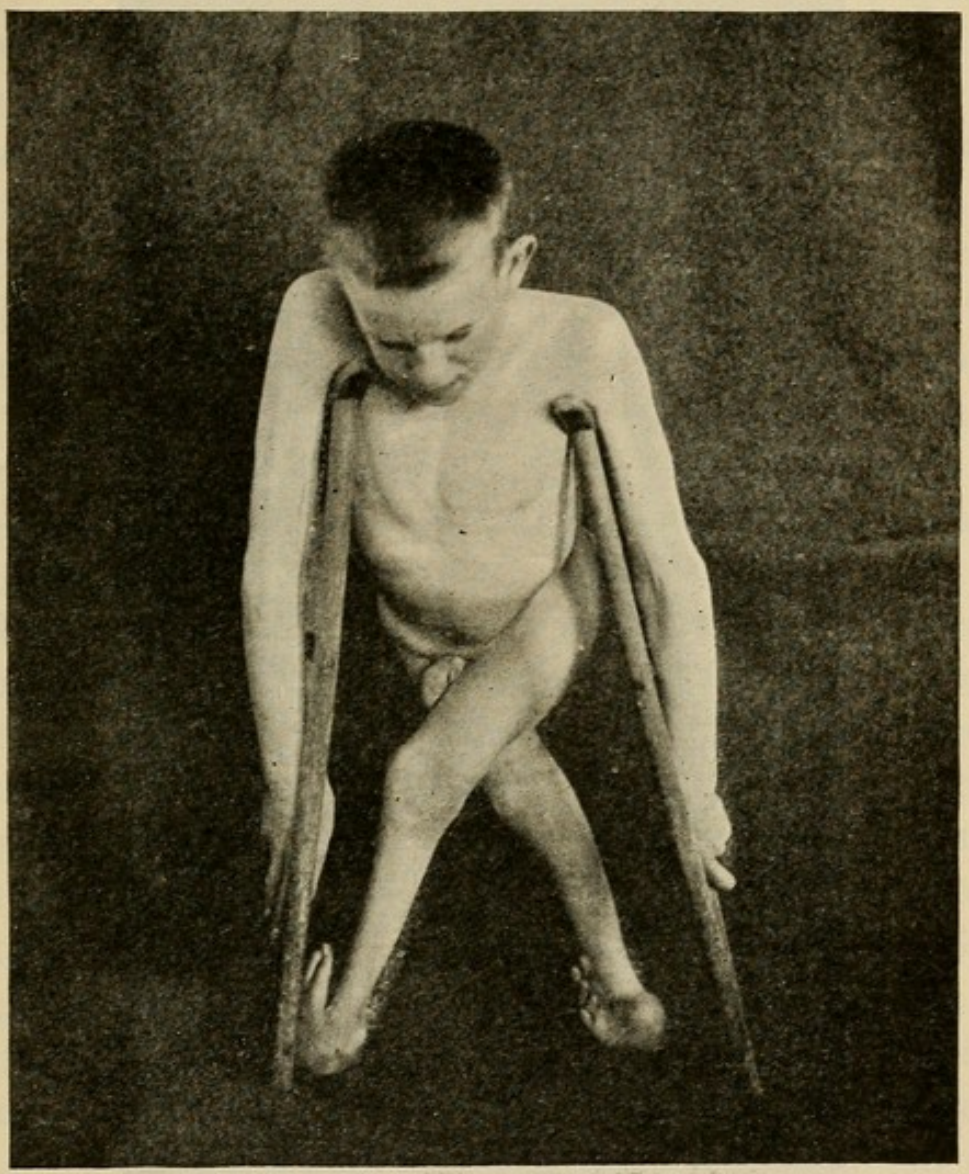


FIG. 78.

Cerebral diplegia ("crossed-leg progression").

(cerebral birth-palsy, infantile meningeal hemorrhage). This form of infantile cerebral palsy depends on meningeal hemorrhage over one or both sides of the convexity

of the brain or on its base, the result of injury during birth. This acute lesion is followed by chronic inflam-

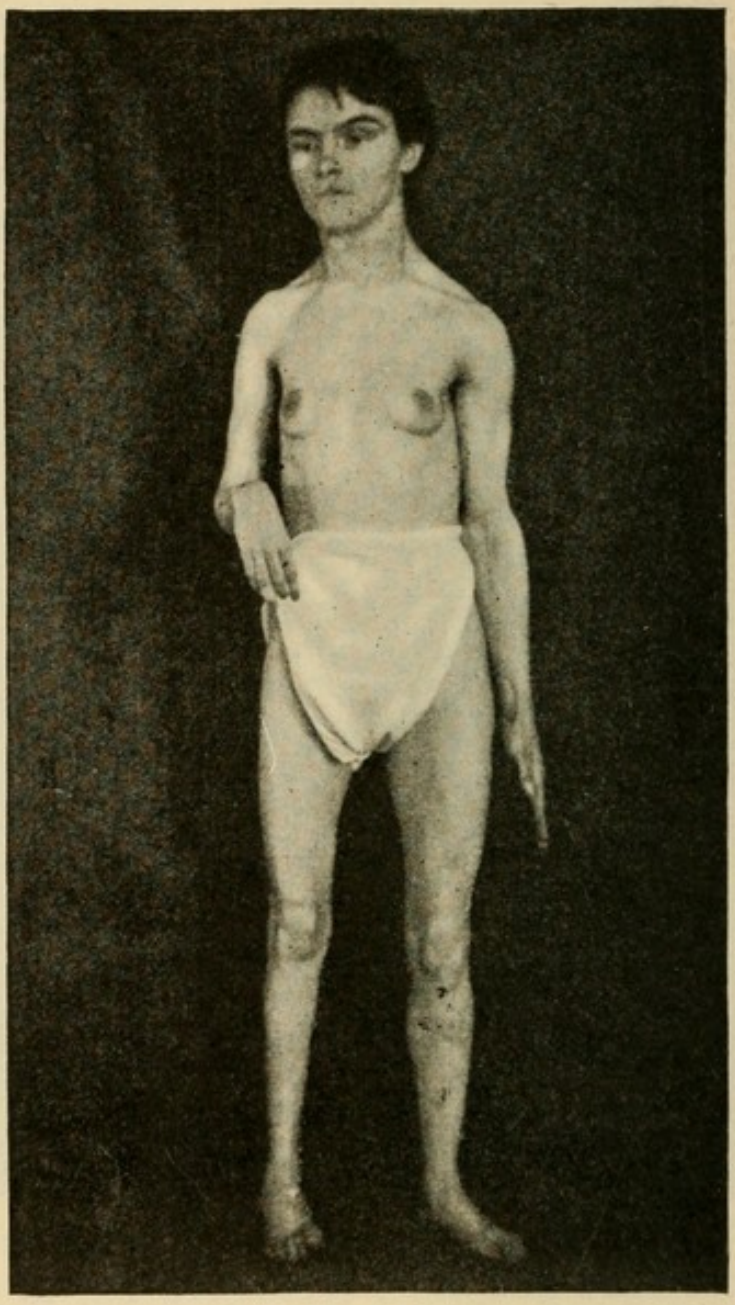


FIG. 79.

Infantile cerebral hemiplegia.

mation of the cerebral cortex and its membranes, finally ending in diffuse or lobar sclerosis, cystic degeneration,

and partial atrophy of the affected parts. In about two thirds of the cases the paralysis is of the hemiplegic form; in one third it is diplegic. The facts that indicate the existence of the congenital form of palsy are:

(1) A history of hemiplegia or diplegia only exceptionally without convulsions dating from birth or from a few days subsequent to birth,—though the paralysis may not have been observed for some time after parturition. There is never a history of distinct onset after birth.

(2) There is a history of some distinct difficulty in birth, either an unnatural presentation or more frequently prolonged and difficult labor with head presentation in primipara. Comparatively few instances can be attributed to forceps delivery alone. Precipitate delivery produces compression of the head similar to that occurring in prolonged labor.

The symptoms in these cases are those already enumerated: Partial paralysis, hemiplegia or diplegia with rigidity and contractures, arrested development, increased reflexes, post-hemiplegic movements, mental impairment, and frequently epileptic seizures. There is nothing distinctive about these symptoms or their combinations, and the diagnosis of this form of cerebral palsy depends on the etiological facts above stated and on the absence of a distinct onset of the symptoms after birth The deep reflexes on the paralyzed side, which are all abolished for a time after the stroke, are regained in a day or two and remain permanently exaggerated. The skin reflexes, on the other hand, are always abolished on the hemiplegic side. Dorsi flexion of the great toe always occurs on the paralyzed side when the sole of

the foot is stroked. The temperature of the hemiplegic parts is about a degree less than on the sound side. More or less marked aphasia occurs in lesions of the left hemisphere in right-handed individuals and in the right hemisphere in the left-handed.

(*III.*) *The Acquired Form of Infantile Cerebral Palsy* (Acute Cerebral Infantile Palsy, Infantile Hemiplegia). There is considerable difference of opinion as to the pathological process which underlies the acquired form of infantile cerebral palsy and it is certain that this is not the same in all cases. The following morbid processes have been found to underlie the condition: Meningeal hemorrhage (most frequent); thrombosis of superficial cortical veins from syphilitic endarteritis and from atheroma; hemorrhage, especially into the cortex, or pia, or subarachnoid space; cerebral embolism; and last but not least, an inflammatory process known as polioencephalitis, involving the motor cortical ganglion cells and supposed to be analogous to the process in the spinal cord known as poliomyelitis. There are good reasons to think that each of these processes may at times be effective in causing infantile hemiplegia, but in the case of thrombosis of cortical veins and particularly of polioencephalitis this supposition is as yet based chiefly on theoretical considerations. The terminal states of the processes just enumerated are the same as those in the congenital form. Aside from a slight temporary awkwardness in its use the tongue itself is rarely paralyzed.

In the acquired form of infantile cerebral paralysis the form of the paralysis is hemiplegic in fully nine tenths of all the cases; diplegias and paraplegias are rare as compared with their occurrence in the congenital form.

The recognition of this form (acquired) of cerebral paralysis depends on the following facts :

1. The onset of the paralysis is acute or sudden, and occurs during infancy or early childhood. In about one half the cases the onset is attended with convulsions, usually one-sided, sometimes general. There may or may not be loss of consciousness lasting hours or days, and there may or may not be fever and vomiting at the onset.

2. In nearly half the cases the disease is distinctly secondary to some other condition. In these cases the exact period of onset of the paralysis may be obscured by the symptoms of the primary disease.

The conditions to which the acquired form are most often secondary are as follows : pneumonia,[1] scarlet fever, measles, whooping-cough and injury to the head, but it occasionally follows almost every acute specific or general disease.

The distinction between the two above-described forms of infantile cerebral paralysis is usually easy, and may be made from the facts already given relating to their etiology and mode of development.

The diagnosis between infantile cerebral palsy and other forms of brain disease is also easy as a rule. The hemiplegia that occurs in tumor is distinguished from that of acute infantile cerebral palsies by the gradual onset and progressive development of the symptoms of the former as distinguished from the acute onset and stationary or retrogressive character of the symptoms in the latter.

Typical cases of infantile cerebral paralysis cannot be confounded with typical cases of poliomyelitis—infantile spinal paralysis—but both are large groups, the

[1] Very extensive subarachnoid hemorrhage is sometimes seen in the cases that develop during pneumonia.

variations within which are such that cases are met with in each that bear a considerable though superficial resemblance to one another. A case of infantile cerebral paralysis in which there is considerable paralysis and atrophy in one arm (the leg having recovered partially or entirely), may closely resemble a case of poliomyelitis involving one arm, and the resemblance may be heightened by the history, in both cases, of an acute onset with fever and convulsions. In such an instance the diagnosis would depend (chiefly) on the electrical reactions, which would be normal, or nearly so, in the cerebral case, but would present some form of RD in the spinal case.

In the so-called paraplegia form of infantile cerebral paralysis the palsy may possibly be confounded with that from disease of the spinal cord. Close examination, however, shows, that there is usually some motor derangement in the upper extremites in the former cases. It is important also to note that the chronic spastic paraplegia that results from spinal-cord disease is exceedingly rare in young children. If vertebral caries be excluded, such paraplegia is almost invariably due to a cerebral lesion. (See Primary Spastic Paraplegia.)

Abscess of the Brain.—The symptoms of abscess of the brain are exceedingly variable and often equivocal. They consist, first, of *diffuse* symptoms, which are much like those of tumor (see p. 375)—headache, vomiting, optic neuritis and mental apathy; and, secondly, of *focal* symptoms, which depend entirely on the situation of the abscess and are even more variable than the diffuse symptoms. In some cases the focal symptoms are entirely wanting. In many instances there are in addition to the diffuse and focal symptoms the general symptoms due to suppuration—fever and repeated rigors. In about three fourths of all the cases there is present a

distinct and recognizable cause of cerebral abscess, such as trauma, caries of cranial bones, chronic ear disease, or suppuration elsewhere, as in the lung.

Of the focal symptoms, *hemiplegia*, often incomplete, is the most constant, occurring as it does in about half of all cases of cerebral abscess. Local convulsions are less common. They are usually associated with hemiplegia, and both symptoms depend on the location of the lesion near or in the motor path. When there is a considerable degree of paralysis of one side there is almost always a great exaggeration of the knee-jerk on this side usually with very pronounced ankle clonus[1] and Babinski's sign.

The course of abscess of the brain is exceedingly variable and necessitates the recognition of two groups of cases: first, cases which run an acute course, and, secondly, cases which run a chronic course.

1. *The acute cases* are those that commence with well marked cerebral symptoms—headache, vomiting, etc.,—which progress rapidly, and without distinct diminution, into a stage characterized by severe general cerebral symptoms—headache, vomiting, optic neuritis, general convulsions, and mental apathy, delirium and coma; perhaps also by focal symptoms—hemiplegia and local convulsions. This is the terminal stage. The entire course of the disease varies in these acute cases from a week to a month.

2. *The chronic cases* are those in which there is a long period of latency between the initial and terminal

[1] Ankle clonus, we believe, is a very regular symptom in cases of cerebral abscess involving the motor path, and is often seen in cases of abscess of the frontal lobe reaching back and slightly encroaching upon the motor path. Violent clonus may be obtainable where there is very slight motor paralysis.

cerebral symptoms, the former being often so slight as to be entirely overlooked.

This period of latency may last weeks, months, or years, and may be complete or incomplete. If incomplete, there may be present some equivocal cerebral symptoms, as headache, occasional vomiting, convulsions, slight mental failure, apathy, etc. Then, after a variable time, all these symptoms increase, and pass, gradually or rapidly, into a terminal stage like that above described, characterized by headache, convulsions, optic neuritis, delirium and stupor deepening into coma. If the latency is complete there are no recognizable evidences of brain disease until the terminal stage of grave cerebral symptoms is suddenly or rapidly reached.

In those cases of abscess in which there are well-marked general and local cerebral symptoms and symptoms indicative of a suppurative process—fever and repeated rigors, in the presence of a cause (*vide ante*) that is adequate to account for a cerebral abscess—the diagnosis is not difficult. In those cases, however, in which the period of latency is complete, or nearly so, and the terminal symptoms come on suddenly or rapidly, a diagnosis of apoplexy from cerebral hemorrhage or acute cerebral softening may be erroneously made. Such an error may be avoided if a recognizable cause for abscess exists and leads to a suspicion of abscess. Hemorrhage and softening could be positively excluded if examination revealed the existence of optic neuritis in the absence of chronic diffuse nephritis.

The two diseases with which abscess is most often confounded are tumor and meningitis. The points of distinction from the former are given on page 379. The diagnosis between abscess and meningitis has been given elsewhere (see p. 343).

Intracranial Tumor.—The symptoms that point to the existence of a tumor within the cranial cavity are of two kinds—diffuse or general and focal. The most significant and frequent *diffuse* symptoms are : (1) headache—severe and persistent ; (2) vomiting—especially without gastric cause and (3) double optic neuritis. Other diffuse symptoms of importance are : (1) general convulsions ; (2) attacks of petit mal ; (3) giddiness ; (4) slowness of speech ; (5) simple mental failure ; and as terminal symptoms, (6) stupor and coma. The *focal* symptoms, unlike the diffuse symptoms, depend on the situation of the tumor and afford an indication of its position. Some of the focal symptoms are irritative in character, others destructive. The symptoms and their combinations that depend on the position of the tumor within the cranium may be inferred from the facts of localization already presented (see Chap. III). The focal symptoms of tumor that are of most frequent occurrence are : (1) motor paralysis, usually of local commencement (monoplegia) with subsequent extension (hemiplegia or partial hemiplegia) ; (2) local spasm—corresponding to the seat of the motor paralysis ; (3) aphasia—usually motor, sometimes sensory ; (4) disturbances of sensibility ; (5) paralyses of cranial nerves.

An exceedingly important general characteristic of all these symptoms of tumor is that they are of *gradual onset* and run a gradually *progressive* course. The gradual onset is as distinctive of tumor as is the sudden onset of vascular disease—cerebral hemorrhage and acute cerebral softening. It is important to note, however, (1) that the course of the tumor is not usually uniformly progressive—periods of intermission often alternating with periods of progress ; and (2) that sometimes symptoms increase rapidly in intensity by causing inflamma-

tion in neighboring structures ; and (3) that some forms of tumor (glioma) may cause symptoms of sudden onset from rupture of vessels within their substance.

When the diagnosis of tumor has been made and the position of the growth determined by the rules of localization already given (*vide ante*), it remains to fix upon the nature of the growth. Sometimes this can be done with a high degree of probability, or even with certainty ; more often the diagnosis of the nature of the tumor can be narrowed down to one of two possibilities ; and more often still the data afford a basis for nothing more than a guess.

Syphilomata, tubercular growths, sarcomata (including gliomata), and carcinomata probably make up at least nine tenths of all intra-cranial growths. In other forms of tumor it is impossible in a very large majority of cases to make a pathological diagnosis. The diagnostic effort is therefore practically limited to the cases included in the first group.

The indications that suggest that the tumor is a *syphilitic growth* are as follows :

1. A distinct history or symptoms of constitutional syphilis.[1]

2. Symptoms indicating that the tumor is in the cortex of the brain (see Localization), and is producing irritative rather than destructive phenomena.

3. Evidences of rapid growth at the onset, followed by a period of slow growth or a stationary condition.

4. Retrogression and gradual arrest of the symptoms of a tumor under antisyphilitic treatment (iodide and mercury).

[1] A history of a chancre without secondaries has a similar significance. A syphiloma cannot, of course, be excluded, unless the possibility of syphilitic infection can be also excluded. It must be remembered that a growth of another nature may coexist with syphilis.

5. The development of the tumor during early or middle adult life (twenty to forty-five years). This is, of course, of little value, as most forms of tumor are most common at this period.

The indications that suggest a *tubercular growth* are:

1. The presence of tubercular disease in some other organ than the brain, or a family history of tubercular disease.

2. A rapid development of the symptoms, which after a time become stationary.

3. Symptoms indicating that the tumor is in the cerebellum or in the pons.

4. The development of the symptoms before the twentieth, and especially before the tenth year. This indication refers also to glioma.

5. A history of injury to the head.

6. Improvement under tonic treatment.

The indications that suggest the existence of a *sarcoma* are: (1) the presence of a sarcomatous tumor elsewhere (this makes the probability very high); evidence that the tumor is not in the brain substance itself, as when it is in the bones (rarely available).

The following indications suggest a *glioma*:

1. The occurrence of sudden loss of consciousness, with exacerbation of all symptoms, in the course of a tumor. This is practically confined to glioma.

2. The situation of the tumor in the cortex, with predominance of irritative symptoms. This character is shared with syphiloma.

3. The absence of all evidence of tubercular and syphilitic disease and of sarcoma or carcinoma in other parts of the body.

4. The age of the patient is under fifty. If over fifty, glioma is improbable.

A *carcinoma* is suggested by :

1. The presence of carcinoma elsewhere. This makes the diagnosis highly probable.

2. By the patient's being over fifty years of age.

Tumor of the brain may be confounded with other organic diseases of the brain, with functional diseases of the nervous system and with diseases outside the nervous system.

Diagnosis from Other Organic Diseases of the Brain.—All organic diseases of the brain may be roughly but conveniently divided into two great groups ;—that in which the symptoms are of sudden onset, and that in which the onset of the symptoms is more or less gradual. The first group comprises a large and very important part of all organic brain diseases—cerebral hemorrhage, and the various forms (*vide ante*) of acute cerebral softening. Cerebral tumor is distinguished from the members of this group of vascular lesions, by the simple but important fact that its symptoms are never of sudden onset and never reach a considerable degree of development in the course of a few hours. In rare cases of tumor (especially cases of glioma), sudden symptoms suggesting a vascular lesion occur, but these symptoms (which are, indeed, due to hemorrhage from rupture of vessels in the tumor) always follow symptoms of more gradual onset. It is therefore necessary to consider only the distinction of tumor from the diseases that comprise the second group above mentioned—in which the development of the symptoms is more or less gradual. This group includes the following conditions : meningitis, acute and chronic, abscess, aneurism, general paralysis, insular sclerosis and bulbar paralysis.

For the diagnosis between tumor and meningitis, see page 342.

Diagnosis between Tumor and Abscess.—Cases of abscess occur that bear a close resemblance to tumor. The diffuse symptoms, especially, are often those of tumor —there is headache, vomiting, double optic neuritis and mental failure. If there are well marked and progressive focal symptoms—as monoplegia, hemiplegia, paralysis of cranial nerves, etc., this is in favor of tumor. The absence of focal symptoms points to abscess. If there is a considerable grade of optic neuritis, this is somewhat in favor of tumor, and if the optic neuritis is intense (six dioptrics or more), the diagnosis of tumor is practically certain. On the other hand, a period of considerable duration in which the symptoms are slight and stationary, followed by a period of rapid increase in the severity of the symptoms, is in favor of abscess. The occurrence of fever and rigors, especially if associated with exacerbation of the symptoms, points to abscess. If severe symptoms of rapid onset gradually lessen, this is in favor of tumor. The etiology may give important aid in the distinction. The absence of any traceable cause for the disease is distinctly in favor of tumor if the symptoms are equivocal. If there is ear disease, or disease of the cranial bones, or a distant focus of suppuration, this makes abscess more than probable. If, however, there has been an injury to the head, this gives no help, for although injury is more commonly a cause of abscess than of tumor this is compensated by the fact that tumor is a more common condition than abscess.

The diagnosis between tumor and the cases of hysteria that resemble it is ordinarily very simple; yet such cases are not rarely mistaken for tumor, and even more often cases of tumor are regarded as instances of hysteria. These errors would seldom or never be made were the facts relating to the distinction of functional and organic

disease elsewhere considered (see Chapter VI.) borne in mind. Only the main points of distinction will be considered here.

The cases of hysteria that are mistaken for tumor are usually those in which there are well marked unilateral manifestations,—local paralysis, monoplegia, or hemiplegia, contractures, spasm, local anæsthesia or hemianæsthesia. There may be in these cases a considerable degree of headache, there may be repeated vomiting, and there may be general convulsions—each of which increases the resemblance to tumor. The peculiar distribution of the hysterical paralyses and anæsthesias, their rapid onset after some emotional disturbance, the absence of local convulsions and of unilateral changes in the reflexes, the absence of optic neuritis and the variations that occur in the hysterical symptoms make the distinction easy, especially in patients showing the mental characteristics of hysteria. The error of ascribing the symptoms of tumor to hysteria is generally made in young women who have an inclination to emotional disturbance, which, in the presence of organic disease has led to the development of distinct hysterical symptoms. In other words, in such a case the symptoms of tumor are associated with and perhaps somewhat obscured by the existence of true manifestations of hysteria. Error can always be avoided by remembering that no symptom or symptoms should be attributed to hysteria until the various evidences of organic disease have been eliminated by a careful search. In tumor of the brain there are always some distinct indications of organic disease—as optic neuritis, focal paralysis, local convulsions, etc., which it is inexcusable to overlook.

Cerebral tumor may be confounded with idiopathic epilepsy in cases where the tumor runs a slow course and

general convulsions constitute a conspicuous symptom. The convulsions of tumor usually have a local commencement and this is an important point of difference from those of epilepsy, but does not constitute an absolute criterion, since the local commencement may be absent in tumor and is in rare cases present in epilepsy. An aura, particularly a special sense aura, preceding the attacks is rather in favor of tumor. But it is rarely necessary to base the distinction on these features; other evidences of organic disease are usually present, —*e. g.*, there is optic neuritis, or there is organic headache, or local paralysis. In the absence of more unequivocal indications, a neurotic heredity, as of epilepsy, chorea, or hysteria, may be allowed some weight as increasing the probabilities of functional disease.

The error of ascribing a combination of optic neuritis and headache in cases of extreme anæmia, Bright's disease or lead poisoning, to an organic cause (and especially to tumor), has already been mentioned (see optic neuritis), and does not require further consideration here.

Intracranial Aneurism. — The term intracranial aneurism as here employed relates only to aneurisms of the main cerebral arteries, and not to the miliary aneurisms that occur on their branches within the brain substance. Miliary aneurisms give rise to no symptoms until rupture occurs, and then the symptoms are those of cerebral hemorrhage. Intracranial aneurisms, when they attain a considerable size and press upon important structures, give rise to symptoms,—differing in this respect from miliary aneurisms. But it is important to note that, in many instances, intracranial aneurisms, like miliary aneurisms, cause no symptoms until they rupture.

The symptoms of aneurism, when they occur, are those

of a small tumor at the base of the brain, pressing usually either upon the motor tract or upon the cranial nerves, or upon both. A certain diagnosis of the presence of aneurism can be made only in cases where there is an aneurismal murmur that is distinguishable on auscultation of the skull. Such a murmur is heard only in rare cases of intracranial aneursim, and probably only in cases where the aneurism is situated either on the internal carotid or the vertebral artery. Hence a positive diagnosis of aneurism can be made only in very exceptional instances. The cases are, however, much more numerous where a suspicion of aneurism is justified, or where even a probable diagnosis can be made. The indications that make the diagnosis of aneurism more or less probable in the presence of symptoms of a basal tumor are as follows :

1. The history or presence of a cause of aneurism which is not also a cause of new growths within the cranium. Such causes are (*a*) arterial degeneration and (*b*) endocarditis. Arterial degeneration, fibroid or atheromatous, is sometimes a cause of aneurism in persons over forty. In many cases of aneurism occurring in persons under forty years of age there are indications of past or present endocarditis. The significance of this circumstance depends on the fact that many cases of aneurism depend on embolism. Syphilis, by causing endarteritis, is a cause of aneurism, but a history of syphilis has no diagnostic value, as specific disease is more frequently a cause of syphiloma or chronic syphilitic meningitis than of aneurism. A history of injury (which is frequent in aneurism) is likewise of no value, because injury is a cause both of aneurism and tumor.

2. Indications that the tumor (lesion) is in the position of a large artery. The most common positions for aneurism are, first, on the middle cerebral artery, next on the

basilar and internal carotid arteries. Much less often aneurisms are situated on the anterior cerebral, posterior communicating, anterior communicating, vertebral, or posterior cerebral arteries. The focal pressure symptoms that result from an aneurism in these different positions may be inferred from the relations of the vessels to surrounding parts, but the symptoms that result from aneurism on the middle cerebral, basilar, and internal carotid arteries may be briefly summarized as they comprise two-thirds of all cases of intracranial aneurism:

a. Middle Cerebral.—Hemiplegia and convulsions are common—paralysis of cranial nerves rare. When on the left side there may be motor aphasia.

b. Basilar.—The symptoms are those due to pressure, unilateral or bilateral, on the pons and on the cranial nerves. The nerves affected are generally several and vary according to the position of the aneurism on the artery. The 5th, 6th, 7th, 8th, 9th, 10th, and 11th may be affected and usually in the frequency of the order named, only rarely the 3d or 12th. There may be severe occipital headache. There may be crossed paralysis. There are no symptoms in about one-third of all cases. Convulsions are uncommon.

c. Internal Carotid.—The chief pressure symptoms are blindness of one eye from pressure on the optic nerve, combined with paralysis of one or more ocular nerves—especially ptosis—from pressure on the nerves in the cavernous sinus. Smell may be lost and occasionally there may be symptoms of pressure on one crus.

In any case where an intracranial aneurism is suspected the diagnosis is much strengthened if rupture occurs, which usually happens in one-half the cases. Such a rupture gives rise to the symptoms of severe apoplexy. If there have been no symptoms to suggest aneurism

before rupture occurs, the cause of the apoplexy would be suspected only in case it occurred in a person under forty years of age who had suffered from endocarditis or syphilis, or who gave a history of trauma to the head.

The chief conditions from which aneurism has to be distinguished are intracranial tumor and chronic syphilitic basilar meningitis. The points on which the distinction from tumor is made, when this is practicable, have been mentioned above. The question of a diagnosis from chronic syphilitic meningitis arises in cases where there is pressure only upon cranial nerves. The distinction is frequently impossible, especially if there is a distinct history of syphilis. The chief criterion in such instances is that afforded by the effect of antisyphilitic treatment : in chronic meningitis there is regularly improvement under treatment ; in aneurism there is none. But this indication, while valuable, is far from absolute.

Nuclear Ophthalmoplegia (Nuclear Ocular Paralysis).—Disease of the nuclei of the motor nerves of the eye (third, fourth, and sixth) is often the cause of paralysis of the ocular muscles—both the external muscles of the eyeball (extrinsic muscles of the eye) and the muscles within the globe (internal or intrinsic muscles). In some cases the paralysis is the result of involvement of an isolated nucleus ; in others, several or all of the nuclei are affected. Some of the lesions that cause nuclear paralysis are acute in their development ; others are chronic. Thus it is that the symptoms of nuclear ophthalmoplegia vary widely in different cases, according to the position and nature of the morbid process.

It is desirable to distinguish clinically two different forms of ophthalmoplegia—an acute form and a chronic form. Owing to its far greater importance the chronic form will be first considered.

Chronic Nuclear Ophthalmoplegia (Chronic Progressive Ophthalmoplegia,[1] Chronic Nuclear Paralysis, Chronic Ophthalmoplegia).—The symptoms of chronic nuclear ophthalmoplegia vary so widely in different cases that it is difficult to say what constitutes the most typical form of the disease. The following are the chief characteristics of the disease :

1. *The gradual and successive paralysis of many or all of the ocular muscles.*

At first only a small number of muscles (or one only) is affected. One of the first indications of this involvement is often double vision (usually transient). The loss of power is at first slight, but gradually increases and extends to other muscles than those first affected. After the lapse of years most of the muscles of both eyes become involved, and even all of them may be included in the paralysis (total ophthalmoplegia). The isolated loss of reflex action of the iris and the isolated paralysis of the ciliary muscle are to be regarded as varieties of chronic progressive ophthalmoplegia. Usually the muscles are involved in a random irregular manner without reference to their function (this is somewhat characteristic of nuclear disease), but not rarely the muscles first affected are associated in action (as the external rectus of one side and the internal rectus of the other, or both internal recti, or the superior recti and levators). Ptosis is usually absent, and when present is rarely complete. Nystagmus and slight exophthalmos are occasional symptoms. The process is sometimes much more advanced in one eye than in the other, and occasionally the paralyses are all unilateral. The intrin-

[1] Chronic progressive ophthalmoplegia is so commonly caused by nuclear disease that the term is used synonymously with nuclear disease.

sic muscles of the eye, though commonly affected in some degree, may escape entirely.

Most cases of chronic ophthalmoplegia are slowly progressive, though in some cases there is little progress for many years. Occasionally cases which are unquestionably of nuclear origin, after reaching an advanced stage of paralysis in both eyes, regain perfectly the power of movement in both eyes. Such cases cannot at present be satisfactorily explained.

2. *The association of these ocular paralyses with evidences of other diseases of the nervous system.*

Sometimes chronic ophthalmoplegia exists by itself, but more often it is associated with other forms of nervous disease. Of these other forms the most common are locomotor ataxia, general paralysis of the insane, multiple sclerosis and optic-nerve atrophy. Psychical disturbance is a common association ; sometimes this is the precursor of the symptoms of general paralysis. In other cases the mental disturbance is apparently functional (hypochondriasis, hallucinatory insanity). The relation of chronic ophthalmoplegia to the associated condition varies in different cases. Either the nervous disease or the associated state may give rise to the first symptoms, and in some cases both sets of symptoms appear and develop almost simultaneously.

The lesion on which chronic nuclear ophthalmoplegia depends is a slow degenerative process in the nuclei corresponding to the muscles paralyzed. In a considerable proportion of these cases a syphilitic history can be traced, and is probable in many cases where it cannot be traced, but there are many cases in which no cause whatever can be found. Sometimes ophthalmoplegia is a congenital and perhaps an hereditary condition.

The diagnosis of chronic nuclear ophthalmoplegia will

be touched upon after briefly alluding to the acute form of the disease.

Acute Nuclear Ophthalmoplegia.—In rare instances many or all of the ocular muscles are suddenly or rapidly paralyzed from disease of their nuclei, and this condition is called acute nuclear ophthalmoplegia. In some cases the condition has been due to hemorrhage, in others to acute softening, in the region of the third nerve nuclei. Whether it is ever really due to acute inflammation of the nuclei (so-called acute polioencephalitis superior) is doubtful. Perhaps some cases of diphtheritic ocular paralysis (including the common form of paralysis of the ciliary muscle-cycloplegia) are examples of nuclear disease.

Having established the existence of paralysis of ocular muscles, an effort must be made to determine whether the lesion is of nuclear origin or of peripheral origin. In some cases a conclusion can be reached which is almost positive ; in other cases it is impossible to reach a conclusion. The indications upon which the diagnosis is based differ somewhat in acute and chronic cases of ocular paralysis, and it is convenient to consider them separately.

In *cases of acute development* a nuclear lesion is probable if the muscles paralyzed correspond to an irregular involvement of the functions of the nerve structures, or if the muscles paralyzed are very unequally involved. Thus, if several of the muscles innervated by the third nerve are paralyzed without reference to function (*e. g.*, the internal and inferior recti), it is highly improbable that the lesion is peripheral. Again, if all or nearly all the external muscles of the eyeball are involved, but some (as the levator) are much less paralyzed than the others, this *suggests* a nuclear lesion. If the intrinsic muscles of the eye escape entirely, while the external muscles are extensively involved, a nuclear lesion is almost certain.

All these indications except the last are greatly strengthened in significance if the palsy of the ocular muscles is *bilateral.* In acute or subacute palsies the associated symptoms may throw light on the position of the lesion. Thus, if the symptoms are such as to suggest a *quadrigeminal* lesion (see Localization), it is certain that the ocular palsy is nuclear. The probable nature of the lesion may aid in the diagnosis. Vascular lesions, for example (hemorrhage, acute softening), can give rise only to nuclear paralysis, or to *fascicular* paralysis, *i. e.*, paralysis involving the various bundles of the third nerve between the nucleus and the surface origin of the nerve. A distinction between a nuclear paralysis and a fascicular paralysis cannot at present be made, either in the case of acute or of chronic lesions. A chronic palsy is almost always nuclear and almost never fascicular, but an acute or subacute palsy is probably as often fascicular as nuclear. The involvement of all the functions of a nerve in almost equal degree indicates a peripheral lesion.

In cases of ocular paralysis of chronic development, the distinction of a nuclear (or fascicular) from a peripheral palsy is based on the following indications :

1. The exemption of the intrinsic muscles of the eye. This is a very valuable indication, but is not absolutely distinctive of nuclear disease.

2. The absence or slight development of ptosis.

3. Bilateral distribution of the symptoms.

4. Irregular, apparently random distribution of the paralysis.

5. The association of chronic ocular palsy with cerebral or spinal symptoms, especially symptoms that suggest a lesion of a degenerative character.

In cases where both the internal and external muscles of both eyes are paralyzed the diagnosis of a nuclear

lesion can be made only after excluding the presence of disease at the base of the brain (*vide* Localization).

Recurrent Ophthalmoplegia (Periodical Ophthalmoplegia).—In rare cases there is observed a transient paralysis of ocular muscles (chiefly those supplied by the third nerve of one side), which makes its appearance at long or short intervals during a long period of time. The character of the attacks varies somewhat in different cases. The onset is rapid and is usually attended with pain, generally severe, in the eye affected, and often there is unilateral headache and vomiting. The paroxysms thus closely resemble attacks of migraine with ophthalmoplegia superadded (*vide* Migraine). The paralysis is sometimes complete, sometimes incomplete. Ptosis is generally present, and the intrinsic muscles of the eye are often involved with the extrinsic muscles. The duration of the attacks varies much in different cases, and even in the same case at different times. Usually the paralysis lasts from two to six weeks, its duration being longest when the intervals are long. The frequency of the attacks also varies greatly in different cases—in most cases the interval between the paroxysms varies from one month to one year.

It is important to note that in some cases *slight loss of power persists during the intervals*. The nature of these cases of recurrent ophthalmoplegia is obscure, but there is good reason to believe that the condition is related, in many ways both clinically and pathologically, to migraine (ophthalmic migraine). The palsy probably depends not on organic changes, but on vascular and perhaps in some cases on nutritional changes in the nuclei.

Multiple or Disseminated Sclerosis.—Insular sclerosis is a degenerative disease of the central nervous system—of the cord as well as of the brain,—in which

patches of sclerosis occur irregularly scattered throughout its white substance. The favorite seats for the islets of sclerosis are the centrum ovale, crus, pons, medulla, and cord, but they may occur in almost any part, even in the peripheral nerves, and entail a corresponding multiplicity in the characters of the symptoms

The characteristic and almost distinctive feature of insular sclerosis is a coarse jerky inco-ordination (*vide* p. 103) of the limbs and especially of the arms. This inco-ordination is present only during voluntary effort ("intention tremor or action tremor"). It is associated usually from the beginning with some loss of power in the limbs affected, and this loss progresses gradually as the disease advances. The reason why this combination of jerky inco-ordination and weakness in the limbs is so constantly observed in cases of disseminated sclerosis is that the pons and cord are very regularly the seat of patches of sclerosis, some of which almost necessarily involve some part of the motor path.

Associated with the jerky inco-ordination and weakness are usually a variety of other symptoms of less constant occurrence. Of these the following are the most important:

1. Nystagmus. This is an early and frequent symptom (present in three fourths of all cases).

2. A peculiar defect in articulation, consisting usually of an undue separation and accentuation of the syllables of words (known as "scanning" speech), or of the words of a sentence.

3. A slight and variable degree of mental defect. This consists generally of loss of memory and an unnatural contentment with the patient's surroundings and condition.

4. Partial atrophy of the optic nerve with gradual loss of sight. Advanced atrophy of the optic nerve is met

with in only a small proportion of the cases, but a slight degree of change (follows on the temporal side of the disc) is present in a very large number of cases (*vide* page 174).

5. Increase of knee-jerks and of muscle reflexes generally is observed in most of the cases in which there is jerky inco-ordination. Babinski's sign is also present.

In addition to (or in place of) these cardinal symptoms of insular sclerosis, others of less frequent occurrence are observed, among them loss of knee-jerk, sensory disturbances, paralysis of cranial nerves—especially the sixth, seventh, and twelfth nerves—headache, vertigo, giddiness, and double vision.

Like other degenerative diseases, disseminated sclerosis is slowly progressive in its course, but it is not generally uniformly progressive, for periods of progress alternate with periods during which the disease appears stationary. Sometimes in the course of the disease there are acute attacks of vertigo and giddiness, associated occasionally with vomiting and accompanied at times with diplopia. After such an attack the symptoms of the disease are aggravated. These attacks usually occur early in the disease, and they may be the first symptoms observed.

Disseminated sclerosis may be confounded with paralysis agitans, locomotor ataxia, intracranial tumor, general paralysis, primary lateral sclerosis, infantile cerebral paralysis, and with some manifestations of hysteria.

The distinction from paralysis agitans is seldom difficult. The tremor of insular sclerosis is coarse, irregular, and jerky, and is present only during voluntary effort; that of paralysis agitans is usually regular, rhythmical, and relatively narrow in range and is continued during rest, except in rare instances in the beginning of the disease. In paralysis agitans the parts that are the seat of tremor

are early rigid and the hand assumes a distinctive attitude of flexion which is absent in insular sclerosis. In the cases where the tremor of paralysis agitans stops during rest, the carriage of the patient, the facies, the fixed gaze and the rigidity are sufficiently distinctive. The head frequently shares in the tremor of insular sclerosis; it is rarely affected in paralysis agitans. Nystagmus is common in insular sclerosis, unknown in paralysis agitans. In insular sclerosis the speech is usually "scanning" in character; in paralysis agitans it may be normal or unduly rapid, with a tendency to confluence of syllables, and a decided monotony of voice. It is very rare for insular sclerosis to commence after the fortieth year, while it is uncommon for paralysis agitans to begin before the thirtieth year. In cases in which tremor is the only marked symptom and the diagnosis seems doubtful, the presence of optic atrophy (usually slight in degree) points to insular sclerosis, and in less doubtful cases strengthens the diagnosis.

The distinction from locomotor ataxia is considered under the latter disease, and from general paralysis of the insane under that title.

A superficial resemblance exists between cases of tumor and insular sclerosis in the cases where tumor of the pons or crus (see Localization) is accompanied with wild jerky inco-ordination almost identical with that of insular sclerosis. There the resemblance stops, and it is hardly possible to confound the other characters of tumor (headache, optic neuritis, local spasm, local paralysis), with the picture of insular sclerosis that has been presented above (combination of tremor and weakness, with nystagmus, mental failure, defective articulation, and optic nerve atrophy).

The diagnosis between insular sclerosis and primary lateral sclerosis is considered under lateral sclerosis.

Infantile cerebral paralysis (diplegic form) occasionally presents symptoms that resemble closely those of insular sclerosis. There may be the wild jerky inco-ordination of the arms upon voluntary movement that is so suggestive of insular sclerosis. If the patient is between ten and twenty years of age, shows little evidence of paralysis, and only slight mental failure and slight speech defect, the distinction from insular sclerosis may not be easy. The most important help in making the diagnosis in such a case is the fact that in infantile cerebral paralysis there is usually a history of trauma during birth, or a history of a distinct *acute* onset, with or without trauma, or an acute specific fever as a factor after birth, while in insular sclerosis there is the history of a *gradual* development of the disease. In infantile cerebral paralysis there is further a history of distinct paralysis which has gradually disappeared. If no history of the mode of development of the disease can be obtained the distinction rests on the objective features of the case. In infantile cerebral paralysis (diplegic form) some of the evidences of the former paralysis are usually present (slight weakness, difficulty in performing fine acts with fingers, atrophy, arrest of development, contractures, etc.). There may be some cranial deformity, with a high arched palate. The mental defect of infantile cerebral paralysis is some grade of dementia; that of insular sclerosis is a slighter grade of simple mental failure combined with a state of undue complacency. The presence of nystagmus would make the diagnosis of insular sclerosis certain.

It is more common for insular sclerosis to be mistaken for hysteria than for the opposite error to be made, but

each mistake is occasionally made. Insular sclerosis may be mistaken for hysteria when the former disease occurs, as it frequently does, in young women who present some actual symptoms of hysteria.[1] Generally this error is avoidable. The mere fact of the existence of nystagmus indicates that the condition is not purely hysterical and strongly suggests organic disease. Sometimes there occurs in hysteria an intention tremor, irregular in character, and thus resembling that of insular sclerosis, but differing from it in being much narrower in range and more rapid, and in being associated with distinct voluntary rigidity of the antagonistic muscles involved. It is usually easy to recognize the combination of the symptoms of the two diseases when this exists, but it may be impossible to say whether certain symptoms belong to the account of the insular sclerosis or to that of the hysteria. Thus, in a case presenting spastic paralysis of the lower extremities, uncertain gait, nystagmus, scanning speech and partial atrophy of the optic nerve, all these symptoms would be explicable by the supposition of an insular sclerosis, but if there existed a persistent rhythmical tremor of one upper extremity combined with these symptoms it would be plain that this was not dependent on the organic disease but on an associated functional disturbance—*i. e.*, that it was of hysterical origin. If, however, there also existed in this instance concentric limitation of the visual fields and loss of sensibility of the extremities it would be difficult or impossible to fix on the correct interpretation of these symptoms, since they might be referable either to the organic or to the functional disturbance.

Bulbar Paralysis.—Progressive bulbar or glosso-labio-laryngeal paralysis is a chronic degenerative disease

[1] Probably no organic disease of the brain is so apt to have associated with it some manifestations, slight or considerable, of hysteria.

of certain nuclei of the medulla oblongata. Its symptoms depend on the locality of the lesion and as this is usually the same, the manifestations of the disease are reasonably constant. The great characteristic of the condition is a *slowly progressive* and *strictly bilateral* paralysis of the tongue, lips, palate, pharynx, and frequently of the larynx, often associated with visible wasting of the tongue and lips. The effect of this paralysis is to cause : (1) imperfect articulation of sounds in which the tongue plays an important rôle—especially lingual consonants, *l*, *n*, *r*, and *t* ; and (2) difficulty in swallowing, from weakness of the pharynx and palate.

After a period which varies in different cases from one to several years from the commencement of the disease, the patient falls into a characteristic condition of partial helplessness, from the progress of the above mentioned paralysis. In this terminal state the tongue lies motionless, the saliva dribbles from the open mouth, the lower part of the face is without expression and articulate speech is impossible. The intellect remains perfectly clear.

Not uncommonly chronic bulbar paralysis is associated with chronic degenerative processes in other parts. Of these associations the most common is progressive muscular atrophy (see p. 446). In some cases the bulbar symptoms, in others the spinal symptoms, make their appearance first and are chiefly conspicuous.

Besides this chronic progressive type of bulbar paralysis there are two other clinical forms of bulbar paralysis. These forms resemble the chronic form in presenting the same combination of glosso-labio-laryngeal paralysis of symmetrical distribution, but differ from it in being of *sudden or rapid onset.* The similarity in distribution of symptoms depends on a similarity in the position of the lesion ; the difference in the onset depends on a difference

in the nature of the lesion. In both the forms mentioned the lesion is a vascular lesion; in one it is hemorrhage, in the other inflammation. In the first form (hemorrhagic)[1] the symptoms come on suddenly, often with vomiting and giddiness, but usually without loss of consciousness. At first there may be some loss of power in the limbs and perhaps some sensory disturbance, but this wears rapidly away, leaving behind a stationary condition of glosso-labio-laryngeal paralysis. In the second form (inflammatory),[2] which is much more rare than the first, the symptoms come on rapidly (acutely) but not suddenly, and are accompanied with fever, vomiting, etc.[3]

The diagnosis of chronic bulbar paralysis is usually not difficult, for the distribution of the paralysis, with its bilateral symmetry and slowly progressive course, is highly distinctive. A tumor outside the medulla, damaging the nerve-roots, or in the substance of the medulla, may cause bulbar symptoms of slow onset resembling those of the degenerative disease. The diagnosis is based on the fact that tumors in either of these situations usually produce unilateral symptoms, or if they produce bilateral symptoms these have not the highly symmetrical distribution that distinguishes chronic bulbar paralysis. In tumor, again, there is usually headache, and often convulsions, and sometimes optic neuritis, all of which are absent in chronic bulbar paralysis. The age at which

[1] These are simply cases of hemorrhage into the medulla, which are symmetrical and occupy the same nuclei as are affected in the degenerative process. Much more often hemorrhage into the medulla is a random process.

[2] These cases are allied to cases of acute poliomyelitis.

[3] Neither form is of course to be regarded as a distinct disease, for the same processes that cause them are much more common in other regions.

chronic bulbar paralysis occurs may help in the diagnosis, for tumors are not frequent after the fiftieth year, while chronic bulbar paralysis seldom commences before this time.

Symmetrical lesions in the motor paths of both hemispheres may give rise to symptoms resembling those of bulbar paralysis, bilateral paralysis of tongue, lips and pharynx—"pseudo-bulbar paralysis" it is called. When such symptoms depend on acute lesions (hemorrhage, softening), as they usually do, the diagnosis is easy. When they depend on chronic lesions (tumor, chronic meningitis), the distinction may be more difficult, and is then based chiefly on the fact that in these cases there are usually evidences of involvement of other parts than the tongue, lips and pharynx, (face, arm, leg)—that there is in fact, or has been, a double hemiplegia. Another point of distinction is that in these cases there is no atrophy of the lips and tongue, and that reflex action of the pharynx is never impaired. But these are not absolute criteria.

General Paralysis of the Insane (General Paresis or Paralytic Dementia).—The symptoms of general paralysis of the insane are exceedingly varied, but in all cases there are present evidences of both mental and physical derangement. Sometimes the mental symptoms, sometimes the physical symptoms, are first prominent. Either set of symptoms may be especially obtrusive during the course of the disease, or both sets may be equally conspicuous.

The mental symptoms that are most characteristic are as follows:

1. Loss of mental power, as shown by defective attention, defective memory, clouded consciousness, impair-

ment of judgment, and early mental fatigue in the performance of ordinary duties.

2. Change in character, as shown by irritability, indifference to the wants or sufferings of others as well as to personal interests, defective will power, with partial or entire loss of moral sense, and silly fabrications.

3. Unsystematized and varying delusions, especially delusions of grandeur, in which the patient believes himself to be some celebrated personage (*e.g.*, Napoleon, Cæsar, Christ), or to be possessed of enormous wealth, or of great personal strength, etc. These exaggerated and variable personal delusions are not usually present during the earliest periods of the disease, but are rarely absent after it has become established. They are always associated with, and generally preceded by, a period in which the patient has an exalted sense of well-being, which, when present, is highly characteristic. Less than one third of the cases present the state of typical exaltation. Depressing delusions are quite as often met with as expansive ideas. As a matter of convenience in more exact clinical description, the demented, agitated, depressed, and expansive forms of the disease are diagnosticated. Side by side with these mental changes are developed a series of physical phenomena of which the most distinctive are as follows :

1. Hesitating and monotonous speech with a tendency to slur the endings of words.

2. Tremor on voluntary movement, especially marked in the upper extremities and in the lips during articulation ; fibrillary tremor in the tongue when protruded, and in the muscles of the face during speech or during excitement.

3. Pupillary changes, especially marked inequality, myosis, and loss of light reaction.

4. Exaggeration of the wrist-, elbow-, and knee-jerks.

The symptoms above enumerated are very constant, the majority of them making their appearance in all cases of general paralysis. In addition to these there are others of considerable importance that are much less constant, but are liable to make their appearance at almost any period of the disease. These symptoms are both mental and physical, and comprise the following phenomena :

1. Melancholia or hypochondriasis, in which headache is prominent. These are usually early symptoms.

2. Maniacal outbreaks.

3. Sudden epileptiform attacks.

4. Apoplectiform seizures characterized by sudden loss of consciousness and hemiplegic symptoms, which latter usually disappear quickly.

In about six per cent. of the cases the symptoms of posterior sclerosis are combined with the symptoms of general paralysis (*vide* p. 462). In such cases the knee-jerks are lost instead of being exaggerated, there are lightning pains, and ataxia is a prominent symptom.

General paralysis, like other progressive degenerative diseases, is not uniformly progressive. Periods of remission, often of considerable duration, alternate with periods of progress, but there are great differences in the duration of the disease (one to six years). In all cases where death does not occur in the acute stages of the disease (apoplectic attacks), or from intercurrent disease, their termination is in complete dementia and physical helplessness. In a fully developed case of general paralysis there are practically no difficulties in diagnosis; the

conspicuous mental failure and characteristic expansive or depressive delusions, in combination with tremor, speech disturbance, etc., make the nature of the disease only too evident. In the early stages of the affection the case is very different; the symptoms may be so equivocal that a positive diagnosis is not possible. The conditions with which general paralysis may be confounded are: (1) neurasthenia, (2) chronic alcoholism, (3) chronic meningitis, (4) tremor, (5) cerebral lues, (6) senile dementia, and (7) the sequelæ of sunstroke. The diagnosis from neurasthenia may be impossible at an early period, but a diagnosis can generally be made when the patient is first seen by the physician. In neurasthenia there may be some mental failure—loss of memory, defective attention, etc., and there may be tremor in the upper extremities, but the mental failure is never so considerable in degree as in general paralysis, and the patient is not only conscious of its existence but exaggerates its importance, while in general paralysis he is only rarely cognizant of the existence of any mental weakness. The neurasthenic is able to concentrate his attention sufficiently to write correctly at dictation. The general paralytic is frequently unable to do this ; he omits letters or transposes them in a way that indicates defective attention, although he may be making a pronounced effort to write correctly. If in addition to mental weakness and irritability there is speech disturbance and tremor on movement of hand and lips, there is a strong presumption that the condition is general paralysis and not neurasthenia, and this is true *a fortiori*, if there is evidence of delusions or if there are immobile pupils.

Hydrocephalus (Dropsy of the Brain).—The word hydrocephalus is employed to designate an accumulation

of serous fluid within the skull. When the accumulation is in the arachnoid space the condition is termed external hydrocephalus ; when within the ventricles, internal hydrocephalus. Hydrocephalus may run an acute or chronic course, may be congenital or acquired, and may be apparently primary or distinctly dependent on some other morbid process.

Acute Hydrocephalus is usually dependent on tubercular meningitis, and owing to the frequency with which some degree of serous accumulation accompanies cases of meningitis the terms acute hydrocephalus and meningitis have been used synonymously. Clinically acute hydrocephalus is of secondary importance to the meningitis on which it depends and of which it is merely one symptom. The serous effusion depends, in acute hydrocephalus, on inflammation of the pia, ependyma, and choroid plexuses.

Chronic External Hydrocephalus is a condition of little clinical interest and may be passed by. *Chronic Internal Hydrocephalus*, on the other hand, is a condition of frequent occurrence and considerable importance. It occurs as a congenital and as an acquired condition. Congenital internal hydrocephalus develops during intra-uterine life. After birth the cranium enlarges rapidly, the face remaining of normal size. The fontanelles become large and prominent, the sutures separate widely and the cranium attains a characteristic globular form. There is usually much mental defect. Death usually occurs after a few months, but in some instances the process becomes stationary and the patient reaches adult life, though with considerable mental weakness and perhaps disorders of motion.

The acquired form of chronic internal hydrocephalus

usually occurs without known cause. Sometimes, however, it is apparently secondary to a morbid process which causes effusion mechanically by the obliteration of the

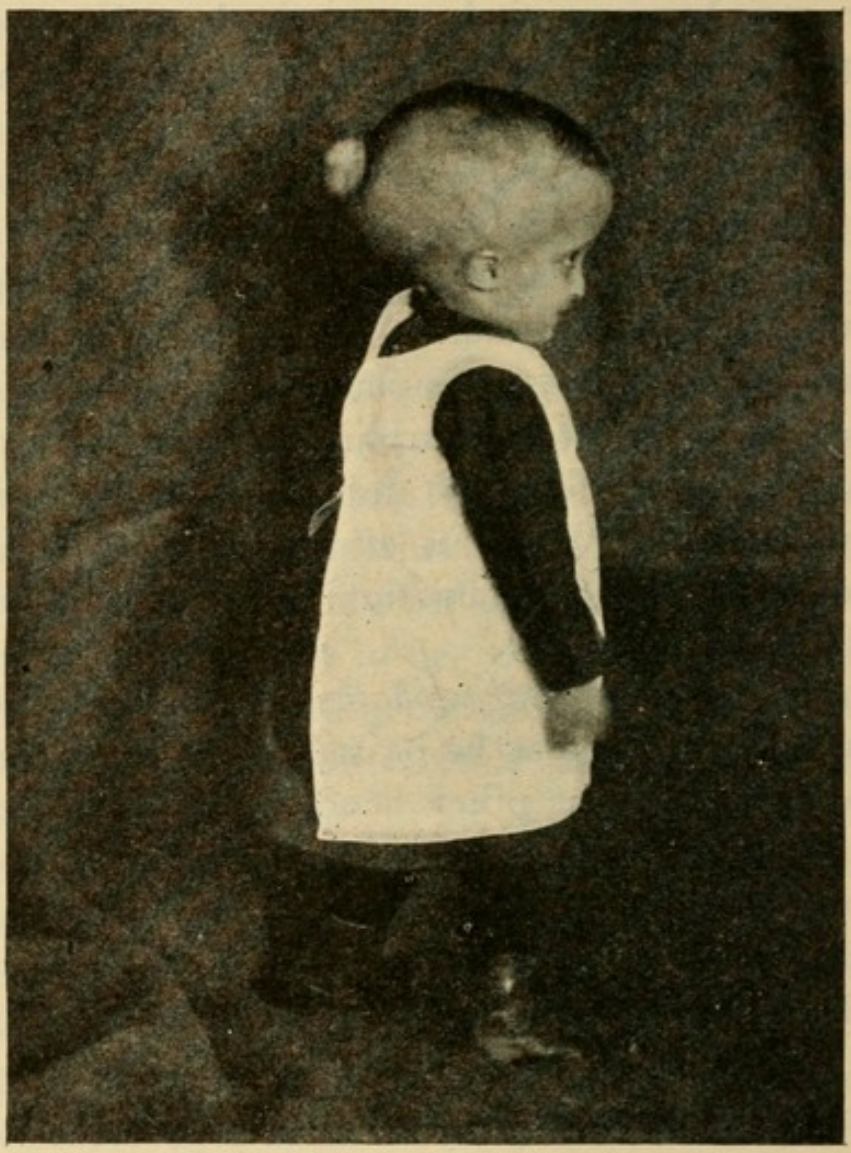


FIG. 48.

Hydrocephalus and Meningocele.

openings[1] through which the ventricular fluid ordinarily passes. Tumors, chronic syphilitic meningitis and acute

[1] It is thought that closure of the openings in the membrane closing the fourth ventricle from the subarachnoid space is the most important mechanical cause of hydrocephalus, but the entire subject of causation is involved in obscurity.

meningitis are the chief conditions by which this result may be brought about. Both primary and secondary forms of acquired chronic internal hydrocephalus occur in children and in adults. In children the symptoms are much the same as in the congenital form. In adults there are no distinctive symptoms.

A positive diagnosis of hydrocephalus can be made only in cases where there is decided and progressive enlargement of the cranium, and in such cases there is rarely any diagnostic difficulty. When the enlargement of the cranium is not considerable hydrocephalus may be confounded with the enlargement of the head that occurs in rickets. The head in hydrocephalus has a globular form, and the fontanelles are always tense. In the typical rachitic head the cranium has a pyramidal shape, there is no bulging of the fontanelles and other evidences of rickets are plain enough. It must not be forgotten, however, that many cases of rickets are associated with hydrocephalus,—the nature of the relation between the rachitic and the hydrocephalic process being but imperfectly understood,—and cases occur in which it is difficult to determine the true character of the head enlargement. The most important indication of a hydrocephalic process is the rapidity of the cranial enlargement and the existence of a bulging fontanelle. When the bones are firmly united, as in adults, the diagnosis of hydrocephalus is extremely difficult and usually impossible. A positive diagnosis is possible only in the rare cases in which there is distinct enlargement of the head and separation of the sutures. But in the absence of such enlargement hydrocephalus may perhaps be *suspected* if mental weakness, somnolence, coma, convulsions, loss of power, etc., follow very slowly on one or more attacks of acute meningitis. In very rare instances general thickening of the cranial bones may be

confounded with the enlargement of the head that occurs in hydrocephalus. External and internal hydrocephalus may sometimes be distinguished by the introduction of an exploring syringe and the withdrawal of fluid in the former case from the arachnoid space, in the latter from the ventricles, but even this method is not infallible.

Diseases of the Spinal Cord.

Spinal Meningitis.—The individual symptoms of spinal meningitis vary considerably in distribution, according to the position of the disease, and in their character, according to the acuteness of the process. In most cases there are present certain local symptoms, as pain, spasm, rigidity, hyperæsthesia, anæsthesia, motor paralysis, etc., and it is convenient to think of these symptoms according as they are due (1) to irritation of the membranes, (2) to irritation of the nerve-roots, (3) to compression of the nerve-roots, or (4) to compression of the cord. The chief symptom of irritation of the inflamed membranes is pain in the back, usually constant and increased by movement. The chief indication of irritation of the nerve-roots are radiating pains and hyperæsthesia from irritation of sensory (posterior) roots, and rigidity and spasm or contractures from irritation of the motor (anterior) roots. When the nerve-roots are compressed instead of irritated, there are analgesia and anæsthesia instead of pain and hyperæsthesia, and muscular paralysis and relaxation in place of rigidity and spasm. If the compression of the nerve-roots is considerable there are trophic changes (atrophy) in the muscles paralyzed, with loss of reflex action. The symptoms of involvement of the cord by pressure resemble those of a transverse lesion of the cord (*vide* page 245) : paralysis of motion and

sensation below the level of the lesion, increased reflex action, etc.

It is convenient to distinguish the following clinical forms of spinal meningitis :

(1) External meningitis, (2) Internal meningitis, (*a*) Acute, (*b*) Chronic.

External Meningitis (External Spinal Pachymeningitis). —External meningitis is probably secondary, in all cases, to adjacent disease, and the particular form of disease to which it is almost invariably secondary is caries of the spine (Pott's disease).

The symptoms of the disease vary considerably, according as the process runs an acute or chronic course, and also according as the symptoms of caries (its usual cause) are obtrusive or inconspicuous.

In acute cases the symptoms of external pachymeningitis closely resemble those of internal meningitis (*vide* internal meningitis). In such cases the following are the chief indications of the disease :

1. Pain in the back, increased by movement.
2. Rigidity of the muscles of the spine.
3. Radiating pains in the extremities, with or without spasm.
4. Hyperæsthesia of the skin.
5. Fever, often considerable in degree.

These symptoms may exist alone, or may be combined with symptoms of pressure or inflammation of the cord itself, especially paraplegia or paraplegic weakness, and excessive reflex action in the lower extremities when the pressure is above the lumbar enlargement.

The symptoms indicating a more chronic process differ from those above enumerated chiefly in the absence of considerable fever and in the less obtrusive character of the irritative symptoms, especially muscular rigidity

and spasm. In very chronic cases there are no distinctively meningitic symptoms. The symptoms are those of the bone disease. The distinction of external meningitis from internal meningitis is impracticable from the symptoms alone. In order to establish the diagnosis it is necessary to consider the etiology of the affection with much care, with a view to discovering evidence of a condition to which the meningitis may be secondary. The presence of caries of the spine makes the diagnosis certain; but as it is easy to overlook the slighter evidences of such disease (local tenderness, irregularity of the spines), caries cannot be positively excluded unless the spine has been repeatedly and carefully examined, including examination by the X-rays. In the majority of cases of external meningitis the evidences of caries are sufficiently distinct to enable the diagnosis of the character of the meningeal affection to be made; in a few cases only is the diagnosis impossible.

Acute Internal Meningitis.—The symptoms that point to the existence of acute internal meningitis are essentially those of the acute form of external meningitis already described (p. 403): pain in the back, rigidity of the muscles of the spine, radiating pains in the extremities, with hyperæsthesia, and spasm brought on especially by movement. The onset of these symptoms is acute, and is generally attended with fever, sometimes by rigors. The grouping of the symptoms enumerated varies with the situation of the disease. When the meninges of the lumbar region are chiefly involved, the pain and spasm and hyperæsthesia are confined to the lower extremities. When the dorsal meninges are chiefly affected, the symptoms extend to the muscles and skin of the trunk. When the cervical meninges are the seat of the disease the symptoms affect the arms, and there

may be bulbar symptoms, as dyspnœa and difficult swallowing.

Acute internal spinal meningitis may be complicated by cerebral meningitis, or the symptoms of cerebral meningitis may precede those of the spinal disease. The symptoms indicating extension to the cerebral meninges are vomiting, headache, delirium and paralysis of cranial nerves (*vide* acute meningitis). The character of the inflammation, whether purulent or tubercular, does not entail any distinctive difference in the character of the symptoms. The duration of the acute symptoms varies from a few days (severe cases) to several weeks. In cases that recover, or in which death does not occur early, the symptoms grow gradually less acute. This subacute stage may endure for months, and may pass into a chronic stage.

The etiology of acute internal spinal meningitis does not usually give much help in the diagnosis of the affection. The disease may follow exposure to cold, overexertion, trauma, sunstroke, acute febrile diseases, septicæmia, etc., but the influence of some of these factors is uncertain, and all of them are related to many other conditions of the nervous system besides meningitis. The principal affections of the nervous system from which acute internal spinal meningitis must be distinguished are acute myelitis, cerebro-spinal meningitis, meningeal hemorrhage and tetanus. In acute myelitis paraplegia occurs early ; in meningitis it is absent, and there is at first little or no motor weakness of any kind. Moreover, the spasm of the limbs and pain in the back, which occur in meningitis, are not present, although there is often rigidity of the limbs in the later stages of myelitis if the lesion is above the lumbar enlargement. When meningitis and myelitis coexist, the decision into which category the

condition belongs must depend on the greater obtrusiveness of the cord symptoms on the one hand, or of the meningeal symptoms on the other, and upon the order in which these symptoms have made their appearance. The distinction from meningeal hemorrhage is given under this disease (p. 409). The diagnosis from tetanus depends on the occurrence in meningitis of fever at the onset, and of marked sensory symptoms, and on the occurrence in tetanus of trismus and the ready excitation of spasms by peripheral impressions.

Chronic Internal Meningitis. — The symptoms of chronic internal meningitis, like those of acute internal meningitis, depend on meningeal irritation, on irritation and pressure of the nerve-roots, and on damage to the cord itself. The character of the symptoms varies much in different cases, according as one or other of these mechanisms in the production of the symptoms is predominant.

In the majority of cases the following are the symptoms observed in chronic internal meningitis :

At first—1. Pain or discomfort in the back.

2. Stiffness of the muscles of the back (retraction of the head when the meningitis is cervical).

3. Radiating pains in the arms, legs, or trunk, according to the position of the disease.

4. Hyperæsthesia.

Followed by—5. Loss of sensation in the regions formerly hyperæsthetic.

6. Weakness in the muscles formerly rigid, with atrophy.

7. Diminished pain.

8. Symptoms of compression of the cord (*vide* p. 420).

In cases where the process of disease affects mainly the pia mater (chronic leptomeningitis), the symptoms that

are due to meningeal irritation are especially conspicuous, *i. e.*, there is marked pain in the back, the radiating pains are severe, and rigidity and hyperæsthesia are prominent symptoms. The cord itself may be involved in these cases, as shown by paraplegic weakness, etc.

In the cases where the inflammatory process affects chiefly the internal surface of the dura mater (chronic internal pachymeningitis,) the symptoms that depend on involvement of the nerve-roots passing through the dura are especially obtrusive. There are at first severe radiating pains, spasm and hyperæsthesia from irritation, and later muscular weakness and muscular atrophy from pressure of the nerve-roots.

When the seat of the disease is the cervical region ("cervical hypertrophic pachymeningitis"), as it sometimes is, the irritative symptoms consist chiefly of pains in the arms, shoulders and neck, and these are followed by muscular atrophy, often extensive, in the arms, and by paraplegic weakness below this level, from cord pressure.

The etiology of chronic internal meningitis is usually too obscure to be of much aid in diagnosis. It is important to remember, however, that the disease sometimes follows chronic alcoholism, syphilis, long exposure to cold and traumatisms, and also that it may be a sequel of the acute form of spinal meningitis, and hence must own the same causes.

In the cases of chronic meningitis where irritative symptoms predominate, the condition may be confounded with muscular rheumatism and rachialgia, or, if the meningitis is of the lumbar region, with locomotor ataxia. The resemblance to muscular rheumatism is never more than superficial. In both conditions there may be periph-

eral pains, but in muscular rheumatism there is never the spinal pain, the hyperæsthesia, or the rigidity that characterizes chronic meningitis. The resemblance to rachialgia may be closer ; spinal pain and rigidity of the muscles of the back are common to both conditions. In rachialgia, however, there is tenderness of a large part of the spine, and there are often local points of great tenderness along the spine. On the other hand the rigidity and radiating pains of meningitis are absent in rachialgia.

When chronic meningitis affects the cervical region, the rigidity of the muscles which it causes, and the pain, may be mistaken for wry-neck. In wry-neck the head is fixed by muscles that are in spasm. In chronic meningitis the head is fixed owing to the pain to which movement gives rise.

The chronic localized pachymeningitis which so often results from bone disease has many symptoms in common with chronic internal meningitis, and the distinction can be made only by discovering a cause of external meningitis. In every case of chronic meningitis the evidences of bone disease, local tenderness of the spine and deformity, should be carefully sought for.

Intra-Spinal Hemorrhage.—1. Hemorrhage into the meninges of the cord.

2. Hemorrhage into the spinal cord.

Meningeal Hemorrhage.—The symptoms of hemorrhage into the membranes of the cord are much the same whether the hemorrhage is within the dura mater or external to it. In either case there is considerable irritation of the meninges and nerve-roots at the level of the lesion. In consequence there are the following irritative symptoms : (1) Severe pain in the back ; (2) radiating pains in the nerves coming from the region of

the meninges involved ; (3) muscular spasm, which may include local or general convulsions. These symptoms resemble closely the symptoms of acute spinal meningitis, but differ from them in one important respect, *i. e.*, they are always sudden and often violent in their onset.

In many cases paralytic symptoms follow very quickly upon these irritative symptoms, in consequence of pressure upon the cord itself. In such instances there is some loss of power and anæsthesia below the level of the lesion, with increased reflexes if the pressure is located above the lumbar enlargement. The paraplegia is rarely complete.

The precise distribution of the symptoms enumerated varies with the level of the lesion (page 245). When this is in the cervical region, the pains are in the arms and neck, there is retraction of the head, and there may be evidences of interference with the functions of the medulla—embarrassed respiration, dysphagia, mydriasis, etc. If the dorsal region is involved, there are severe pains about the chest or abdomen. When the lumbar meninges are the seat of the hemorrhage the pains are in the legs and there is diminished sensation and loss of power in the legs, loss of patellar reflex and sphincteric weakness. If the cauda equina is involved, there are often characteristic localizing symptoms (*vide* page 251).

In some cases of meningeal hemorrhage there is temporary loss of consciousness from shock ; usually the mind remains clear.

For purposes of diagnosis it is important to note that injury to the spine, direct or indirect, is the great cause of meningeal hemorrhage. Very rarely it has resulted from prolonged and severe muscular exercise or from the rupture of an aneurism of the aorta into the vertebral canal after erosion of the vertebræ.

Meningeal hemorrhage may be confounded with meningitis, myelitis, tetanus, and with hemorrhage into the cord.

Meningitis differs from meningeal hemorrhage in that the symptoms which are similar do not come on suddenly, and in the presence of fever from the commencement. It is important to note that meningitis may be secondary to meningeal hemorrhage, and that both conditions then co-exist. In tetanus the symptoms are never of sudden onset, and severe radiating pains do not occur. On the other hand, trismus, an early and regular symptom of tetanus, is absent in meningeal hemorrhage. The tetanic spasm that occasionally occurs in newly born children is due to meningeal hemorrhage from trauma, and not to true tetanus.

Hemorrhage into the Substance of the Spinal Cord.— Hemorrhage into the substance of the spinal cord is rare. The indications that point to the occurrence of hemorrhage into the substance of the spinal cord are the symptoms of a lesion of the spinal cord, of sudden onset and unaccompanied by loss of consciousness. Those symptoms which are of most constant occurrence are: (1) paraplegia or paraplegic weakness with anæsthesia of corresponding distribution; (2) loss of control over the sphincters; (3) pain in the legs or trunk. The height to which the motor and sensory paralysis extends and the precise character of the loss of power over the sphincters depend upon the level of the lesion (*vide* p. 236). The condition of the reflexes is similarly variable. At the onset or soon after the onset of the symptoms there may be general convulsions or clonic spasm of the paralyzed muscles. Occasionally there is brief loss of consciousness at the onset. After a few days the symptoms of myelitis (secondary) develop, and there is a rise in temperature and perhaps an extension of the symptoms.

As with meningeal hemorrhage, the chief cause of hemorrhage into the cord is injury. Occasionally the condition is preceded by exposure to cold, violent exertions, or repeated coitus, and sometimes, but rarely, it is of spontaneous occurrence.

Hemorrhage into the cord can hardly be confounded with meningeal hemorrhage. Both conditions have one important feature in common, *i. e.*, the sudden onset of the symptoms, but in meningeal hemorrhage irritative symptoms take the lead and predominate, while in hemorrhage into the cord the symptoms are essentially those of a destructive and not an irritative process. Hemorrhage into the cord may occur in the course of myelitis (hemorrhagic myelitis), but in such cases, although the symptoms of hemorrhage are sudden in their onset, they are preceded by symptoms that point to myelitis and not to hemorrhage. The premonitory symptoms are often slight, but if they are of longer duration than a few minutes they should be regarded as evidences of primary myelitis, to which the hemorrhage is secondary. The presence of fever at the time of the symptoms of hemorrhage is strongly indicative of the secondary character of the hemorrhage. From all other organic diseases of the cord the sudden onset of hemorrhage into the cord is in itself sufficiently distinctive.

Myelitis.—The clinical forms of myelitis requiring separate consideration are based upon (1) the mode of onset of the myelitic process, and (2) upon the distribution of the lesion. When the symptoms of myelitis come on rapidly and attain their full development in the course of a few days or weeks, the process is *acute;* when the onset extends through a few weeks or months, the process is *subacute;* and when several months elapse in its development, it is spoken of as *chronic.* According to

the distribution of the lesion the following forms are recognized: (1) those in which the gray substance is chiefly or exclusively involved—*poliomyelitis;* (2) those in which both gray and white matter are involved, including (*a*) *diffuse or general myelitis*, in which a considerable part of the cord is diseased; (*b*) *transverse myelitis*, in which the entire transverse extent of the cord is inflamed; (*c*) *focal myelitis*, where the lesion is confined to one small area; and (*d*) *disseminated myelitis*, in which there are many foci of inflammation scattered irregularly through the cord.

For clinical purposes it is convenient to recognize the following forms of myelitis:

A. Acute Myelitis, including transverse, diffuse, focal, and disseminated myelitis.

B. Chronic Myelitis.

C. Acute Poliomyelitis (Infantile form).

D. Subacute Poliomyelitis (Form in adults).

Acute Myelitis.—The symptoms that point to the existence of acute myelitis are rapidly developed indications of interference with the chief functions of the spinal cord. The degree and extent of these symptoms vary much with the degree and extent of the myelitis. The following are the phenomena observed in transverse myelitis,[1] which may be taken as the type of the disease:

1. Motor paralysis, of rapid but not sudden onset, below the level of the lesion, partial or complete in degree.

2. Loss of sensation, partial or complete, below the level of the lesion.

3. Hyperæsthesia and girdle pain at the level of the lesion.

[1] It will be noted that these are essentially the symptoms enumerated under total transverse lesions of the spinal cord (*vide* Localization).

4. Loss of reflex at the level of the disease ; increase below this level.

5. Loss of control over the sphincters.

6. Trophic changes in the skin and muscles, varying with the level of the lesion.

7. Vaso-motor disturbances.

8. Changes in the tone of the paralyzed muscles—rigidity, spasm, contracture, or loss of tone, flaccidity, etc., according to the level of the lesion.

According as the lesion is in the cervical, dorsal, or lumbar region of the cord, the combination and distribution of the symptoms enumerated vary in many important respects (see Localization, transverse lesions). In cervical myelitis there is paralysis of motion in the arms, legs and trunk, with corresponding sensory disturbance, increased skin reflexes in trunk and legs, excessive tendon reflexes in lower extremities, rigidity in the parts below the lesion, retention of urine with subsequent incontinence and incontinence of fæces. If the lesion involves a considerable vertical extent of the cervical cord, there may be considerable muscular atrophy in the upper extremities. The pupils may be contracted and there may be optic neuritis.

The symptoms that indicate dorsal myelitis are practically those of cervical myelitis, minus those that relate to the involvement of the arms, and excepting also the pupillary changes and the rarer optic neuritis.

The symptoms that point to involvement of the lumbar enlargement are paraplegia, loss of sensation in the lower extremities, incontinence of urine and fæces from the first, loss of reflex action, and atrophy, with RD and loss of muscle tonus, in the lower extremities.[1]

[1] Atonic paralysis of the lower extremities with loss of control of the sphincters and loss of knee-jerks is occasionally observed in acute myelitis, not only of the lumbar but also of the cervical region of the

In the majority of cases of acute myelitis the focal symptoms that have been enumerated are preceded or associated in their development with constitutional symptoms, such as characterize most acute inflammatory processes—malaise, chilliness, or signs of prostration, and fever, slight or considerable in degree. The distinctively spinal symptoms are rapid in their onset and reach a high degree of severity in the course of a few days. Irritative symptoms, such as clonic spasm, or even general convulsions (especially in children), radiating pains, and pain in the spine, may attend the onset, but the evidences of irritation are soon displaced by those of destruction. After the symptoms of the onset have attained their height, the distribution of the symptoms may remain stationary, or there may be an upward or downward extension of symptoms of cord destruction.

The diagnosis of the vertical extent of the lesion is determined by indications that are elsewhere discussed (*vide* page 245). If the vertical extent is slight, the case is one of *transverse myelitis ;* if it is considerable in extent, it is one of *diffuse myelitis.* The recognition of *disseminated* foci of myelitis is often difficult. When possible, it depends on symptoms of interference with the functions of the cord as a central organ, at different levels —*i. e.*, on the presence of muscular atrophy and RD or loss of reflex, of such distribution as may be caused by foci at different levels and cannot be explained by the presence of a single lesion. Cases of anterior *poliomyelitis* are cases of focal myelitis ; but in addition to

cord. The symptoms are usually of rapid onset and soon change in character, the paralysis becoming of the spastic type, the knee-jerks returning and growing excessive, and the sphincteric control becoming re-established. The initial flaccid type of paralysis may be due to inhibition from the irritative lesion in the cord (see crush of spinal cord)

this form of focal myelitis cases sometimes occur in which there are both sensory and motor symptoms of unilateral distribution and acute onset, and these belong to the category of acute myelitis. When symptoms of commencing acute myelitis are followed by great extension and severer symptoms of cord interference of *sudden onset*, the exacerbation is probably due to hemorrhage (*hemorrhagic myelitis*).

The symptoms of subacute myelitis are essentially those of acute myelitis, with the difference that they reach their development more slowly and are unaccompanied with such marked constitutional disturbance at the onset.

The etiology of acute myelitis is not sufficiently distinctive to be of much aid in diagnosis. The process may follow exposure to cold, over-exertion, sexual excess, syphilis, chronic alcoholism, acute febrile diseases, traumatism of various kinds, and adjacent inflammatory conditions (as acute internal meningitis, etc.), and rapid or slow compression.

When the vertical and transverse extent of the lesion has been determined by a consideration of the distribution of the motor and sensory paralysis, the state of reflex action and the muscular irritability, the next question to decide is whether the process is primary or whether it is secondary to compression. This may be done by finding or excluding the causes of compression of the cord, especially bone disease (*vide* Compression of Cord).

The diseases with which primary acute myelitis may be confounded are hemorrhage into the cord, poliomyelitis, meningitis, meningeal hemorrhage, cauda equina hemorrhage and neuritis, multiple neuritis, acute ascending paralysis and hysterical paraplegia.

The distinction of myelitis from hemorrhage into the

cord is based mainly on the *sudden* onset of the symptoms in the latter disease as contrasted with the rapid but not sudden onset in the former. In hemorrhagic myelitis the symptoms become suddenly much aggravated, but the presence of slight premonitory symptoms as fever, numbness, tingling, etc., lasting more than a few minutes, render the distinction from primary hemorrhage easy. Speaking generally, the symptoms of meningitis are those of tissue irritation ; the symptoms of myelitis are those of tissue destruction in the cord. The irritative symptoms (pain, spasm, rigidity) that are so prominent in meningitis, are absent in myelitis, and thus afford a simple diagnostic distinction, but it must not be forgotten that myelitis and meningitis often co-exist, and that in these cases the order and relative intensity of the symptoms determines the category to which they belong.

Myelitis is readily distinguished from meningeal hemorrhage by the absence of marked irritative symptoms and the gradual or more sudden development of symptoms indicating a destructive cord lesion. When myelitis occurs in young females, especially those of hysterical leanings, the affection may be regarded as a hysterical paraplegia. In lumbar and cervical myelitis such a mistake can scarcely occur, but in dorsal myelitis the absence of muscular atrophy in the legs and the presence of increased knee-jerks somewhat favor the error. The presence of a girdle pain, of incontinence of urine and fæces, or of trophic changes, makes the presence of organic disease exceedingly probable. The presence of ankle clonus, as well as increased knee-jerks, is in favor of organic disease, but does not in itself settle the question. If there be, in addition to clonus, strong extensor spasm in the legs and Babinski's sign, the organic nature of the disease is probable.

Three conditions allied clinically with acute myelitis remain to be noticed under this head. They are : (1) Abscess of the Spinal Cord ; (2) Embolism of the Cord ; and (3) Caisson Paralysis.

Abscess of the Spinal Cord is an exceedingly rare condition. When it occurs it is associated with purulent spinal meningitis, and the symptoms are those of an acute irritative lesion of the cord followed rapidly by those of a destructive lesion. When fully developed the symptoms are essentially those of acute transverse myelitis. The distinction from myelitis, when this is possible, is based on the presence of a cause of *septic* suppuration, and not on the symptoms themselves. The disease has followed pulmonary gangrene and also gonorrhœa. It is said also to occur after severe injury to the vertebræ without a wound. In most of the cases recorded the abscess was in the lumbar enlargement. There may be several foci of suppuration at different levels of the cord, and abscess of the cord may coexist with that of the brain.

Embolism of the Spinal Cord.—Sudden paraplegia occasionally occurs in persons who have organic heart disease, under circumstances that suggest that embolism of the spinal cord may be the cause of the symptoms. In order to make the diagnosis of embolism of the cord with any degree of certainty in a given case, it is necessary (1) that the focal symptoms should have been of *sudden* onset ; (2) that the patient should have organic heart disease ; and (3) that there should be evidence of embolism elsewhere, in the kidney, spleen, or some part of the brain.

Caisson Paralysis ; Diver's Paralysis ; Paralysis from Lessened Atmospheric Pressure.—Men who work at a considerable depth beneath the water, and are exposed to an

atmospheric pressure greater than twice that in which they ordinarily live, are liable to become suddenly or rapidly paraplegic soon after coming back to the surface, especially if such pressure is too suddenly reduced. The paralysis generally comes on within a few hours after the return to the surface. There are usually premonitory symptoms, such as pains and tingling in the legs, with slight weakness. Then the paralysis of the legs develops rapidly or suddenly. In severe cases the sphincters are involved, and there is anæsthesia of the lower extremities. If the paralysis is partial, recovery occurs in a few days; if complete, the paralysis persists weeks or months with partial or complete recovery, or it may terminate fatally within a few days or weeks of the onset. The diagnosis of this condition presents no difficulties if its occurrence is known, for its etiology is distinctive. Symptomatically this form of paraplegia resembles both that from hemorrhage and that from myelitis. The pathological condition which underlies it is obscure. In its terminal stage the lesion resembles myelitis.

Chronic Myelitis.—The symptoms of chronic myelitis vary, as do those of acute myelitis, with the situation of the myelitic process. Motor paralysis, sensory paralysis, atrophy with RD, rigidity and contracture may all be present in chronic myelitis, but the intensely irritative symptoms of acute myelitis, severe pain and acute spasm, do not occur. In all cases the development of the symptoms is slow and gradual, and the symptoms do not attain a high degree of development until many months have elapsed.

The most common form of chronic myelitis is that which involves the dorsal region. The symptoms in such cases are paraplegic in distribution. There is loss of power in the legs, developing very gradually, paræsthesia, slight or considerable loss of sensation in the

legs, increased knee-jerks and later ankle clonus and spastic gait. There is often some impairment of the power of the sphincters. According to the transverse situation of the lesion, the symptoms may be quite symmetrical or much more marked on one side than on the other. When the lumbar or cervical enlargement is invaded, muscular atrophy with RD is added to the other symptoms. It is most important to remember that chronic myelitis is often a "random" process—that is, a process which damages in an irregular way the motor, sensory, reflex, and trophic functions of the spinal cord, without any tendency to confine itself to structures that subserve a particular function. The most important conditions from which chronic myelitis has to be distinguished, are slow compression of the cord and primary lateral sclerosis. In all cases of slow compression of the cord there are nerve-root symptoms, at first of an irritative character. These symptoms, which comprise pain and acute spasm, are inconspicuous or absent in chronic myelitis.

Compression of the Spinal Cord.—Compression of the spinal cord may be of rapid or sudden development, or it may be gradual in its progress. Rapid or sudden compression of the cord is almost always dependent upon injury which causes fracture or fracture-dislocation in some part of the spine, or which causes hemorrhage into the membranes or cord. The symptoms in these cases are the symptoms of a partial or total transverse lesion of the cord, and the distribution of the symptoms, in a given case, depends on the level of the cord sustaining the compression (*vide* Localization). The cord is often lacerated as well as compressed in cases of fracture-dislocation.

Slow compression of the cord may be caused by a

number of different pathological conditions, which are separately considered elsewhere. It is convenient to indicate here the symptoms common to most cases of slow compression, without special reference to its causes. Two classes of symptoms are observed in every case of slow compression of the cord: (1) root-symptoms, and (2) cord-symptoms. The root-symptoms are those that depend on irritation and compression of nerve-roots at the level of the compression. They include the following:

(*a*) Pain, sharp and neuralgic, in the distribution of the roots involved. These pains may occur in the arms or legs, thorax or abdomen, according to the seat of the disease.

(*b*) Hyperæsthesia of the skin followed in course of time by anæsthesia. The pains persist notwithstanding the existence of anæsthetic areas, and this condition is called *anæsthesia dolorosa.*

(*c*) Muscular weakness of gradual development, accompanied with atrophy and RD. The distribution of these symptoms depends, of course, on the level of the cord affected.

To these root-symptoms may be added spasm in the muscles that undergo wasting, but this is unusual. In every case of slow compression, cord-symptoms are after a time superadded to the root-symptoms. These cord-symptoms are the effects of imperfect conduction of impulses through the cord itself, and include the following:

(1) Motor paralysis in the parts below the level of the lesion, usually of slow, sometimes of rapid, development.

(2) Increase of superficial and deep reflexes below the lesion.

(3) Paræsthesiæ, numbness, tingling, formication, sometimes diminished sensibility, and often delay in the conduction of painful stimuli from parts below the lesion.

(4) Defective control of the sphincters.

Both root-symptoms and cord-symptoms are commonly bilateral from the first, but in cases where one side of the cord suffers before the other, the symptoms may be for a time chiefly or even entirely unilateral.

The slow and gradual development of the combination of cord- and root-symptoms, above enumerated, justifies the diagnosis of slow compression of the spinal cord. The presence of marked local tenderness of the spine at a level corresponding with the supposed seat of compression is a highly corroborative symptom. The chief conditions that give rise to such compression are caries of the spine, new growths of the spine, new growths of the dura or pia spinalis, thickening of the dura and compression by an aneurism which has eroded the bones and pressed on the spinal membranes. Of these various causes of compression spinal caries is by far the most common.

Crush of the Spinal Cord.—The spinal cord is frequently crushed in consequence of injury causing fracture-dislocation of vertebræ (usually cervical vertebræ or lower dorsal vertebræ). The cases in which this occurs are of course surgical in nature, but important questions of a medical character arise in connection with them, which have a direct bearing on prognosis and the question of operative treatment.

The most important thing to determine in any case of crush of the cord is the position and extent of the injury to the cord. The vertical level of the injury and its vertical extent may be determined with the aid of the facts already presented in connection with total transverse and unilateral lesions of the cord (see p. 241).

Another aid in the determination of the level of the lesion of the cord is the position of the bone lesion, as shown by the external evidences of damage to the vertebræ. The transverse extent of the injury to the cord is much more difficult to estimate. It is, however, very important that an effort be made to determine this, for on it depends, in large degree, the prognosis of the case. In making this effort it is essential to consider especially: (1) the state of motor power; (2) the state of sensation; (3) the state of the reflexes (particularly the knee-jerks); and, in the case of damage to the lower part of the cord, (4) the state of the sphincters of the bladder and rectum. Partial motor paralysis below the level of the lesion indicates that the laceration of the cord involves only part of its tranverse extent.[1] This view would be borne out by the absence of sensory symptoms in the parts below the lesion, or by the slight degree of such symptoms, and by the preservation or exaggeration of the knee-jerks, if the lesion be above the lumbar enlargement. The slighter the degree of motor or sensory loss below the lesion, the slighter is the degree of transverse damage to the cord. Complete motor paralysis below the level of injury indicates that at least a considerable transverse laceration of the cord has occurred. If this complete motor loss is associated with complete loss of sensation below the lesion and abolition of the knee-jerk, the laceration of the cord may be regarded as involving practically the entire transverse extent of the cord.[2] If,

[1] Cases of this class ultimately make a good recovery (though often with considerable weakness and contractures) in some cases. Some of these cases certainly constitute the most favorable cases for operative interference, but many of the cases which would do well if operated on would also do well if left alone.

[2] Cases of this type invariably terminate fatally, whether operated upon or not.

however, the sensory loss is slight in degree, or partial in extent, and the knee-jerk is not abolished, the lesion, though considerable, is not a total transverse lesion, and the structures that subserve the transmission of sensibility are only partially damaged.[1]

In cases of crushing of the lumbar enlargement of the cord the indications of the degree of involvement of the cord are the same as stated above, except that loss of knee-jerk has not necessarily the same sinister significance as in cases of injury above the enlargement. Loss of power in the rectal sphincter and continuous dribbling of urine indicate extensive crushing of the lower part of the lumbar enlargement.

It is exceedingly important to distinguish laceration or crushing of the cord from laceration of nerve-roots. The distinction is not usually difficult if the importance of making it be borne in mind. When the cord is extensively crushed, the nerve-roots at the level of injury almost always suffer in some degree, but the symptoms dependent on their damage are lost in those of the cord injury. When, however, the nerve-roots suffer alone or much more than the cord,[2] the recognition of the condition is usually simple. The symptoms referable to such nerve-root damage are paralysis, atrophy, loss of reflex, and anæsthesia in the parts corresponding to the level of the lesion, and, in cases where irritation is added to

[1] Some ultimate recovery of power may occur in these cases. The majority of them, however, terminate fatally. Cases in which there is complete motor paralysis and persistent loss of knee-jerk *without* considerable or complete loss of sensation, practically belong in the same category with the cases where sensation is entirely lost, for they eventually terminate fatally, though perhaps after a greater lapse of time.

[2] These cases are much less common than those in which the cord is badly crushed. They constitute a very favorable class of cases for operative interference.

destruction, pain, hyperæsthesia, and spasm. These symptoms are often unilateral, or much more marked on one side than on the other. When they are well developed, they contrast strongly with the slightly developed paralysis and anæsthesia and well nourished muscles *below* the level of the lesion.

The great distinguishing features of crushing of the cord are the *immediate* development of the symptoms, paralysis, anæsthesia, etc., after an injury of the spine, and the preponderance of symptoms indicating a *destructive lesion* of the cord over those indicating an irritative lesion. In spinal meningeal hemorrhage the symptoms are of sudden onset, but the symptoms (spasm and pain) indicating an *irritative lesion* greatly preponderate over those that point to a destructive lesion (paralysis, anæsthesia). Cases of hemorrhage into the substance of the cord (hæmatomyclia), of traumatic origin, may closely resemble, in the suddenness of their symptoms and the preponderance of paralytic phenomena, cases of crush of the cord. Indeed, it is doubtful whether any reliable points of distinction can be given. This inability is, however, not very serious, for in most cases of traumatic hæmatomyelia the cord is in some degree lacerated, and the prognosis depends more on the degree and character of the symptoms than on the exact nature of the destructive lesion.

Poliomyelitis.—Poliomyelitis, inflammation of the anterior cornua of the spinal cord, occurs as an *acute* and as a *chronic* or *subacute process.* Acute anterior poliomyelitis is of much more common occurrence than the chronic form, and from its frequency and enduring effects is a disease of great importance. Acute anterior poliomyelitis is especially a disease of later infancy, but it occurs at all periods of life.

Acute Anterior Poliomyelitis (Acute Atrophic Paralysis; Infantile Paralysis).—In general terms the symptomatic characters of acute anterior poliomyelitis are as follows:

(*a*) There is an acute or rapid, never a sudden, onset of the symptoms.

(*b*) The onset is attended very generally, but not always, with fever and other constitutional disturbances.

(*c*) There is paralysis of rapid development, and often, at first, of extensive distribution. The paralysis soon wears away except in the region or regions most affected, and in these muscular atrophy sets in.

(*a*) This muscular atrophy advances rapidly, and the muscles involved are flaccid and present the RD.

The constitutional symptoms attending the onset of acute poliomyelitis vary considerably in severity in different cases. The temperature, which is rarely above 103°, is associated with prostration, headache, etc. In young children there may be delirium, convulsions, and vomiting; in adults and older children cerebral symptoms are rare, but sharp pains in the limbs are not uncommon. The constitutional symptoms of acute poliomyelitis are no doubt due to (general) infection, the local symptoms—paralysis, atrophy, RD, and loss of reflex—to the destruction of the ganglion-cells of the anterior horns.

The paralysis may at first involve all four extremities, but is generally much less extensive, and may be confined to part of one limb. Usually two or three limbs are affected—both legs, both arms, a leg and arm, or both legs and an arm—the lower extremities more often than the upper. When two or more extremities are affected, one usually suffers much more than the others. The cranial nerves are rarely affected. In severe cases, in which the legs suffer, the sphincter may be paralyzed, but this is rare.

After a few days the paralysis lessens in range and becomes confined to parts in which the muscles rapidly waste and develop RD. After a time permanent shortening of the paralyzed muscles occurs, and various deformities are produced. In children the bones of the paralyzed limbs undergo an arrest of development, and add much to the degree of the deformity. The leg suffers more often than the arm. Sometimes all the muscles of the leg are paralyzed and atrophied; *as a rule the muscles below the knee are more severely affected than those above.* The anterior tibial and peroneal groups suffer most often, as shown in Fig. 49, and their paralysis results in talipes equinus—the most common type of deformity in poliomyelitis. Sometimes the posterior tibial group suffers most, with resulting talipes calcaneus. When there is leg paralysis in poliomyelitis, the extensors of the knee are often affected, and their involvement, together with interruption of the muscle reflex arc by disease of the gray matter in the cord, *causes the loss of the knee-jerk.* In the arm all the muscles are never entirely paralyzed. The thenar muscles of the hand and the interossei are often involved. The deltoid also is commonly paralyzed, more often, indeed, than any of the arm muscles. Often the extensors and flexors of the forearm are included in the paralysis of the hand muscles. Both in the arm and leg the distribution of the paralysis is random; occasionally functionally related muscles only are involved.

The recognition of acute poliomyelitis at the time of the onset of the symptoms is by no means always easy, indeed sometimes it is impossible. The symptoms with which the disease is ushered in—fever, prostration, and perhaps convulsions—are of common occurrence in children in various general conditions, and are to be regarded

as evidence of a general process until the distinctive nervous symptom—localized paralysis—makes its appearance. In young children it is very easy to overlook localized paralysis in the early stage of the disease, since

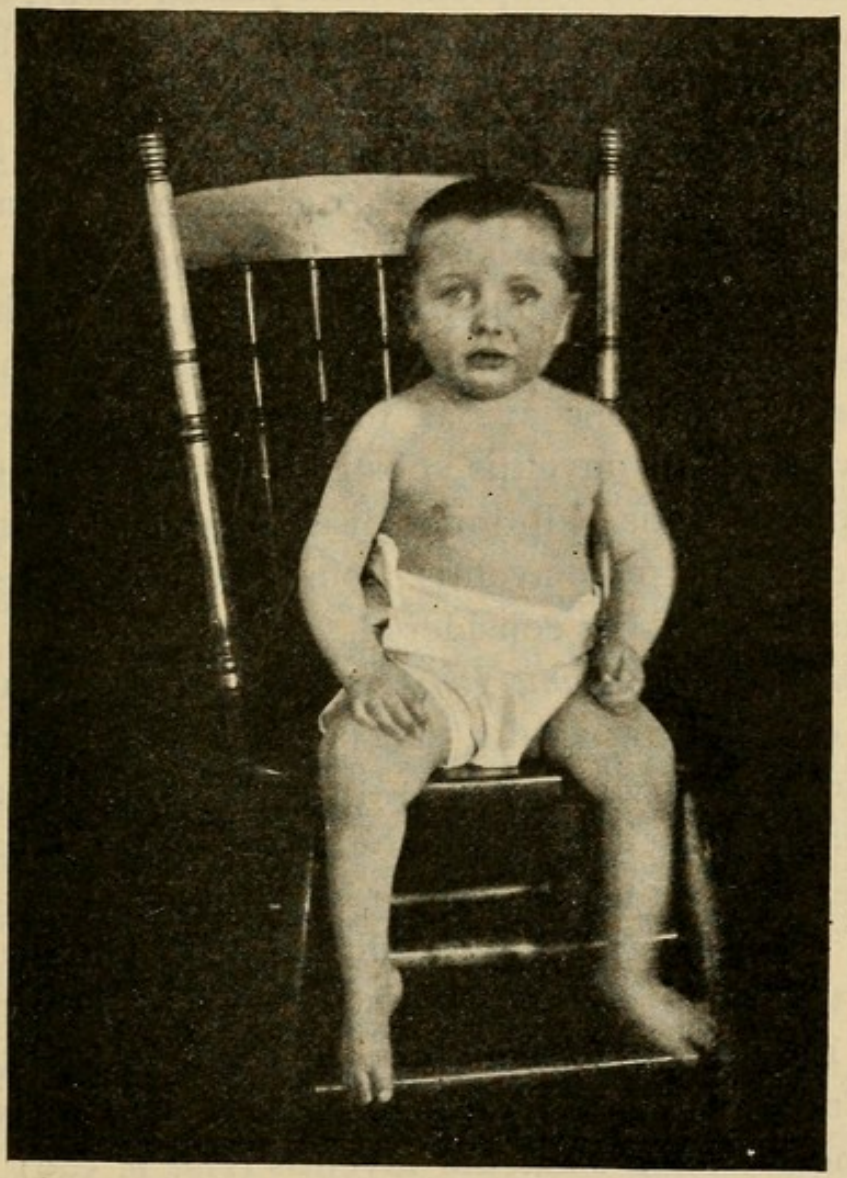


FIG. 49.

Poliomyelitis affecting the right leg only (tibial and peroneal type).

the prostration that accompanies it may cause partial and temporary immobility. *Continuous* local immobility, as of one leg or one arm, cannot, however, be explained by existing prostration and indicates paralysis. If there is

paralysis of hemiplegic distribution (arm and leg), and the onset is accompanied with decided cerebral symptoms (vomiting and convulsions), the possibility of a cerebral palsy must be kept in mind.

After the initial symptoms have passed away, and the obvious *paralysis* is accompanied with *wasting*, the diagnosis is simple. The presence of RD in the wasted and flaccid muscles, the loss of reflexes, and the absence of anæsthesia, bear out the diagnosis of poliomyelitis. Yet while the diagnosis may usually be made without difficulty, there are numerous conditions with which the disease may be at times confused. The most important of these conditions are as follows: Infantile cerebral paralysis, acute myelitis, obstetrical paralysis (Erb's), multiple neuritis, hip-joint disease and rickets. The distinction of poliomyelitis from certain of these conditions is elsewhere considered.

Subacute and Chronic Poliomyelitis.—It sometimes happens, especially in adults, that poliomyelitis is subacute, instead of acute in its onset—the symptoms of paralysis not reaching their height in the course of a few days, but in the course of one, two, or three weeks. These are cases of subacute poliomyelitis. They pursue essentially the same course as cases of acute poliomyelitis. They may terminate fatally in the stage of onset. The cases must be carefully distinguished from cases of multiple neuritis and subacute myelitis (*vide* p. 509)

Very rarely cases are seen in which chronic muscular atrophy and paralysis are due to chronic inflammatory lesions of all the nervous structures of the anterior horns, which are pathologically different from the lesions of progressive muscular atrophy. Clinically these cases of poliomyelitis cannot be distinguished from pro-

gressive muscular atrophy (*vide* progressive muscular atrophy).

Degenerative Diseases of the Spinal Cord.—The degenerative diseases of the spinal cord may be grouped as follows :

a. Primary and secondary *spastic paraplegia*—dependent on primary and secondary sclerosis of the lateral pyramidal tracts.

b. Locomotor ataxia—dependent on sclerosis of the posterior columns of the cord, the posterior root-zones, and peripheral sensory nerves.

c. Ataxic paraplegia—dependent on sclerosis of both posterior and lateral columns.

d. Friedreich's disease (hereditary ataxic paraplegia, a form of ataxic paraplegia)—in which the lesion is a combination of the lesions of ataxic paraplegia with those of locomotor ataxia, developing in families during the adolescent period.

e. Progressive muscular atrophy—in which the lesion is a slow degeneration of the ganglion-cells of the anterior horns of the spinal cord and anterior root fibres associated generally with degeneration of the pyramidal tracts.

Spastic Paraplegia.—In every case (except some acute lesions) of transverse lesion of the spinal cord above the level of the lumbar enlargement the paralysis of motion is accompanied after a time with well defined symptoms of a spastic character below the level of the lesion. The symptoms consist, first, of increased myotatic irritability, as evidenced by increased knee-jerks and the presence of ankle clonus, and, secondly, of tonic spasm or rigidity in the paralyzed extremities. To this combination of symptoms the term "spastic paraplegia" is very appropriately applied. The spastic phenomena

depend on secondary degeneration of the pyramidal tracts, and hence the clinical condition may be denominated "secondary spastic paraplegia."

The diseases of the spinal cord on which secondary spastic paraplegia may depend are numerous and important, and the degenerative changes in the pyramidal tracts, on which the spastic symptoms depend, are generally of secondary interest to the primary lesion. The lesions of the cord that most often cause spastic paraplegia are compression of the cord from spinal caries, and acute and chronic myelitis. Less frequently spastic paraplegia results from compression of the cord from tumors, thickening of the dura mater, etc. Secondary spastic paraplegia may result from other than spinal cord lesions. Thus it follows any cerebral condition that causes bilateral degeneration of the motor path, as, for example, all cases of bilateral ordinary (capsular) hemiplegia, bilateral meningeal hemorrhage in children, general paralysis of the insane, etc. In some forms of cerebral disease (*e. g.*, some cases of infantile cerebral paralysis), the symptoms due to bilateral secondary degeneration of the pyramidal tracts are much more obtrusive than the evidences of the original cerebral lesion, and in such cases the distinction from primary spastic paraplegia may be difficult or impossible.

Primary Spastic Paraplegia.—The term primary spastic paraplegia is used to designate cases in which there is a gradual development of weakness in the lower extremities, with increase of knee-jerks and ankle clonus and tonic spasm, often very marked, and especially conspicuous in the gait of the patient, without any evidence of a primary focal lesion in the spinal cord or brain. The cases of spastic paraplegia which are due to an unquestionably primary sclerosis of the lateral tracts are cer-

tainly of extremely rare occurrence. The symptoms in such cases are as follows :

1. Weakness of the lower extremities of exceedingly slow development (many months or years). The weakness generally involves chiefly the flexors of the hip, knee and ankle. At first the weakness may be greater in one leg, but after a time both are equally affected. The arms are rarely affected, but occasionally they are the seat of some loss of power.

2. Rigidity in the muscles of the lower extremities. The spasm affects especially the extensors. It is least when the extremities are flexed and greatest when they are extended. Roughly speaking the spasm is proportioned to the degree of paralysis.

As a result of the combined spasm and paralysis the gait is peculiar. The rigid leg is dragged forward as a whole, the toes touching the ground, the heel elevated. In children there is often a tendency to "cross-legged progression" from spasm of the adductors.

3. The knee-jerks are exaggerated and there is ankle clonus and typical Babinski's sign. The excess in myotatic irritability is so great in some instances that there are attacks of spontaneous and violent clonic spasm in the leg muscles, such as is produced by dorsal flexion of the foot. When the ball of the foot rests on the ground there may be excited similar clonic spasm.

There is little or no wasting of the extremities in primary spastic paraplegia, and sensory symptoms are absent. The sphincters are usually not affected, but they may be involved from the first.

As regards the etiology of the disease there is little that is of service in diagnosis. The most important point in this connection is that the disease most often begins between the twentieth and fortieth years, seldom

later than this, and not infrequently between the tenth and twentieth years.

In every case in which the slow onset of paralysis, spasm, and myotatic increase suggest the existence of primary spastic paraplegia, it is very important to exclude all indications of a focal lesion. Secondary spastic paraplegia is far more common than the primary form, and we cannot be too careful in looking for evidences of brain disease or of spinal cord disease, before concluding that the case is one of primary spastic paraplegia. The very gradual onset of the symptoms of primary spastic paraplegia is of much importance in diagnosis. We may exclude at once from this category all cases in which weakness and spasm are of sudden, acute, or subacute development. The presence of very slight subjective sensory symptoms—numbness, tingling, formication—does not of itself remove the case from the category of primary spastic paraplegia, but the existence of any considerable degree of anæsthesia makes it certain that the lesion is not confined to the lateral pyramidal tracts.

Cases of primary spastic paraplegia are not rarely mistaken for hysterical paraplegia. Many cases of primary spastic paraplegia are seen at the time of life when hysterical manifestations are common. In primary spastic paraplegia the rigidity and spasm vary much with the position of the limbs—considerable relaxation occurring on passive flexion. In hysterical paraplegia the spasm does not vary with the posture of the limb. In primary spastic paraplegia the myotatic excess is very pronounced ; in hysteria there may be increased knee-jerks, but pronounced and true ankle clonus is rare, and the presence of such clonus should excite suspicion of organic disease. When hysterical sensory and mental phenomena are present, the question of diagnosis need hardly arise,

but it must be remembered that hysterical symptoms may co-exist with this form of organic disease.

For a time the leg and arm of the same side may suffer chiefly, and in such cases the distinction from hemiplegia must be made. The slow character of the onset, absence of cerebral symptoms, non-involvement of the face, and slight involvement of the leg and arm of the side opposite suffice to make the diagnosis clear.

Locomotor Ataxia (Tabes Dorsalis, Posterior Sclerosis).—The symptoms of locomotor ataxia depend mainly upon a degenerative process in the *sensory* structures of the central and peripheral nervous systems. The most constant and significant alterations observed in this disease are degeneration of the posterior columns of the cord (especially the posterior median columns), the posterior root-zones, and the peripheral neuromuscular (sensory) segment—structures that are purely sensory in their functions. In the brain the degenerations of locomotor ataxia do not confine themselves so strictly to sensory structures. Thus while degenerative changes occur, not rarely, in the optic nerves (sensory structures), the third and other motor nerves may be similarly involved.

Cases of locomotor ataxia present a great variety of symptoms. Of these symptoms some are of such diagnostic importance and are so often observed that they may be looked upon as of cardinal importance; others, though often observed, or even regularly present in advanced cases, are common to so many diseases that they may be considered of secondary diagnostic value.

The cardinal symptoms of locomotor ataxia are as follows:

(1) *Lightning Pains.*—These are paroxysmal pains, of neuralgic character, and often of great severity. They

are felt chiefly in the legs, but may occur in the trunk, arms, or head.

(2) *Ataxia.*—The inco-ordination of movement is chiefly of the legs, but may extend to the arms. It is unassociated with any loss of gross power or muscular atrophy.

(3) *Loss of Knee-Jerk.*

(4) *The Argyll-Robertson Symptom.*—That is, loss of pupillary reaction to light with preservation of reaction during accommodation. The diagnostic value of each of these symptoms and of certain combinations of them is discussed later.

The order in which the symptoms of locomotor ataxia develop varies much in different cases. Certain symptoms, however, usually develop early—as loss of knee-jerk, lightning pains, Argyll-Robertson symptom, and optic atrophy, when it occurs; while others, as considerable ataxia, belong to a later period, and others still, as loss of sphincteric control, belong to an advanced stage. A purely arbitrary division of the symptoms has been made, having reference to their most usual order of development. The convenience of this division and its general accuracy, if nothing more, warrants its use. The grouping is as follows :

I. *Neuralgic or Pre-Ataxic Stage* of locomotor ataxia.

The chief symptoms of this stage of locomotor ataxia are 1st, lightning pains, and 2d, loss or diminution of knee-jerk. There is no alteration in the gait and no ataxia, but there is often inability to stand with the feet together and the eyes closed (Romberg symptom).[1] An-

[1] A considerable sway when the eyes are closed and the feet are close together is seen in many neurasthenic and hysterical persons, but inability to stand with the eyes closed is probably always the result of organic disease.

other symptom frequently present in this stage is the loss of light reflex of the iris with preservation of reaction during accommodation (Argyll-Robertson symptom). The symptoms of the neuralgic stage, which are of secondary importance and inconstant occurrence, comprise the following : localized hyperæsthesia of the lower extremities, numbness and slight anæsthesia of the feet, diplopia and ptosis, sexual excitement, and amblyopia from optic nerve atrophy.

II. *The Second or Ataxic Stage.*

The great characteristic of this stage, which cannot of course be sharply separated from the first, is the presence of distinctly marked inco-ordination. The gait is slightly or considerably ataxic, but the patient is still able to walk, at least with help. Of course the cardinal and secondary symptoms of the neuralgic stage continue and become more fully developed. In addition, severe paroxysms of pain in the viscera (crises—laryngeal, gastric, vesical) may develop, there may be loss of sexual power, and peculiar joint conditions (*vide* page 169) may appear. Loss of control over the sphincters is a feature of this stage.

III. *The Third or Pseudo-Paralytic or Terminal Stage.* —In this stage the patient becomes unable "to use his legs" owing to the high degree of inco-ordination. On account of this and other symptoms he has become practically helpless. The symptoms of the second stage persist, and some of them are more fully developed.

The separation of the symptoms of the first and second stages is useful as emphasizing the fact, which is of considerable practical importance, that the development of inco-ordination *is preceded by other important symptoms.* While this is generally true, it must be noted that inco-ordination is sometimes a very early symptom. It may indeed develop before the knee-jerks are lost.

As in other forms of organic disease the diagnosis of locomotor ataxia should be made so far as possible on objective grounds. In strict subordination to these grounds, the etiological facts of the disease should be allowed weight. These facts are that it is essentially a disease of middle and early adult life, occurs chiefly in men, and is in about ninety per cent. of the cases a more or less remote sequel (five to twenty-five years) of syphilis.

The diagnosis of a fully developed typical case of locomotor ataxia, with its lightning pains, ataxia, loss of knee-jerks, altered sensibility, and pupillary symptoms, presents no difficulties even to a novice. The problem is by no means so simple when we have to deal with the early stage of the disease, or when we encounter atypical forms. To the consideration of such cases we will now pass.

In the early stage of locomotor ataxia we may be compelled to make a diagnosis in the presence of a very few symptoms. Loss of knee-jerk, combined with lightning pains or Romberg's symptom, justifies a diagnosis of locomotor ataxia in its first stage, if we can rule out alcoholic neuritis, diphtheritic neuritis, and diabetic neuritis. These same symptoms may occur in cases of disease of the anterior horn or anterior nerve-roots, but the presence of paralysis, atrophy, and RD would readily exclude such cases. Loss of knee-jerk with Argyll-Robertson pupils points positively to locomotor ataxia. The latter symptom may, however, depend on general paralysis, and the combination with loss of knee-jerk might depend on a complicating and not a primary locomotor ataxia. Mental and other physical symptoms are usually sufficiently marked in such cases of general paralysis to prevent error.

The knee-jerk may be present for a time in the early stage of locomotor ataxia, or it may be merely diminished instead of lost, or it may be lost on one side only. In such cases a diagnosis of locomotor ataxia can be justified only by the existence of typical ataxia. If, in addition to such ataxia, there are lightning pains or Argyll-Robertson pupils the diagnosis is greatly strengthened.

When characteristic lightning pains are the first symptoms of locomotor ataxia we can hardly do more than make a probable diagnosis until other symptoms appear. Lightning pains associated with Argyll-Robertson pupils constitute sufficient evidence for a diagnosis of locomotor ataxia, and lightning pains combined with incontinence of urine or diminished sexual power should lead to a decided suspicion of the disease, but do not suffice, by themselves, for a positive diagnosis.

Locomotor ataxia may be confounded with some affections of the peripheral nerves, spinal cord, and brain. Of the peripheral diseases with which it may be confounded two are of especial importance—alcoholic multiple neuritis and diphtheritic multiple neuritis (*vide* pp. 510 and 520. Of the spinal cord cases in which confusion may arise the most important is ataxic paraplegia (*vide* p. 438). No confusion should arise with cases of spastic paraplegia. In such cases there are loss of power, spasm, and increased knee-jerk, and usually ankle clonus. Actual loss of power does not occur in tabes until late in the disease, when the other characteristic symptoms—inco-ordination, loss of knee-jerk, etc.—are present. The spasm of spastic paraplegia is never seen in tabes, and the increased knee-jerks and ankle clonus contrast sharply with the lost knee-jerks of the latter disease. In spastic paraplegia, moreover, there is never true ataxia.

General paresis cannot be confounded with tabes, although the latter may form a part of the former. Sometimes (twenty per cent. of cases) symptoms of spinal involvement (tabes) develop first in general paresis,—at least before the cerebral symptoms become at all marked. Loss of knee-jerk in any case of general paralysis indicates strongly an associated tabes.

Argyll-Robertson pupils are common to both diseases and do not help in the distinction, and this is true of a moderate degree of inco-ordination. Generally speaking the problem is settled in favor of the diagnosis of general paresis if the symptoms of dementia accompany those of tabes.

The inco-ordination of locomotor ataxia may be confounded with the ataxia of cerebellar disease, such as occurs in cases of cerebellar tumor. The differences between cerebral ataxia and spinal ataxia have been pointed out (*vide* p. 102), but sometimes the ataxia of locomotor ataxia resembles closely that of cerebral tumor. Loss of knee-jerk makes locomotor ataxia exceedingly probable, but it does not settle the point, because the knee-jerk may be temporarily absent in cerebellar tumor. The presence of lightning pains, sensory disorders, or loss of light reflex, on the one hand, and of cerebral symptoms (optic neuritis, headache) on the other, usually enable the distinction to be easily made.

Ataxic Paraplegia.—The symptoms of ataxic paraplegia are essentially a combination of the symptoms of locomotor ataxia and those of spastic paraplegia. This peculiar combination of symptoms is intelligible when we consider that the pathological conditions which underlie it present the chief characters of the lesions of the two diseases, locomotor ataxia and spastic paraplegia—that is to say, there is present in cases of ataxic paraplegia a degeneration both of the lateral pyramidal

tracts and of the posterior columns. But these lesions, though bearing a close resemblance to those of locomotor ataxia and ataxic paraplegia, are not identical with them. The most important difference in the lesions relates to those that resemble locomotor ataxia. In the first place the degeneration of the posterior columns instead of being most intense, as in locomotor ataxia, in the lumbar region of cord, is quite as well or better marked in the dorsal region of the cord. Secondly, the root-zone of the postero-external column of the cord is much less intensely sclerosed than in locomotor ataxia, or it presents no degenerative changes whatever. These facts have an important bearing in explanation of the symptoms of ataxic paraplegia.

The essential and typical symptoms of ataxic paraplegia are as follows :

1. *Gradual Loss of Power*, especially in the lower extremities. The muscles involved are particularly the flexors of the thigh and knee, and the effect of their weakness is first shown in walking. One leg may be considerably more affected than the other.

2. *Ataxia*, especially of the lower extremities, developing gradually and *pari passu* with the loss of power.

Gradual development of spasm in the lower extremities; together with

4. Increased knee-jerk, Babinski's sign, and, usually, ankle clonus.

Roughly speaking, we may say that a case of ataxic paraplegia presenting the typical symptoms above enumerated is clinically spastic paraplegia plus the symptom ataxia. Many cases, however, develop other symptoms, symptoms which may ally the case especially with locomotor ataxia on the one hand, or with spastic paraplegia on the other. Of these symptoms,

which are virtually of secondary diagnostic importance in the majority of instances, the following are the most important : (*a*) slight impairment of sensation in the legs (occasional) ; (*b*) dull pain over the sacral region (common) ; (*c*) girdle pain (rare) ; (*d*) lightning pains (very rare) ; (*e*) hyperæsthesia of lower extremities (rare) ; (*f*) loss of sexual power (frequent) ; (*g*) loss of control over sphincters ; (*h*) loss of light reflex ; (*i*) slight difficulty in articulation. In very rare cases the knee-jerks may be lost, and there may be anæsthesia of the legs. Such cases are closely allied to locomotor ataxia. As regards the etiology of ataxic paraplegia there is nothing sufficiently distinctive to be of any help in diagnosis.

During the early period of ataxic paraplegia, when in-co-ordination is the most obtrusive symptom, the disease may be mistaken for locomotor ataxia. The great point of distinction is the state of the knee-jerk. This is lost in locomotor ataxia, exaggerated in ataxic paraplegia. Occasionally during the early stage of tabes (*vide* Tabes) the knee-jerk is retained, but in such cases it is never exaggerated. The occurrence of extensive spasm in the lower extremities, such as occurs in spastic paraplegia, is also strongly in favor of the existence of ataxic paraplegia. Other points of difference are the absence in ataxic paraplegia of marked sensory disturbances, including lightning pains, the absence of Argyll-Robertson pupils, etc. The diagnosis is far more difficult in the rare cases that show loss of knee-jerk and considerable impairment of sensation. Such cases, indeed, resemble ataxic paraplegia very closely in their pathological anatomy. In ordinary cases of ataxic paraplegia the posterior root-zones, as above stated, are only slightly or not at all involved, and this exemption explains both the absence of

sensory symptoms and the retained knee-jerk. But in the cases under consideration this zone is degenerated, and hence the loss of reflex and disturbed sensation. The diagnosis from tabes would depend in such a case on the presence of spasm and weakness in the lower extremities, due to degeneration of the lateral pyramidal tracts.

The distinction of ataxic paraplegia from spastic paraplegia is simple. It is based on the presence of incoordination. When the symptoms of ataxic paraplegia develop in the course of a few months—that is, have a subacute onset, as they sometimes do, the distinction from focal subacute myelitis, involving chiefly the posterior and lateral columns, may be difficult and even impossible. The more progressive the symptoms the greater the likelihood that the disease is ataxic paraplegia ; if there is a retrogressive tendency this is in favor of focal myelitis. Some cases of tumor of the middle lobe of the cerebellum cause both ataxia and weakness of the lower extremities—ataxia from the direct involvement of the co-ordinating centre, weakness and spasm from pressure upon the neighboring pyramidal tract. The presence of headache suggesting organic disease, of vomiting, of optic neuritis, and often of cranial nerve paralysis, in such cases makes the diagnosis from ataxic paraplegia sufficiently simple.

Friedreich's Disease (Hereditary Ataxia).—Friedreich's disease is a degenerative disease of the posterior and lateral columns of the spinal cord which is closely related to ataxic paraplegia. In some respects it also bears a resemblance to locomotor ataxia. It resembles ataxic paraplegia in that there is degeneration of the lateral pyramidal tract of the cord, sometimes also of the direct pyramidal tract, and degeneration of the pos-

terior columns. But the degeneration is more intense in the posterior column, especially in the lumbar region, than is the case in ataxic paraplegia. Moreover, the posterior nerve-roots, which escape degeneration in ataxic paraplegia, are commonly involved. In both these respects the lesions of Friedreich's disease resemble the lesions of locomotor ataxia. Hence we may say that, as regards pathological anatomy, Friedreich's disease is intermediate between ataxic paraplegia and locomotor ataxia, some cases inclining more to the one type, some to the other.

In the symptoms of Friedreich's disease this relation to ataxic paraplegia and to locomotor ataxia is as clearly shown. The principal symptoms are :

(1) *Ataxia*, first in the legs, later in the arms. The ataxia is shown first as unsteadiness in standing and walking, later in a reeling, drunken gait. Ataxia is the first and most obtrusive symptom. After a time the head often becomes the seat of irregular ataxic movements.

(2) *Gradual loss of power* in the lower extremities. Generally there is no considerable loss until late in the disease, but sometimes it occurs early. It very rarely amounts to complete paralysis.

(3) *Loss of knee-jerk.* This is a very early and constant symptom. In a small percentage of cases the knee-jerk is normal, or even exaggerated, throughout the course of the disease.

(4) *Speech disturbance.* This symptom does not usually develop until several years after the development of inco-ordination. The character of this speech defect varies considerably in different cases. Most often there is simple elision of syllables, or hesitation, or drawling, or the speech is jerky in character. The defect may be very slight.

(5) *Nystagmus.* This is a later symptom than the affection of speech, and of much less frequent occurrence. The oscillation usually does not occur except when the eyes are directed laterally.

Beside these characteristic and important symptoms of Friedreich's disease there may be others of less diagnostic significance. Of these the most noteworthy are the following: dull rheumatoid pains in the legs and back (not uncommon), slight anæsthesia of the legs, lightning pains (rare), loss of sexual power, fibrillation or jerky movements of the tongue, slight muscular wasting (late), lateral curvature of the spine, talipes equinus, contractures from muscular paralysis, and vaso-motor disturbances.

There are two etiological facts regarding Friedreich's ataxia that are of much diagnostic importance. The first of these is that the disease is essentially a disease of the adolescent period of life and of the years just preceding puberty. The second is that the disease has a very strong tendency to occur in several members of the same family, although *direct heredity* is unusual. But it is important to note that isolated cases occur.

Bearing in mind the facts of symptomatology and etiology that have been enumerated, the distinction of Friedreich's ataxia is usually not difficult. But the diagnosis is not always easy. Cases of ataxic paraplegia occur (tabetic form) that bear a very close resemblance to Friedreich's ataxia. Friedreich's ataxia can scarcely be confounded with the ordinary type of ataxic paraplegia, because in the former there is regularly loss of knee-jerk, in the latter increase of knee-jerk and ankle clonus, and because the nystagmus and defective articulation of Friedreich's ataxia do not occur in ataxic paraplegia. The knee-jerk is, however, absent in the tabetic type of

ataxic paraplegia, and without other points of distinction to help us the diagnosis may rest on the early age of development of Friedreich's ataxia, and the occurrence of the disease in several members of a family. But even these criteria sometimes fail, and it may be impossible to make an absolute distinction between the two diseases.

The diagnosis between Friedreich's ataxia and locomotor ataxia is never difficult. The important features which the diseases have in common are the ataxia and the loss of knee-jerks. The early and distinctive sensory symptoms of tabes (lightning pains, etc.), loss of iris reflex, etc., separate a typical case of tabes from Friedreich's ataxia. The occurrence of speech defects and of nystagmus shows clearly that the case is not one of tabes but of Friedreich's disease. The age of onset also becomes an important differential point. Friedreich's ataxia always develops before the twentieth year, tabes only very rarely before the twenty-fifth. The family occurrence of Friedreich's disease is a most important differential feature when available.

Friedreich's ataxia and multiple sclerosis may have in common three important symptoms — inco-ordination, speech defect, and nystagmus. But the inco-ordination of multiple sclerosis is distinctive in the violent and wide and jerky movements of the upper extremities. The speech disturbance of multiple sclerosis is usually of the scanning or staccato type ; in Friedreich's ataxia there is usually simple elision or running together. The nystagmus of multiple sclerosis is constant and has no reference to voluntary movements—that of Friedreich's disease is excited by lateral movements. Besides these differences in the common symptoms there are the following important differences : the knee-jerks are lost in Friedreich's

ataxia, increased in multiple sclerosis; there is almost always some mental failure in multiple sclerosis, rarely any in Friedreich's ataxia; multiple sclerosis occurs in isolated cases, Friedreich's ataxia almost always in groups in the same family.

Muscular Atrophies.—Cases of disease are not infrequently met with, of which the chief characteristic is slowly progressive muscular wasting, the wasting being usually narrow in its distribution, at any rate at first. Muscular wasting of limited extent (and rapid or slow development) is, as we have already seen (*vide* p. 170), a regular accompaniment of various forms of cerebral, spinal cord, and peripheral nerve disease—that is, it occurs in such cases as one of several equally important symptoms. It is proposed to consider here not that class of symptomatic muscular atrophies, but a class of cases in which slowly progressive muscular wasting is the one great distinctive feature. It must be owned that the pathology and even the pathological anatomy of some of these forms of muscular atrophy is as yet obscure. Some of the cases (progressive muscular atrophy) of chronic muscular atrophy are unquestionably due to chronic degenerative disease of the spinal cord, some are probably due to primary changes in the muscles themselves (muscular dystrophy), and others still (peroneal type) cannot now be safely classified. The forms of muscular atrophy that are supposed to depend on primary disease of the muscles (progressive muscular dystrophies) do not, strictly speaking, come within the range of this section, but their clinical features are such as to justify their consideration in connection with the diseases of the spinal cord. The conditions to be treated under the head of muscular atrophies are as follows:

A. Progressive Muscular Atrophy. (Due certainly to spinal cord disease.)

B. The Peroneal Form of Progressive Muscular Atrophy. (Due probably to spinal cord disease.)

Progressive Muscular Dystrophies.

C. Pseudo-Hypertrophic Paralysis.

D. Erb's Form of Progressive Muscular Dystrophy.

E. The Landouzy-Déjérine Type of Progressive Muscular Dystrophy.

(Due probably to primary disease-defect of the muscles.)

Progressive Muscular Atrophy (Chronic Spinal Muscular Atrophy, Wasting Palsy, Amyotrophic Lateral Sclerosis, Chronic Poliomyelitis).—The distinctive clinical feature of progressive muscular atrophy is the slow and usually extreme wasting of certain groups of muscles. The pathological basis of the disease consists of a gradual degeneration of the ganglion-cells of the anterior horns of the cord, at a level of the cord corresponding to the muscles wasted, and of a like degenerative process in the anterior root-fibres with which these cells are connected. In association with these lesions in the ganglion-cells and their prolongations the root-fibres, there is usually a degeneration of the direct and crossed pyramidal tracts of the cord—a degeneration which may be traceable a considerable distance along the motor path in the brain.

Like the other degenerative diseases of the spinal cord that have been described, progressive muscular atrophy fails to conform to any rigid type in its symptoms. On

the other hand, it resembles these diseases in having a symptomatology that varies within considerable limits. Now in order to comprehend the meaning of these variations in type it is essential to bear in mind certain variations in the lesions which will be mentioned in the description of the symptomatology of the disease.

As already stated, the striking clinical feature of progressive muscular atrophy is *slow muscular atrophy*. This



FIG. 50.

Atrophy and paralysis with *main en griffe* in chronic anterior poliomyelitis. (Starrs.)

muscular atrophy commences in the arms in about nine tenths of all the cases (Aran-Duchenne type). In the majority of the cases beginning in the arm the atrophy commences in the muscles of the hand, but a considerable number commence in the muscles of the shoulder. Of the hand muscles those of the thenar and hypothenar eminences (thumb and little finger) are usually the first to suffer, but the interossei and especially the *first dorsal*

interosseus (abductor indicis) usually suffer very early. This commencement of the atrophy in the small muscles of the hand and interossei is so common as to be a characteristic feature of the disease.

The atrophy generally begins on one side and does

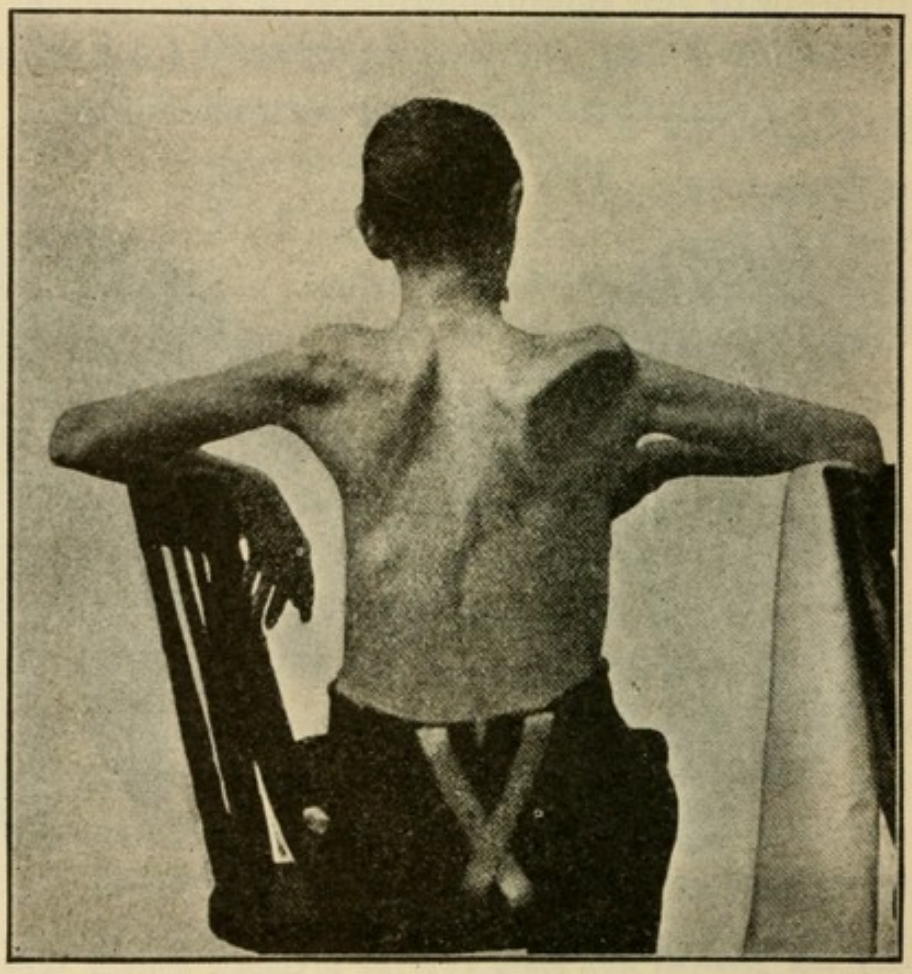


FIG. 51.

Atrophy of the muscles about the shoulder-blades and arms in a case of chronic anterior poliomyelitis. The triceps and latissimus dorsi have escaped. (Starrs.)

not appear on the other until some time (often a year) has passed. When the atrophy commences in the shoulder the deltoid generally suffers first, but whether hand or shoulder first shows the wasting it soon extends to other parts of the limb. Thus both the flexors and extensors of the wrist, the supinators, the biceps, and triceps

may in time become one or all involved. Often the various muscle groups are unequally atrophied.

Usually after a time the muscles of the back, especially the trapezii, suffer. A peculiarity of the trapezius atrophy is that its upper part (*ultimum moriens*) often remains intact when the rest is much wasted. The sterno-mastoid also is often involved. The respiratory muscles may suffer early or late, and thus threaten life. Both intercostals and diaphragm may be affected.

In exceptional cases the atrophy commences, not in the upper extremity, but in the legs. There is good reason to think that the lesion that underlies these cases that *commence* in the leg is of the same nature as that which underlies the cases that begin in the upper extremity, but as there is no direct proof of this, this class of cases is considered elsewhere (*vide* Peroneal Type). Although it is exceptional to meet with cases in which the atrophy commences in the leg, it is not very unusual for cases commencing in the arm to show slight wasting of the legs after a long lapse of time. The face is rarely involved in the atrophy, but the lips may be wasted owing to the not infrequent complication of progressive muscular atrophy with bulbar paralysis. The muscles that undergo atrophy gradually fail in power. This loss of power *is in a general way proportioned to the degree of the wasting*, to which it is usually due. There is an important exception to this general rule. It is that the legs may gradually lose power where there is no wasting, or such slight wasting that the loss cannot be due to this. The paralysis that accompanies wasting depends, like the wasting, on disease of the ganglion-cells of the anterior horns; the paralysis that occurs without wasting, or is greatly in excess of it, depends on degeneration of the pyramidal tracts of the cord. In addition to the atrophy and weakness that

occur in progressive muscular atrophy there are the following important conditions:

a. Fibrillary Contractions of the atrophied muscles. This is so frequent a symptom in progressive muscular atrophy as to be characteristic when present, but it is not of invariable occurrence. There is also increased mechanical irritability.

b. Changed Electrical reactions. When the wasting is slow there is usually simple diminution of irritability to faradism and galvanism. When the wasting is rapid the faradic loss may be out of proportion to the galvanic loss, and there may even be partial or complete RD in certain muscles.

c. Changes in Reflex Action. As a rule, there is loss of myotatic irritability in the atrophied muscles. The knee-jerk is lost as soon as there is even slight wasting of the anterior thigh group, in those cases where the leg is affected. When the legs are paralyzed without wasting (that is, from degeneration of the pyramidal tracts), the knee-jerks are exaggerated and there is clonus. There may, indeed, be true rigidity, and the leg symptoms may thus resemble closely those of spastic paraplegia.

d. Changes in the Tone of the Muscles. The atrophied muscles are ordinarily flaccid and without tone; they are in a state of "atonic atrophy." Occasionally the muscles are in a state of rigidity from the first, *i. e.*, they are in a state of "tonic atrophy."

Beside the symptoms of progressive muscular atrophy that have been enumerated there may occur others of minor importance. Thus, an early symptom is aching pain in the parts that become atrophied, and later there may be numbness in the parts, but there is never any true anæsthesia. The unequal paralysis of antagonistic muscles may lead to various deformities; in the hand the bird-

claw hand (*vide* ulnar paralysis) is apt to occur. Sexual power is frequently lost. The sphincters are unaffected.

Some of the variations in the symptomatology of progressive muscular atrophy have been briefly stated (variations in reflex state, muscle tonus, and degree of wasting). It remains to be indicated how these variations form the basis of certain clinical types.

We may regard as typical forms of progressive muscular atrophy those cases in which there is considerable atrophy of the *atonic* sort in the upper extremity and upper part of the trunk, combined with weakness and rigidity, *without atrophy* or with *very slight atrophy*, in the legs. The atonic atrophy in the upper part of the body depends on the degenerative changes, already mentioned, in the ganglion-cells of the anterior horns. The weakness and spasm in the legs (with increased knee-jerk and clonus) depend, on the other hand, upon degeneration of the pyramidal tract (*vide* symptoms, Rigidity or Spasm), and on those fibres of the tract that pass to the leg centres. For, although the pyramidal tract may be degenerated throughout its entire extent, the degeneration of those fibres that go to the arm-centres of the cord does *not* cause overaction in the arm muscles. This is because the ganglion-cells of the anterior cornua of the cervical cord are so extensively degenerated that no degree of degeneration of the pyramidal tract can cause the arm muscles they innervate to overact. Were these ganglion-cells less degenerated, or were some of them intact and others degenerated, the case would be different. The degenerated pyramidal tract would in this case cause overaction in the cervical ganglion-cells, with resulting rigidity of the upper arm and upper trunk muscles. This is precisely what happens in some cases—namely, the cases of progressive muscular atrophy in which there is

tonic atrophy (often slight) in the arms, and simply weakness and spasm, or spasm and weakness with moderate atrophy, in the legs.

It is instructive to picture still another pathological variation in the same direction. Cases occur in which the degeneration of the pyramidal tracts forms the chief lesion, and in which the degeneration of the cervical ganglion-cells is very slight indeed. These cases are closely allied to a condition that has been already described, viz., spastic paraplegia—in fact, they differ from it clinically merely in the slight atrophy of certain muscles of the upper extremity.

Finally, another variation, opposed in character to the last, must be noted. Cases occur in which the ganglion-cells of the cervical and lumbar cord are all so extensively degenerated that notwithstanding the presence of degeneration of the pyramidal tracts, there is no rigidity or spasm in the lower extremities. There is, in such cases, what may be called universal atonic atrophy—the arms and legs are atrophied (often extensively), flaccid, and have lost their myotatic irritability.

Enough has been said to show that in progressive muscular atrophy there are extensive variations in type, based on corresponding variations in pathological anatomy. There is, indeed, every conceivable gradation in the combination of spasm, atrophy, weakness, and myotatic increase, between widespread tonic atrophy, verging on pure spastic paraplegia, on the one hand, and on universal atonic atrophy on the other.

The forms of progressive muscular atrophy in which spasm is marked have been designated amyotrophic lateral sclerosis, and have been considered pathologically distinct from the atonic forms. This view is untenable, since lateral sclerosis is present, as we have

seen, even in cases that present no spasm or myotatic excess; the effect of the lateral sclerosis, in producing overaction in the anterior cornual cells, being rendered inoperative by the degeneration of those cells. It is, therefore, more in accord with the facts of pathology to regard all cases coming under the designation amyotrophic lateral sclerosis as varieties of one varying pathological state, than to attempt their establishment as a distinct condition.

Progressive muscular atrophy has been attributed to a large variety of different conditions, but none of these bear a sufficiently distinctive relation to the disease to aid us materially in diagnosis (*vide* p. 289). Two etiological facts, however, it is well to bear in mind: first, that the disease is essentially one of adult life, though it may commence in adolescence or in advanced life; and second, that when hereditary influences can be traced they are only rarely direct.

The Peroneal Form of Progressive Muscular Atrophy (Leg Type, Primary Neuritic Atrophy).—The peroneal form of progressive muscular atrophy has many features that ally it with the typical progressive muscular atrophy that has just been described. It is, moreover, probable, upon the whole, that it depends on changes in the spinal cord similar to those that cause the atrophy of the typical form, though some authors consider it a peripheral affection.

The feature of this disease is *slowly progressive muscular atrophy* of peculiar distribution. The atrophy begins in the lower extremities. Very often the beginning is in the extensor hallucis longus or the common extensor of the toes, from which it extends to the peronei muscles. The small muscles of the foot may or may not be affected. The calf muscles atrophy after the

peronei, and, last of all and often at a late date, the thigh muscles are involved. After a long time the atrophy extends to the small muscles of the hand. The atrophy is always atonic, and usually symmetrical. The knee-jerks are lost when the thigh muscles waste—that is, late. The electrical changes present the same variations as in typical progressive muscular atrophy. Fibrillation occurs in the wasted muscles not infrequently, but not with the constancy that it occurs in progressive muscular atrophy. Vaso-motor disturbances are common, and slight sensory disturbance may occur. This form of progressive muscular atrophy is sometimes known as the "leg type," to distinguish it from the usual form, which may be called the "arm type."

The leg type generally develops during early youth. There may be direct hereditary influence and several cases may occur in the children of the same parents.

The distinction between the two described forms of muscular atrophy is of course most simple as a rule. As the arm type may involve the leg, and as the leg type may eventually involve the arm, an error might occur if we failed to discern the order of spreading of the atrophy. Without this knowledge, a diagnosis might be difficult; but the known difference in the influence of heredity, and the fact that the arm type causes often unilateral atrophy in the leg, while the atrophy is symmetrical in the cases of the leg type, might be of service in diagnosis. Both types are distinguished from poliomyelitis by their gradual development and progressive course. The leg type cannot be confused with congenital club-foot if due care is taken in bringing out the history of the case. The further distinction of these types from other conditions may be conveniently postponed.

Progressive Muscular Dystrophies.—The progressive muscular dystrophies are those forms of muscular atrophy that depend, not on disease of the central nervous system, but on primary lesions of the muscles. The progressive muscular dystrophies have several general characters in common, which serve to separate them clinically from the typical form of progressive muscular atrophy. These characters are as follows: (1) development in early youth; (2) the occurrence of true or false hypertrophy of the muscles; (3) the absence of degenerative electrical reactions; (4) the absence of fibrillary contractions.

The forms of progressive muscular dystrophies to be discussed are as follows:

1. Pseudo-hypertrophic paralysis.
2. Erb's form of progressive muscular dystrophy.
3. The Landouzy-Déjérine type of progressive muscular dystrophy.

Of these three forms, the first may be separated in classification from the second and third without hesitation. The second and third forms, however, have so many features in common, that we are scarcely justified in making a sharp line between them. They are here considered separately for the sake of convenience.

Pseudo-Hypertrophic Paralysis (Pseudo-Muscular Hypertrophy).—The chief characters of this disease are as follows:

(*a*) Weakness in the muscles of the leg, and a waddling gait, associated with

(*b*) Apparent increase in the size of certain leg muscles, especially the muscles of the calf, or the calves and thighs. There may be also considerable enlargement of some of the muscles of the shoulder, especially the infra-

spinati and deltoids. Eventually many of the hypertrophic muscles may undergo atrophy, as shown in the

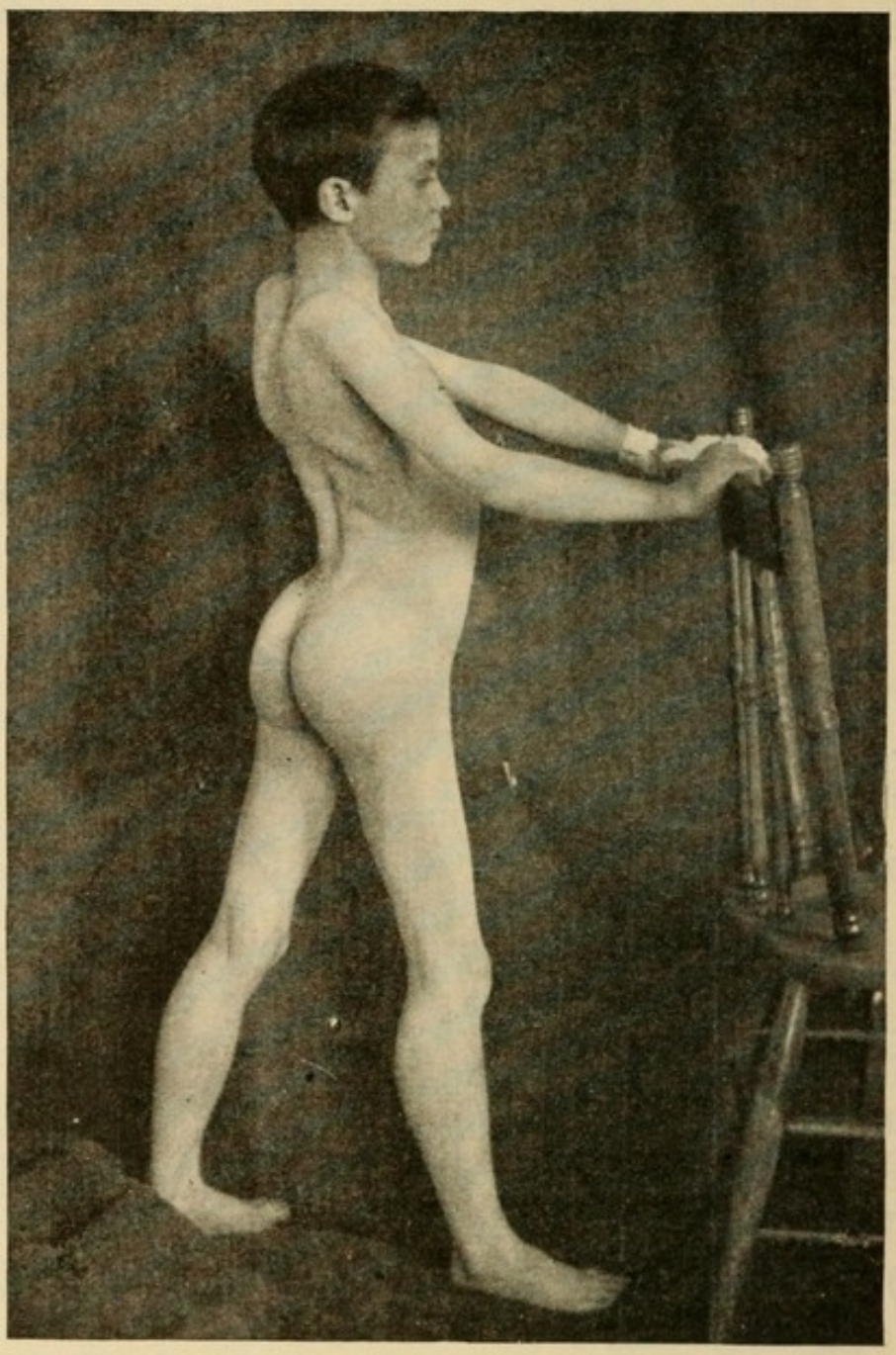


FIG. 52.

Pseudo-hypertrophic paralysis.

muscles of the shoulder girdle in Fig. 52. In rare instances the muscles are at no time larger than normal.

(*c*) A peculiar difficulty in rising from the ground, due to weakness of the extensors of the knee and hip. The hands are placed upon the knees in rising, to transfer part of the weight of the body from the upper to the lower part of the femur,—thus giving the extensors of the knee a distinct mechanical advantage. This and other peculiarities in rising have justified the saying, "the patient climbs up on himself." The difficulty is highly characteristic of pseudo-hypertrophic paralysis, but is not quite pathognomonic.

(*d*) After the disease has lasted for a variable period (several years), contractions occur in the affected muscles (this contraction in the calf muscles causes talipes equinus). Often lumbar lordosis is a late symptom.

(*e*) The knee-jerk is usually lost when the extensors of the knee are involved. The electrical reactions are normal, except in a late stage of the disease, when there is often diminished electrical irritability to both currents. There is no disturbance of sensibility and there are no fibrillary contractions.

(*f*) The disease is slowly progressive.

(*g*) The disease is one of early youth, and is generally inherited through the mother, though the mother may not herself be a subject of the disease.

Pseudo-hypertrophic paralysis is sometimes confounded with cases of spastic paraplegia (*vide* p. 429). The two conditions resemble one another in that there are weakness in the lower extremities and contraction of the calf muscles, in that the calf muscles may be large in each, and in the fact that both occur in children. In spastic paraplegia, however, there is active contracture of the calf muscles, which can be overcome by pressure, the gait is spastic, the knee-jerks are increased, there is ankle clonus, and there is no difficulty in rising from the ground.

Erb's Type of Progressive Muscular Dystrophy

(so-called "juvenile form" of progressive muscular dystrophy).—This is an exceedingly rare form of muscular atrophy possessing the following characters:

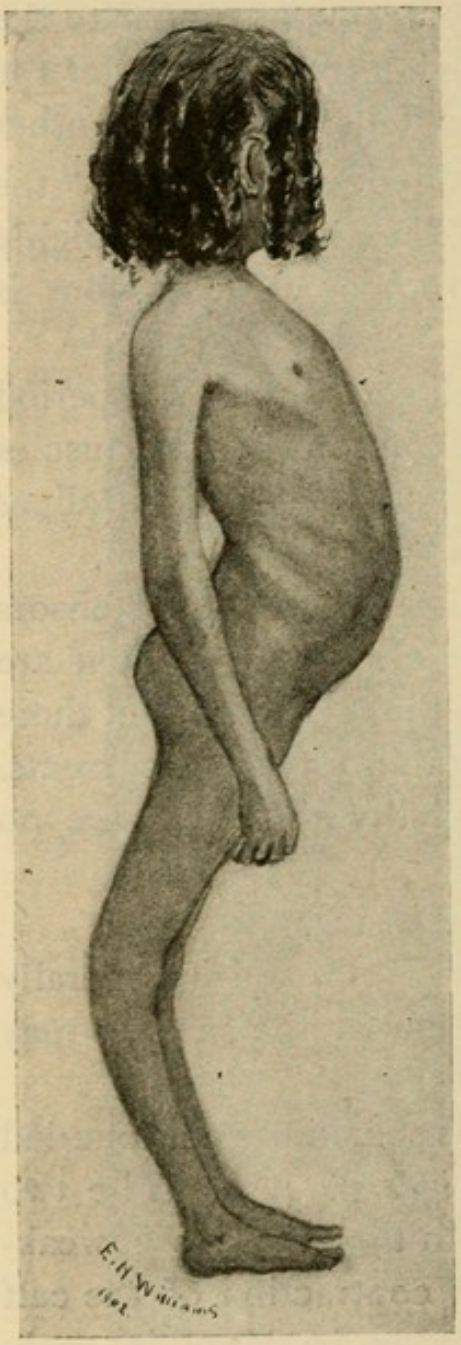


FIG. 54.

Erb's type of progressive muscular dystrophy.

(*a*) There is progressive atrophy and weakness in many groups of muscles, especially the muscles of the shoulder, the upper part of the arm, the pelvic girdle, the thigh, and the back. Most often the atrophy begins in the arm. The fore-arm and leg muscles suffer very late, or not at all.

(*b*) The atrophy of the muscle fibres may or may not be associated with pseudo-hypertrophy of some of the muscles—usually there is no pseudo-hypertrophy.

(*c*) Fibrillary contractions do not occur in the atrophied muscles.

(*d*) Galvanic and faradic irritability are reduced in the affected muscles in proportion to the degree of wasting. The RD is never present even in slight degree.

(*e*) The disease usually affects several members of the

same family, and generally begins in infancy or the adolescent period of life.

The Landouzy-Déjérine Type of progressive muscular dystrophy resembles Erb's type very closely. As in Erb's type, there is widespread, slowly progressive muscular atrophy and weakness, fibrillary contractions never occur, there is diminished electrical excitability of the muscles but no RD, the affection is one of early life, and it occurs usually in families.

The chief point of difference between the two types is that in the Landouzy-Déjérine type the muscles of the face, shoulder, and arms are involved in the atrophic process, as shown in Fig. 55. The involvement of the face muscles is such that it gives to the patient a peculiar dull expression—the "myopathic facies" it is termed. In consequence of the implication of the orbicularis oris the lips are habitually separated, the lower lip is unduly prominent, and the patient is unable to "pout" with the lips. With the exception of the involvement of the face, the distribution of the muscular atrophy is much the same as in Erb's type. The atrophy usually begins in the face, and in the cases where it begins elsewhere the face suffers early. It is said that there is never any pseudo-hypertrophy of muscles in cases of progressive muscular dystrophy in which the face is involved, but this is not so, as shown in Fig. 55 (p. 484).

Both the forms of progressive muscular dystrophy that have been thus briefly touched upon (Erb's type and the Landouzy-Déjérine type) are sometimes observed in members of the same family, and this is another reason for hesitating to separate the two types except for convenience.

Other forms of progressive muscular dystrophy, based on the locality in which the atrophy commences, have

been described, but when the validity of these forms is tested by their association with differences in etiology, there is found to be little reason for making any further separation into types—even for purely clinical purposes.

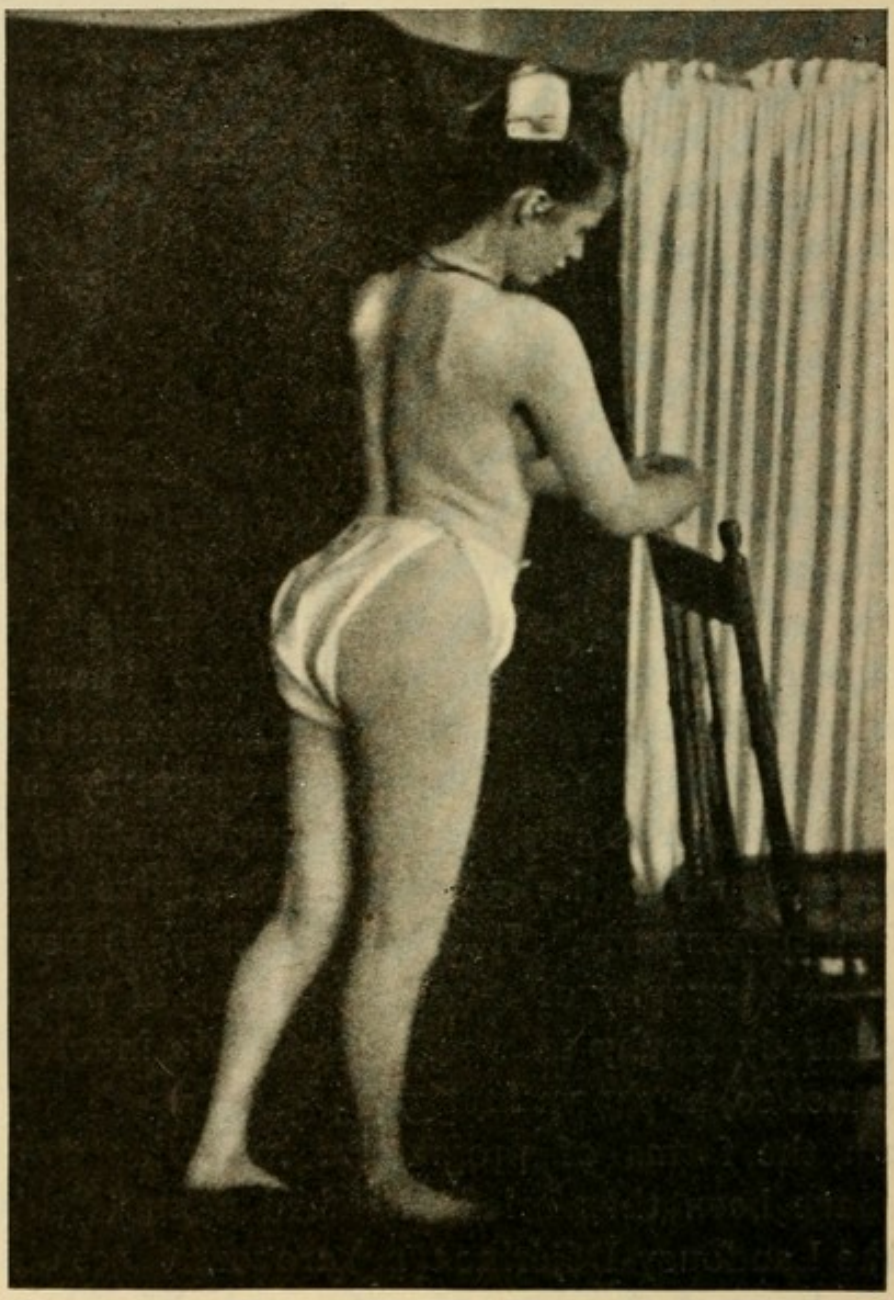


FIG. 55.
Landouzy-Déjérine type of progressive muscular dystrophy.

Intra-Spinal Tumor.—Tumors within the spinal canal vary considerably as to the symptoms they produce.

according as they originate in the cord on the one hand, or in the spinal membranes or the vertebræ on the other. Yet even in the cases which present the fewest clinical features in common, there is a resemblance sufficiently close to make it desirable to consider both classes of cases together.

The chief symptoms of intra-spinal tumor are :

(*a*) Pain, severe and constant at the level of the lesion and in the parts below it. Usually the pain is first felt on one side and extends after a time to the opposite side. The spine may be locally tender, and there is often a distressing girdle pain.

(*b*) Paralysis, of gradual onset and slow but certain progression. The progress of the paralysis is rather characteristic : one leg is first involved, then the paralysis extends with variable rapidity to the other leg, and the patient eventually becomes completely paraplegic. In some cases one leg suffers very soon after the other, but usually the extension to the other leg is not rapid. Sometimes the tumor causes a rather rapid increase in the paralysis,[1] instead of the more characteristic gradual progression.

These two symptoms, pain and paralysis, commencing on one side, and extending to the other, may be called the cardinal symptoms of intra-spinal tumor. Besides these there are other symptoms of considerable though less diagnostic importance. The chief of these are rigidity of the muscles of the spine and legs, contractures and spasmodic movements in the limbs, sensory disturbances, increased knee-jerks and ankle clonus and increased skin-reflexes. The exact distribution of the symptoms of course varies with the position of the tumor. The

[1] This depends not on the destruction caused by the tumor itself, but on the myelitis which the growth causes.

nature of these variations may be largely inferred from what has been said elsewhere about unilateral lesions and total transverse lesions of the spinal cord (see p. 241 and 245). When the lesion is above the lowest third of the lumbar cord and involves mainly one half of the cord at its level, the typical Brown-Sequard paralysis may be produced (see p. 241). Usually the lesion at no time involves exactly one half of the cord, and one therefore meets with many deviations from the typical Brown-Sequard paralysis. Late in the progress of the disease a considerable part of the transverse extent of the cord is often involved, and the well-known and readily intelligible symptoms of a total transverse lesion are then more or less closely reproduced (see p. 245). The nature of the variations that arise, according to the vertical position of the tumor within the spine (cervical paraplegia, dorsal paraplegia, lumbar paraplegia), need not be touched upon here.

When the presence of an intra-spinal tumor seems probable, an endeavor must be made to determine whether the growth springs from the cord or from the membranes or vertebræ. Very often it is impossible to determine this. When the diagnosis is possible, it is based, not upon any one peculiarity of the symptoms, but upon the consideration of a variety of elements in the case.

The conditions that point to the existence of a *tumor of the membranes* or the bones are : (1) great severity of the root symptoms (pain, spasm) ; (2) the late development of cord symptoms (paralysis and anæsthesia below lesion) ; and (3) the unilateral distribution of cord symptoms for some time (many months) after cord symptoms first appear. On the other hand, the conditions that *indicate a cord tumor* are as follows : (1) the absence and trifling character of root symptoms, such as radiating

pains and early spasm; (2) the early development of cord symptoms; and (3) the rapid extension of cord symptoms from one side to the other (two weeks to several months).

The diagnosis of multiple tumors can be made only by the successive appearance of focal symptoms that differ considerably in seat, but agree in character. The opportunities for such a diagnosis are extremely rare.

The diagnosis of the nature of the intra-spinal mass is almost always difficult. The causal indications sometimes help very much. The existence of constitutional syphilis or a history of such disease makes syphiloma likely. A tubercular history makes solitary tubercle likely. In almost all cases of solitary tubercle of the cord there is pulmonary tuberculosis, and in many cases the physical signs are unequivocal. The presence of multiple sarcomata or neuromata in other parts of the body furnishes an exceedingly important diagnostic indication, but it is only rarely available. A tumor of the membranes may be almost anything but glioma and tubercle, but is usually a sarcoma. A tumor of the cord is probably a syphiloma or massive tubercle, perhaps a glioma.

The distinction between caries of the spine with nervous symptoms and intra-spinal tumor is sometimes difficult at first. The important points in diagnosis are the following: in caries root-pains are rarely severe, the symptoms of compression of the cord are bilateral from the first, or become so very soon after their beginning, and the evidences of vertebral disease (local tenderness, irregularity of spines), are early present.

Sometimes the diagnosis between chronic transverse myelitis and intra-spinal tumor, especially tumor of the cord, is very difficult. The chief points of difference

are : (1) that root symptoms of considerable severity are seldom absent in tumor, while they are often absent or slight in chronic myelitis, in which there is more apt to be a constant girdle-pain ; and (2) that the symptoms of tumor are usually actively progressive, while progress in chronic myelitis is generally slow. It is important to remember that considerable acute or subacute myelitis may be the consequence of any tumor that compresses the cord, and that an error in diagnosis may be made if the nature of the original symptoms be not taken into account.

The early nerve-root pains of tumor should not be confounded with neuralgia. The pain of neuralgia is rarely so constant and persistent as that of intra-spinal tumor, and seldom has the same localization and radiating character.

Syringomyelia.—Syringomyelia is a condition of the spinal cord characterized by the presence of one or more pathological cavities within the substance of the cord. Such cavities may originate in two ways. First, they may depend on faulty closure of one of the divisions of the primary central canal of the cord.[1] Secondly, they may depend on the disintegration of a gliomatous formation, which originates, usually, in embryonal tissue about the central canal. A sharp separation of these two forms of syringomyelia is not feasible, since it is probable that in some instances at least, the neurogliar hyperplasia originates in superabundant epiblastic tissue left behind during the defective development of the central canal.

[1] In the course of development the primary central canal of the cord becomes divided into two parts, an anterior and a posterior. The anterior division forms the permanent central canal. The walls of the posterior division gradually come together and form the posterior fissure. The imperfect closure of either of these divisions of the primary central canal may give rise to syringomyelia. The word hydromyelus is sometimes used to designate a cavity of congenital origin.

The cavity in syringomyelia varies much in extent and situation in different cases. It encroaches very irregularly on the gray and white substance of the cord, and there is consequently much variety in the nature and distribution of the symptoms to which the condition gives rise. So indistinctive are the symptoms of syringomyelia that it is impossible in many instances to recognize the condition during life.[1] In a certain number of cases, however (probably a minority), there are peculiarities in the symptoms that enable the diagnosis of syringomyelia to be made with a considerable degree of confidence.

The following are the chief symptoms of typical syringomyelia :

(1) Slow muscular atrophy resembling in its characters that of progressive muscular atrophy. This atrophy depends on the involvement of the anterior horn, and may be uni- or bilateral, according to the position of the cavity or the gliomatous infiltration in which the cavity formation is going on. The atrophy often commences in the arm muscles, and is apt to involve them in much the same order as in the Aran-Duchenne type of progressive muscular atrophy (p. 440). This almost regular involvement of the arm muscles corresponds to the fact that in the cervical region the cord is usually most extensively involved by the lesion. The wasted muscles are the seat of fibrillary contraction, and very often present the RD, especially in its partial forms (see p. 198). The loss of power in the wasted muscles is proportional to the atrophy, the wasted muscles are flaccid, and the triceps-jerk is lost. In a large majority of cases the muscles of

[1] Sometimes the first intimation of the nature of the case is found at autopsy. In many cases in which the diagnosis of syringomyelia has been made by good observers the autopsy has failed to confirm this diagnosis.

the face and lower extremity are not implicated in the atrophy.

(2) Sensory symptoms of peculiar and distinctive character. One of the earliest symptoms is a loss or diminution of sensibility to temperature (heat and cold), and generally to pain, *without* interference with the tactile sense. This peculiar partial or dissociated sensory disturbance corresponds in a rough way to the parts that are the seat of atrophy. Thus, if the atrophy affects the upper extremity, the sensory loss involves the skin of this part, and probably extends over the shoulder and upper part of the chest. The sensory loss is not distributed in patches, but covers a continuous though often irregular area. When the atrophy is extensive and involves the face, the latter may be the seat of sensory disturbances. Sometimes the peculiar sensory loss involves one entire lateral half of the body, the mucous membrane of the lips, eyes, and anus being also affected. It occasionally happens in advanced cases of syringomyelia that also tactile sensibility is lost. Pain in the cervico-brachial region may be among the earliest symptoms of syringomyelia.

(3) Trophic disturbances, other than muscular atrophy, are often among the earliest symptoms. The vitality of the atrophied parts is almost always lessened, and the loss of sensibility to pain renders the skin of these parts especially liable to suffer from slight injuries, which lead to ulcerations, eruptions, etc. True trophic disturbances, such as overgrowth of the skin and nails, glossy skin, nutritive alterations in the bones, whitlows, etc., are not of rare occurrence. Œdema, and changes in the temperature and color of the affected limbs are very common.

Among the conditions of secondary diagnostic importance that are observed in syringomyelia may be mentioned scoliosis of the vertebral column. The knee-

jerk is often increased on the side affected, from interruptions of the pyramidal tract. When the lumbar region is implicated the knee-jerk is lost. The deep reflexes of the upper extremity are lost when the disease is in the cervical region. The etiology of syringomyelia is too obscure to be of aid in diagnosis. One fact, however, regarding its occurrence, it is well to note. The disease almost always begins between the fifteenth and thirtieth years of life.

The peculiar sensory disturbances of syringomyelia serve to distinguish it from progressive muscular atrophy, which it may resemble very closely in the character and distribution of its atrophies. It is probable that the sensory disturbances of syringomyelia may be absent at times during the early stage of the disease,—*i.e.*, before the neurogliar hyperplasia has begun to undergo the disintegration that leads to cavity formation; but there is excellent reason to believe that whenever there is recognizable muscular atrophy in syringomyelia there is also disturbance of sensibility. Hence, in cases of slow muscular atrophy in which there arises the question of a diagnosis between progressive muscular atrophy and syringomyelia, the condition of sensation is to be allowed great weight in reaching a decision.

The cavity formation in syringomyelia may depend not on the breaking down of a diffuse gliomatous infiltration, but on the disintegration of a more or less limited gliomatous mass—*i.e.*, a glioma. In such cases the more distinctive symptoms of syringomyelia (local muscular atrophy, partial sensory disturbances, and trophic changes) may be preceded by the symptoms of a tumor of the substance of the cord (see p. 440).

The common form or multiple neuritis (alcoholic form) is sufficiently distinguished from syringomyelia by its

subacute development, and by the presence of sensory loss of the ordinary type (tactile pain and temperature sense suffering about equally). Another form of multiple neuritis, namely, anæsthetic leprosy, may be confounded with syringomyelia. The features which the two conditions may possess in common are muscular atrophy of the Aran-Duchenne type, sensory disturbances, and trophic changes,—perhaps including loss of some of the phalanges. The most important point in the distinction of the two conditions is the fact that in anæsthetic leprosy the areas of anæsthesia are small and sharply circumscribed. Usually, too, all forms of sensibility suffer in leprosy ; but too much importance must not be given this fact, for cases occur in which there is the same partial sensory loss that is so strongly suggestive of syringomyelia. The presence of the characteristic lepra nodules on some part of the body may determine the diagnosis.

There is strong reason to believe that the condition known as Morvan's disease depends on syringomyelia. The chief characteristic of Morvan's disease is the occurrence of multiple and recurrent whitlows, accompanied with paresis, muscular atrophy, and analgesia. Both upper extremities are usually involved. It has been thought that the occurrence of whitlows is distinctive of Morvan's disease, and that the occurrence of dissociated sensory symptoms is pathognomonic of syringomyelia. But it is certainly true that whitlows occur in undoubted syringomyelia. It is also true that dissociated anæsthesia was present in some admittedly typical cases of Morvan's disease.[1] Probably the most rational view of

[1] Dissociated anæsthesia (loss of pain and temperature sense, with preservation of tactile sense) has been occasionally observed in hysteria, hemorrhage into the cord, locomotor ataxia, and in compression of the cord and nerve-roots. It is therefore very far from being a pathognomonic symptom.

the matter is that what is called Morvan's disease is merely syringomyelia with preponderating trophic symptoms.

Diseases of the Peripheral Nervous System.

Neuritis.—The symptoms of *neuritis* (or inflammation of nerves) are chiefly local, and vary in distribution with the nerve or nerves affected. Of these local symptoms the following are the principal :

(1) *Pain* is by far the most obtrusive symptom of neuritis. The nerve inflamed is always the seat of pain, which may be intense, is often burning or boring in character, and is increased by anything that causes passive congestion of the part affected. Frequently the pain is not confined to the *nerve* but is present in the parts to which it is distributed.

(2) *Pressure upon the nerve at the part inflamed increases the pain.*

Besides these symptoms, which are present in every case of neuritis, there are others which, though less constant, are of considerable importance and may be of aid in the diagnosis of neuritis. Thus, if the nerve is accessible, it may often be found to be *swollen* at the part inflamed. Besides pain there may be various other sensory disturbances ; numbness, tingling, and hyperæsthesia are common in the distribution of the affected nerve. After some time these symptoms may be followed by anæsthesia, which is not complete except in small irregular areas. In many cases of neuritis of motor or mixed nerves there are decided motor symptoms, including fibrillary contractions in the affected muscles, tremor, loss of power (sometimes complete paralysis), and muscular atrophy. The electrical reactions vary in different cases, ac-

cording to the severity of the process and the character of the nerve involved, from no alteration whatever to complete RD. Trophic and vaso-motor symptoms are usually present. Especially common are the changes in the nutrition of the skin which cause the condition known as "glossy skin." Eczema, pemphigoid and herpetic eruptions and ulcers are much less common. The temperature of the parts may be changed—at first there may be increase in temperature, later a lowering of temperature may occur. Changes in the joints with resulting limitation of movement are of frequent occurrence.

In acute cases of neuritis there may be some constitutional disturbance at the outset. In cases that are chronic from the beginning such symptoms are absent. The pain in chronic cases is less severe than in acute cases. In cases of slight neuritis the symptoms may subside after a few weeks, but in most cases the disease runs a much longer course, and the majority of acute cases pass into a chronic stage. In some cases of neuritis the symptoms extend up the limb affected ("neuritis migrans"), and if the inflammatory process ascends to the plexus from which the nerve is given off, all the nerves of an extremity may be affected. In some cases (especially cases of so-called reflex bladder paralysis) the spinal cord or its membranes become involved (myelitis, meningitis), and through this involvement certain nerves of the opposite side may become the seat of neuritis. The variety of neuritis in which several nerves, in symmetrical parts, are simultaneously involved (multiple neuritis) will be separately considered.

In most cases in which the symptoms point to the existence of neuritis a cause for the condition can be found. Most often this is injury of some kind (often slight); sometimes it is adjacent inflammation (as of *a*

joint). Sometimes the cause is a preceding acute infectious disease, or a blood disease, or exposure to cold, but not very rarely no adequate cause can be traced.

The distinctive localization of the symptoms of neuritis to the distribution of a particular nerve or combination of nerves, serves to separate neuritis from the pains of acute rheumatism. The acute pains of some diseases of the spinal cord (especially locomotor ataxia, myelitis, etc.) may be mistaken for the pains of neuritis, but the absence of permanent local tenderness, the irregular distribution of the pains, and usually their paroxysmal nature, will prevent an error. Moreover, in spinal cord disease evidences of the central character of the disease are seldom wanting.

It is exceedingly important and not always easy to distinguish between certain cases of chronic neuritis and others of neuralgia. The considerations on which the diagnosis is based are considered elsewhere (Chapter VI.—Neuralgia).

Diseases of Special Nerves.—It is convenient to review the clinical forms of disease that depend on morbid processes affecting special nerves in the anatomical order of these nerves, commencing with the cranial nerves. The diseases of cranial nerves that subserve special forms of sensibility (olfactory, optic, auditory) have already been sufficiently considered elsewhere, (Chapters III. and IV.).

Paralysis of the Ocular Muscles.

Paralysis of the Third Nerve (Oculo-motorius Paralysis.)—The special symptoms (exclusive of symptoms common to all ocular palsies—strabismus, diplopia, secondary deviation) of paralysis of the whole of the third nerve are:

(1) *Entire loss of upward and inward movement* of the eye from paralysis of the superior and internal rectus and

inferior oblique (which helps to elevate the eyeball), and *partial* loss of downward movement from paralysis of the inferior rectus (the superior oblique being capable of moving the eyeball slightly downward and outward).

(2) *Ptosis*, from paralysis of the levator palpebræ superioris.

(3) *Loss of pupillary reaction to light*, from paralysis of the sphincter iridis.

(4) *Loss of the power of accommodation* from paralysis of the ciliary muscle.

Usually all the muscles supplied by this nerve are affected in a peripheral palsy, but they may suffer unequally and some may escape altogether; not rarely the levator and superior rectus are chiefly involved, and sometimes both escape together.

Paralysis of the Fourth Nerve, supplying only the superior oblique muscle, causes a partial loss of movement of the eyeball in a downward and inward direction. This defect in movement causes convergent strabismus when the patient looks down, and diplopia, which may be very troublesome, when the patient goes down stairs.

Paralysis of the Sixth Nerve, supplying only the external rectus, gives rise to a defect in the outward movement of the eyeball, with convergent strabismus and diplopia when the patient looks *to* the side of the paralyzed muscle. The head is usually held inclined *to* the side of the paralyzed rectus.

Having established the presence of paralysis of one or more of the motor ocular nerves, it remains to determine the exact seat of the lesion and the nature of the morbid process causing the paralysis. Paralysis of ocular muscles may be the result of (1) disease of the nerves themselves, or (2) of the fibres of origin between the superficial origin and the nucleus, or (3) of the nuclei of the

nerves. We have to do here only with disease of the *nerves* themselves, the other conditions (2 and 3), and the diagnosis between them and lesions of the nerve trunks being considered elsewhere (see Nuclear Ophthalmoplegia).

If one of the three oculo-motor nerves (3d, 4th, or 6th) is paralyzed *alone*, it is extremely probable that the lesion causing it *is at the base of the brain.* In the case of the third nerve, the nerves of both sides may be affected and the lesion nevertheless be a basal lesion, for these nerves lie very close together in the space between the peduncles (*vide* Fig. 13).

But in the case of the fourth and sixth nerves, which are widely separated at their superficial origins and from their mates of the opposite side, the involvement of the nerves of opposite sides almost always excludes a lesion of the trunk. The sixth nerve has a long course between its superficial origin and its passage through the sphenoidal fissure, and therefore suffers more frequently than any other ocular nerve. As it passes near the fifth nerve in its course *over* the pons, these two nerves may suffer together, and this combination points certainly to a basal lesion. Paralysis of the sixth, seventh, and eighth also indicates a basal lesion, but paralysis of the sixth with the seventh and *without* the eighth, shows that the lesion is not at the base, but within the pons. If *all* of the motor ocular nerves of *one* eye are involved, the disease is certainly at or near the sphenoidal fissure. These indications will serve, in most instances, to locate the position of the morbid process.

Various morbid processes cause paralysis of the oculo-motor nerves. Probably the most frequent causes are syphilitic new growths and chronic syphilitic meningitis at the base of the brain. When the nerves are damaged

in the orbit it is usually as the result of cold (so-called "rheumatic" neuritis), or of cellulitis, or of an orbital growth. When third-nerve paralysis depends on "rheumatic neuritis" the paralysis is always one-sided, and usually only one nerve is involved, or a single branch of a nerve, or two or more adjacent branches. The onset of such cases is generally attended with pain about the orbit. Bilateral partial ophthalmoplegia probably sometimes depends on alcoholic neuritis. Occasionally diphtheria is followed by paralysis (usually partial) of the external (as well as the ciliary muscle) muscles of the eyeball. Usually the internal recti suffer most (or alone), but all the muscles may be involved. Similar paralysis may follow influenza.

The ocular paralysis that so frequently accompanies locomotor ataxia is usually of central (nuclear) origin, and it is uncertain whether it is ever the result of peripheral disease. The decision as to the nature of the lesion (new growth, aneurism, gumma, chronic syphilitic meningitis, etc.), in a given case of ocular paralysis from disease within the cranium, will depend on the application of principles that have been already stated (see Diagnosis of the Nature of the Lesion; also Intracranial Tumor, Meningitis, and Aneurism). Not rarely it is impossible to determine with certainty the nature of the lesion.

Paralysis of the Fifth Nerve.—The symptoms of paralysis of the fifth nerve vary according as they result from disease of the sensory or motor root or from disease affecting both roots.

The chief symptoms of disease of the *sensory division* are:

I. *Loss of sensibility* in the parts supplied by the fifth nerve. This loss of sensibility may involve the entire region supplied by the fifth when there is disease of the

root of the nerve, or when all three branches (first, second, and third) are involved by a lesion at the base of the brain. When individual branches are involved the anæsthesia is limited to the regions supplied by them. There is also loss of sensibility in the mucous membranes supplied by the nerve (nose, tongue, and mouth on the side of the lesion). In some cases the back of the tongue and the soft and hard palate share in the sensory loss, but in others (even where the entire nerve is probably involved) only the anterior two thirds of the tongue is insensitive.

II. *Pain*, often sharp and burning, may precede the development of anæsthesia.

III. *Loss of taste.* Loss of taste on one side of the tongue and palate results from severe disease of the sensory portion of the fifth. When taste is not involved in lesions of the fifth it is probably because the disease is partial or the lesion is within the pons. Loss of taste confined to the anterior two thirds of the tongue occurs where the lingual branch of the fifth is diseased between the peripheral distribution and its junction with the chorda tympani (Fig. 54).

IV. *Trophic changes.* The chief trophic alterations that occur in consequence of disease of the sensory portion of the fifth nerve are those of the eye. The cornea becomes dry and opacities appear; ulceration occurs, and this may go on to perforation and eventual destruction of the globe of the eye. These ocular changes are especially frequent in irritative disease involving the Gasserian ganglion or the nerve anterior to it, and are rare in lesions between the ganglion and the superficial origin of the nerve from the pons. Another trophic change that results especially from lesions that affect the Gasserian ganglion or the nerve anterior to it is herpes zoster, which most often affects the superior maxillary distribution of the nerve.

The principal symptoms of disease of the *motor* portion of the nerve are weakness of the masseter, temporal, and external pterygoid muscles on the side of the lesion. The weakness of the masseter and temporal muscles is evident when the finger is placed over these muscles and the patient brings the upper and lower teeth forcibly in contact.

The pterygoid paralysis is shown by defective lateral movement of the lower jaw, which can be moved towards but not away from the side of the paralyzed muscle. In the course of a few weeks the paralyzed muscles atrophy and the temporal and zygomatic fossæ show this by growing sunken.

The tensors of the palate and tympanum are supposed to be paralyzed by a lesion of the fifth, but no clinical evidence of it can be detected.

It is probably correct to attribute to disease of the *descending root* (see p. 49) of the fifth the peculiar and rare condition known as facial hemiatrophy, in which there is a gradual and considerable wasting of one side of the face, the bones and subcutaneous tissues being the chief seat of the wasting.

Complete paralysis of the fifth nerve, including paralysis of the motor portion of the nerve, can hardly be confounded with any other condition. When, however, the sensory portion is alone affected and there is anæsthesia of adjacent areas, difficulties in diagnosis may arise.

In hemianæsthesia the loss of sensibility includes the very parts supplied by the sensory portion of the fifth. But in hemianæsthesia the loss involves also the back of the head, the limbs and the trunk on one side, and often there is hemianopsia. When pain is the only symptom of disease of the fifth, as it may be for a time, it is impor-

tant to avoid confounding the condition with neuralgia, which it may closely resemble (see Neuralgia).

The diagnosis of the position of the disease that gives rise to paralysis of the fifth is based mainly on the extent of the anæsthesia and the conditions associated with the paralysis.

Paralysis of all three divisions of the sensory portion, with paralysis of the motor portion of the nerve, points to disease at the base of the brain or possibly at the Gasserian ganglion. If there is also paralysis of the sixth nerve the lesion is surely near the point of emergence of the fifth at the side of the pons. If the loss of function is confined to the distribution of the superior maxillary division of the nerve, the lesion is probably at the sphenoidal fissure or in the orbit, and this belief is strengthened if there is also paralysis of the nerves to the eyeball. If the inferior maxillary division of the nerve is alone affected the lesion is probably in the spheno-maxillary fissure or in the superior maxillary bone. Paralysis of the second and third divisions of the nerve, without involvement of the first division, is generally due to disease in or near the spheno-maxillary fossa.

Crossed paralysis of the fifth nerve (fifth on one side, arm and leg on the other) depends usually on a lesion *within* the pons. The association of conjugate deviation (to the side of the lesion) with paralysis of the fifth is positive evidence of a pontine lesion.

A knowledge of the locality of the lesion aids us in determining its nature. Thus, a pontine lesion causing fifth nerve paralysis is either a vascular lesion (hemorrhage or softening), a tumor, or a sclerotic patch. Which of these it is must be decided by the help of other indications (see Chap. IV.). A basal lesion is probably chronic

meningitis, tumor, or caries of bone. Paralysis of the superior maxillary division may depend either on tumor or cellulitis of the orbit, or on carotid aneurism, or on a new growth in the pituitary region. Paralysis of the second and third divisions depends often on tumor of the parotid region or disease of the antrum. Sometimes the fifth is paralyzed from primary neuritis and occasionally from trauma (as in punctured wounds through the mouth).

Paralysis of the Facial Nerve.—Clinically it is important to distinguish two forms of facial paralysis, viz. :

I. *Facial paralysis involving all the muscles supplied by the facial nerve* (see p. 52) including the orbicularis palpebrarum and the frontal portion of the occipito-frontalis. In these cases of complete facial paralysis the voluntary and involuntary movements of the face suffer equally, and in the course of time there are the changes of electrical irritability in nerve and muscle that characterize degenerative processes in nerves. In some slight cases the electrical changes are slight or absent. The senses of taste and of hearing may or may not be affected in this form of paralysis.

All cases of facial paralysis belonging to this group are due to disease of the *lower* segment of the facial path, *i. e.*, to disease (1) of the facial nerve itself (peripheral paralysis), or (2) of the nerve in its course through the pons to its nucleus, or (3) of its nucleus (nuclear paralysis).

II. *Facial paralysis* in which the muscles of the upper part of the face (orbicularis palpebrarum, and frontalis) are little or not at all affected, and in which the paralysis is practically confined to the muscles about the angle of the jaw.

In this form of paralysis the voluntary movements of the face are lost but the *emotional and reflex movements*

of the face are preserved or little affected. Moreover, in these cases the electrical reactions are normal or only slightly changed; there is no RD. This type of facial paralysis always depends on disease of the upper *segment* of the facial path above the facial nucleus (supra-nuclear paralysis) either in the pons, crus, internal capsule, centrum ovale, or the cortex (see Chaps. I. and III.). It is generally associated with other evidences of brain disease and often forms part of typical hemiplegia (p. 74). A rough diagnosis of the position of the lesion that causes facial paralysis may be made from the facts that have just been presented—that is, we may infer from these facts whether the disease is in the upper or lower segment of the facial path. A more accurate localization must depend on other indications. The facts which relate to the exact localization of facial paralysis from disease in the upper segment of the facial path (supra-nuclear paralysis) have been sufficiently considered elsewhere (see Localization).

If (owing to the correspondence of the symptoms to the first type described) it has been determined that the lesion is *not* supra-nuclear, an effort must be made to locate it more exactly by the associations of the paralysis. Thus, if the facial is the only cranial nerve paralyzed and the sense of taste is normal on the side of the paralysis, the lesion is outside the stylo-mastoid foramen or just within the Fallopian canal (see Fig. 54).

If the sense of taste is involved on the anterior two thirds of the tongue on the side of the lesion, the seat of disease is in the Fallopian canal at some point between the junction with the chorda tympani and the geniculate ganglion (where the great superficial petrosal nerve containing the taste-fibres joins the trunk of the nerve (see Fig. 54). Disease of the nerve at the geniculate ganglion

has been thought to cause paralysis of the palate, but it is extremely doubtful if this is so. Inequality of the palate is almost always congenital and has no localizing significance in cases of facial paralysis. If there is complete deafness in the ear on the side of the lesion, and if this deafness dates from the time of the facial paralysis, the disease is in the trunk at the base of the brain, or (in rare cases) in the internal auditory meatus. If paralysis

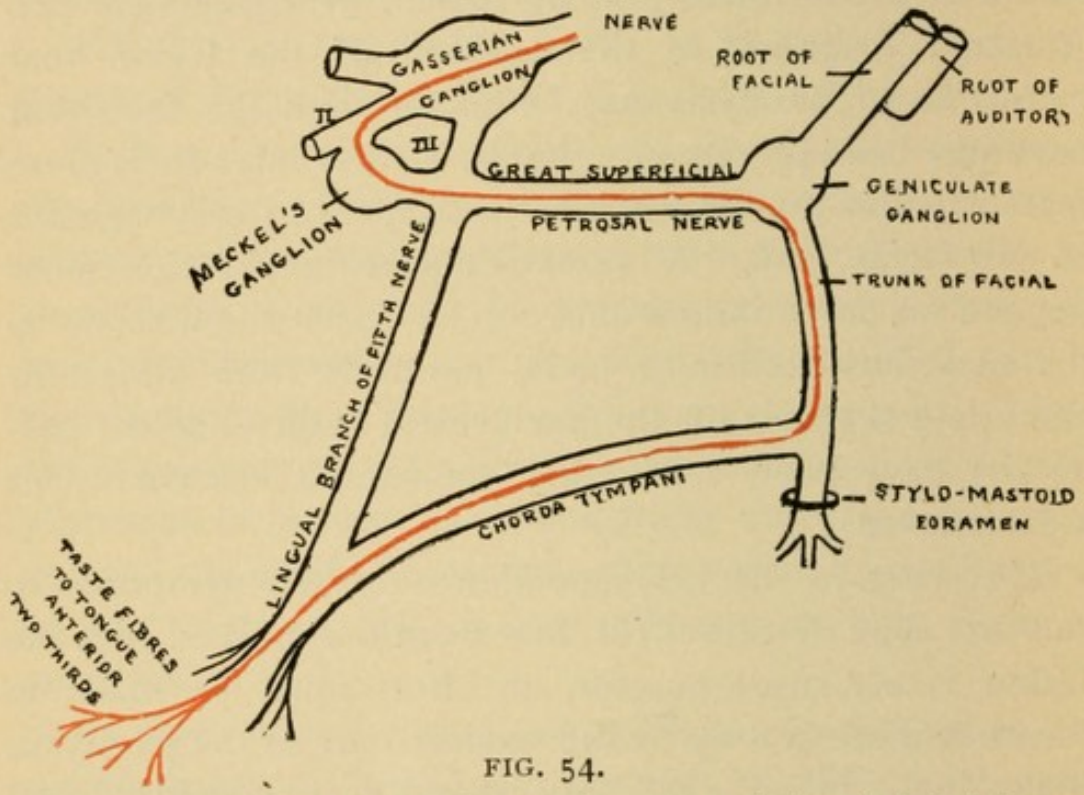


FIG. 54.

Diagram illustrating some of the more important relations of the facial nerve between its superficial origin at the base of the brain and its division into branches after leaving the stylo-mastoid foramen.

of the sixth nerve is associated with paralysis of the facial, the lesion is no doubt in the pons, and in all probability in its posterior part, where the fibres of the seventh pass round the sixth nucleus (see Fig. 30). Crossed hemiplegia also points to a pontine lesion (see Localization). Disease of the facial nucleus is probably indicated where all muscles supplied by the nerve are

paralyzed except the orbicularis oris (which is probably innervated by the twelfth nerve).

Facial paralysis may result from a great variety of morbid processes. Some of these may be briefly enumerated. Disease in the pons (nucleus, nerve, or both) may be hemorrhage, acute softening, degeneration (as in bulbar paralysis), tumor, or inflammation, as in the rare cases of facial paralysis that complicate poliomyelitis. Which of these processes is operative in producing the facial paralysis must of course be determined by the use of other indications of the nature of the lesion (Chap. IV.). When the paralysis is due to a lesion of the nerve at the base of the brain the process is usually chronic meningitis or tumor. When the paralysis is due to disease in the Fallopian canal it may be dependent on a neuritis from cold ("rheumatic neuritis"—very common cause), or it may depend on damage secondary to ear-disease (common cause in children), or to pressure on the nerve from fracture of the petrous portion of the temporal bone in a lesion at the base. Paralysis from a lesion outside the skull is seen in some cases of tumor of the parotid region, in cases of wounds (surgical or accidental) about the ramus of the jaw, and in some cases of pressure from the forceps during delivery.

Most of the causes of facial paralysis that have been mentioned give rise to one-sided paralysis, but in rare instances double facial paralysis is seen. This occurs in some cases of pontine disease, in certain cases of extensive disease (chronic meningitis) of the base, in rare cases of rheumatic neuritis, sometimes from double otitis media, and occasionally in diphtheritic and alcoholic neuritis, in facial erysipelas, and in bulbar paralysis. There is one point in the diagnosis of facial paralysis the neglect of which (though it is very elementary) some-

times leads to error. During the early stage of facial paralysis the drooping of the mouth on one side makes it only too evident that this is the side paralyzed. But after the lapse of five or six months, if there is some recovery of power, contracture develops in the paralyzed muscles (especially marked in the zygomatici), and the angle of the mouth is drawn up on the affected side. Hence an error may be made as to the side affected, unless the patient is made to move both sides of the mouth, when it is immediately evident that the palsy is on the side of the contracture.

The average duration of moderately severe cases of facial paralysis is two or three months. The electrical reactions (see page 190) afford the best means of gauging the severity of the process.

Facial Spasm (tic convulsive, mimic spasm).—This name is given to cases of spasm in the muscles supplied by the facial nerve. The spasm is usually unilateral and affects chiefly the zygomatici and the orbicularis palpebrarum. It is generally clonic in character, but may be tonic, or tonic and clonic. Usually the disease is primary. Sometimes, however, it occurs as a result of facial paralysis.

Primary facial spasm must be distinguished from that which is secondary to facial paralysis, and the distinction is based chiefly on the absence of *paralysis* and on the absence of secondary contracture. Primary facial spasm is easily distinguished from cases of chorea in which the face is chiefly affected, by the fact that in these cases of chorea the spasm is never *confined* to the face. "Blepharospasm," or spasm limited to the eyelids, differs from primary facial spasm (though it is indeed a variety of such spasm) in its limitation to the eyelids, its bilateral

distribution, and its dependence upon a reduction in the general health.

Having concluded, in a given case, that the facial spasm is primary, it is important to determine the position and character of the disease. Facial spasm may occur from disease of the cortical face centre, from disease of the facial nucleus in the pons, or from disease of the trunk of the nerve. Sometimes the disease is organic in nature; more often it is functional or nutritional. If paralysis of the facial muscles *follows* the spasm, the disease is probably organic, and in such a case other evidences of an organic lesion are usually present. If the spasm includes both the corrugators and other facial muscles on *one side* only, the disease is probably cortical. Cortical disease may, however, cause partial and strictly unilateral facial paralysis. If the spasm appears due to cold or to neuralgia, there is probably functional derangement of the nucleus. If there is no apparent cause for the spasm, or if it appears related to mental influences (emotion, etc.), the disease (nutritional) is probably in the cortical cells or perhaps in the cells of the facial nucleus.

Paralysis of the Glosso-Pharyngeal Nerve.—The ninth nerve seldom suffers alone and the symptoms of its isolated paralysis are not definitely known. Probably it causes loss of sensibility and partial loss of power in the upper part of the pharynx. The nerve is not rarely diseased in company with other cranial nerves, and is especially apt to suffer in disease of the nerve-roots of the medulla (tumor, meningitis).

Paralysis of the Pneumogastric Nerve.—Owing to the varied distribution of the tenth nerve disease involving it may give rise to many different symptoms, according to the position and nature of the morbid process. Generally

speaking, the chief symptoms of paralysis of the nerve are (1) paralysis of the larynx, (2) increased frequency of the pulse, and (3) retardation of respiration. With these symptoms of paralysis may be associated the symptoms of irritation of the tenth, of which the chief are (1) laryngeal spasm, and (2) vomiting. Some of these symptoms have been sufficiently described elsewhere (see Laryngeal Paralysis, Laryngeal Spasm, Cheyne-Stokes' Respiration.) It is necessary to mention here only two other results of pneumogastric derangement, paralysis of the pharynx and spasm of the pharynx. *Paralysis of the pharynx* is shown by the inability of the patient to swallow food, which, when placed in the pharynx remains there. Pharyngeal paralysis may result from disease of the nuclei of the tenth, or from disease at the base (meningitis, tumor), involving the roots at the side of the medulla, and sometimes from diphtheric neuritis. Unless the pharyngeal nerves are involved on *both* sides the difficulty in swallowing is slight.

Spasm of the pharynx never results from organic disease. In some cases of hysteria it is associated with a sensation as of a large mass in the pharynx, known as the "globus hystericus." Its nature is readily recognized by the existence of other hysterical symptoms. The spasm is of short duration in these cases. Pharyngeal spasm is also seen in some cases of hydrophobia.

The diagnosis of the exact position of the lesion which causes vagus paralysis depends largely, as in many other cranial nerve paralyses, on the associated conditions. The roots of the nerve at the side of the medulla and the branches of the nerve are much more often the seat of disease than the trunk of the nerve. If the lesion involves the roots at the side of the medulla, there are the signs of implication of other nerves, especially the

hypoglossal. The recurrent laryngeal nerves are the branches of the vagus most often involved, and the symptoms attending their disease have been already described (see p. 135). The causes of damage to the vagus are numerous. Disease of the nucleus, which is usually bilateral, may be either softening, hemorrhage, or degeneration, and in most cases is associated with the signs of involvement of adjacent nuclei in the medulla. When the nerve is injured at the side of the medulla the lesion is commonly meningitis or tumor, sometimes vertebral aneurism. The trunk of the nerve is damaged occasionally by wounds of the neck, sometimes inflicted during surgical operations; sometimes the trunk is compressed by new growths of the neck, enlarged nodes, etc. The recurrent laryngeal is often compressed by aneurism (especially on the right side) within the thorax, and sometimes by cheesy nodes or new growths in the chest. The diagnosis in such cases is greatly aided by the presence of physical signs of thoracic disease, but sometimes such signs are absent and the diagnosis is difficult. Neuritis of the vagus is the lesion in some cases of diphtheritic paralysis (which is very apt to involve the vagus) and in the cases of locomotor ataxia in which laryngeal "crises" occur. It is said that in rare instances exposure to cold may cause symptoms of vagus derangement from neuritis of the trunk.

Paralysis of the Spinal Accessory Nerve.—Paralysis of the "accessory" portion of the eleventh nerve (see Anatomy) gives rise to paralysis of the larynx (see p. 135), and need not be considered here. Paralysis of the "spinal" portion of the nerve, which supplies the sterno-mastoid and trapezius muscles, causes characteristic symptoms dependent on the loss of function of these muscles.

Each *sterno-mastoid* muscle by its contraction rotates

the face towards the *opposite side*, while it draws down the head *to* the side on which it contracts. If *one* sterno-mastoid muscle is paralyzed it produces little or no effect on the position of the head or in its movements, as the loss is compensated by other muscles. The defective rotation to the opposite side may, however, be easily determined. The unparalyzed muscle may incline the head to the side opposite the paralyzed muscle, by its contracture. The fact that mere paralysis of the sterno-mastoid causes no deformity when the head is at rest is of some practical importance. When both sterno-mastoids act they support the head in the anatomical position, and when the head is put back they together draw it forward. Paralysis, therefore, of *both* sterno-mastoids makes it very difficult for the patient to bring forward the head when it falls back. The sterno-mastoid atrophies when it is paralyzed.

The *trapezius* consists of three portions, an upper, a middle, and a lower portion.[1] Disease of the eleventh causes paralysis of the *upper* part and partial paralysis of the middle part. The upper part is used mainly during respiration, and its paralysis is shown chiefly by a sinking in of the muscle which causes a change in the contour of the neck, especially marked during deep respiration. Paralysis of the middle portion of the trapezius (which elevates the scapula and the shoulder, and rotates the scapula with the lower angle out), causes a dropping of the scapula and shoulder and a rotation in of the lower angle of the scapula, with defective elevation of the arm. This middle part of the trapezius is, however, only partially paralyzed in spinal-accessory disease.

[1]It is now generally believed that branches from the lower cervical and upper dorsal nerves only pass through the trapezius muscle, and that this muscle is wholly supplied by the spinal accessory.

The parts affected waste and show altered electrical reactions, which vary with the character of the disease causing the paralysis. Bilateral trapezius paralysis causes a falling forward and sinking of the head, the chin resting on the sternum.

The precise distribution of the paralysis of spinal accessory disease varies with the position of the lesion. When disease (usually degenerative) involves gray matter from which the spinal accessory arises, the paralysis is never confined to the sterno-mastoid and trapezius, and the affection is bilateral (see Bulbar Paralysis). The entire trapezius is usually involved in degenerative disease of the gray substance. When the nerve-trunk is the seat of the lesion, the paralysis involves the sterno-mastoid and the *upper* part of the trapezius. If the spinal accessory nerve is damaged in its course within the skull, both portions of the nerve (spinal and accessory) may be involved. There is then paralysis of the larynx on the side of the lesion, in addition to the muscular paralyses that characterize spinal accessory disease. With these symptoms there may be associated paralysis of one half of the tongue, owing to the proximity of the eleventh and twelfth nerves. Paralysis of the palate (levator palati) on the side of the lesion is sometimes seen in these cases, and may depend on disease of the eleventh or perhaps on disease of the twelfth nerve. When the eleventh nerve is damaged, as is sometimes the case, after it has passed through the sterno-mastoid, the paralysis is confined to the trapezius.

Spasm of the Muscles Supplied by the Spinal Accessory Nerve ("Wry-Neck," "Torticollis").—The term "wry-neck" is applied to several different affections in which muscular contraction produces an abnormal position of the head. In some cases the unnatural position of the

head depends on persistent shortening of a muscle (usually the sterno-mastoid). Such cases are usually congenital, and this form of wry-neck is known as congenital or fixed wry-neck. In other cases, which are of much greater importance from a diagnostic point of view than the congenital cases, the unnatural position of the head depends, not on muscular contraction, but on some quite different condition—usually on caries of the upper cervical spine. Such cases are sometimes spoken of as cases of "false wry-neck." In a third class of cases the deviation of the head is due to muscular rheumatism (acute myositis), and the tender muscles are the most important cause of the unnatural fixation of the head. In other cases still wry-neck is produced by hysterical spasm. None of these cases will be considered here except in so far as they are liable to be confounded with true wry-neck.

Spasmodic or True Wry-Neck depends on active spasm, usually of the muscles supplied by the spinal accessory nerve. The cause of this spasm appears to be a peculiar instability in the nerve-cells (cortical or spinal) that control the muscles involved. In no case has an organic lesion been detected as the cause of the condition.

The distribution and character of the spasm vary in different cases. Several muscles are usually affected, most often the sterno-mastoid and trapezius (upper part) of the same side. Not rarely the spasm involves also the splenius, and occasionally the scaleni and platysma myoides are included. Sometimes the spasm, which has a tendency to extend, includes other muscles than those of the neck, as the muscles of the face, jaw, or arm. Occasionally the spasm is limited to one muscle of the neck, which is almost invariably the sterno-mastoid, and in rare cases all the neck muscles participate in the spasm. Generally the spasm involves chiefly or exclusively the muscles of

one side; occasionally the muscles of both sides are about equally affected.

Owing to such wide differences as these in the distribution of the spasm, and to similar differences in its degree, the resulting change in the position of the head varies much in different cases. The chief positions observed are as follows: (*a*) rotation of face to side opposite spasm and slight inclination of head to side of spasm,—dependent on spasm of one sterno-mastoid; (*b*) rotation of face to side opposite spasm and great inclination of head to side of spasm—dependent on spasm of sterno-mastoid and trapezius of one side; (*c*) extreme rotation of head to one side, dependent on spasm of sterno-mastoid of one side and of the splenius of the other—*i. e.*, the side to which the face turns; (*d*) retroflexion of head ("retrocollic" spasm), due usually to bilateral spasm of trapezii, splenii, and sterno-mastoid. Sometimes the spasm of torticollis is tonic, at others clonic, and sometimes clonic and tonic spasms occur together. The spasm varies in degree and character from time to time, and at times there may be short or long intermissions, or the spasm may be continuous. The onset of the spasm is usually gradual, rarely acute, and is often attended or preceded by pain. The muscles involved never waste, but may hypertrophy in time. Spasmodic torticollis is usually a disease of long duration and in severe cases persists during life with little change.

The diagnosis of spasmodic wry-neck is rarely difficult. Sometimes it is confounded with the false wry-neck already mentioned, when, as not rarely happens, the latter is among the earliest evidences of caries of the cervical spine. There are several points of distinction between wry-neck of functional origin and that which depends on vertebral disease, and error may always be avoided by

bearing them in mind. In spasmodic wry-neck the face is turned to the side *opposite* the contracting sterno-mastoid muscle. In false wry-neck the sterno-mastoid is rigid on the side to which the face is turned. The former condition is painless, except at the onset; in the latter there is often pain, neuralgic in character, in the distribution of the sensory branches of the first and second cervical nerves over the occiput. Moreover, in the latter condition this pain is greatly aggravated or induced by sudden pressure on the top of the head.

Hysterical wry-neck differs from the true form in that the spasm rarely remains limited to the muscles of the neck, in its dependence on emotion, in its rapid onset, and in its occurrence in women under thirty years of age, before which time true wry-neck is very uncommon in females.

Paralysis of the Hypoglossal Nerve.—The chief effect of disease of one hypoglossal nerve is loss of power in the tongue. When the tongue is at rest in the mouth, its base is a little higher on the paralyzed than on the unparalyzed side. When the tongue is moved *within* the mouth, there is imperfect power of motion *to* the paralyzed side; but when the tongue is *protruded*, the tongue *deviates to the paralyzed side*, because it is pushed to that side by the fibres of the genio-hyo-glossus on the normal side In disease of both hypoglossal nerves the tongue cannot be moved in the mouth and cannot be protruded. In such cases there is very defective articulation.

Paralysis of the tongue may result from disease anywhere in the motor path to the tongue, disease between the cortex and the hypoglossal nucleus, disease of the nucleus, or disease of the nerve, in or out of the medulla. The associations of the paralysis give the data for the localization of the lesion which causes loss of power in

the tongue. When the disease is in the upper segment of the motor tract to the tongue,—*i.e.*, is supra-nuclear, the tongue paralysis is associated with hemiplegic weakness on the side of the paralysis. Whether this disease is situated in the hemisphere (including disease of lowest part of the third frontal convolution), in the crus, or in the pons, must be determined by other indications (see Localization, also Hemiplegia). When disease involves the nucleus, the paralysis is bilateral, owing to the closeness of the nuclei to one another (see p. 59). There is also atrophy of the tongue in such cases, and generally paralysis of the lips. Nuclear disease is almost always degenerative (bulbar paralysis, rarely locomotor ataxia) in character, but may result from vascular lesions (hemorrhage, softening). If the hypoglossal fibres are involved within the medulla, there is usually, but not necessarily, paralysis of the limbs on the opposite side (crossed paralysis), owing to the contiguity of the nerve and the motor path. Both hypoglossals may be involved by a bilateral lesion. Vascular lesions or tumors may cause paralysis from disease of the fibres within the medulla. Disease of the hypoglossal at the side of the medulla is one-sided, and is accompanied by damage to the roots of the spinal accessory, which causes paralysis of the larynx and palate on the same side. Chronic syphilitic meningitis and new growths are the chief causes of such paralysis. The tongue wastes to some extent in disease of any part of the lower segment of the path to the tongue-nucleus, nerve, or path in the medulla between nucleus and nerve.

Spasm of the tongue, causing its deviation to one side on protrusion is occasionally met with as a symptom of hysteria. It then is usually associated with other more distinctive signs of this disease, especially convulsion, but may occur apart from any other spasmodic symptoms.

Paralysis of the Phrenic Nerve.[1]—The consequence of paralysis of the functions of the phrenic nerve is inaction of the diaphragm. If the paralysis is unilateral the diaphragm does not descend on that side during inspiration, but the loss of movement is not considerable, as the other side of the diaphragm continues to act. When both phrenics are paralyzed the inaction of the diaphragm produces a decided effect upon respiration. The movement of the thorax is increased, but if the hand is placed on the abdomen below the ribs, the advance of the abdominal wall and the descent of the viscera which occurs during normal inspiration can no longer be felt.

The phrenic nerve may be paralyzed from disease or injury of the spinal cord (most common cause), from damage to the roots of the nerve, and from disease or injury to the nerve trunk, either in the neck or thorax. When the paralysis depends on cord disease, it is bilateral, and other paralyses are associated with that of the diaphragm. Compression and crush of the cord are the most frequent causes of such paralysis. When the nerve trunk is the seat of the lesion only the diaphragm suffers and the paralysis is generally unilateral. The nerve may suffer in the neck from deep wounds, or in the thorax from tumors or aneurisms. Sometimes it is the seat of neuritis from cold or from diphtheria, and the paralysis may then be bilateral.

In some cases of hysteria the diaphragm may be little used for a considerable period of time, the upper part of the thorax acting as when the diaphragm is paralyzed. Knowledge of this fact and the detection of activity of the diaphragm will avoid an error, but it may be necessary to observe for some time before forming an opinion.

[1] The phrenic nerve is derived from the third, fourth, and fifth cervical nerves.

The movements of the diaphragm are arrested or diminished in some cases of diaphragmatic pleurisy, but the pain which accompanies this condition will serve to prevent an error in diagnosis.

Paralysis of the Posterior Thoracic Nerve.[1]—Damage to the posterior thoracic nerve results in paralysis of the serratus magnus muscle. The chief effects of this paralysis are: (1) rotation of the scapula on its vertical axis when the arm is put forward, with recession of the edge of the scapula from the thorax—so-called "winged scapula" (this is characteristic of serratus paralysis); (2) the lower angle of the scapula is rotated inward and upward when the arm is carried forward; (3) the power of elevating the arm above the level of the shoulder is greatly weakened. There is usually severe pain in the neck and shoulder during the development of the paralysis.

The evidences of isolated serratus paralysis are so characteristic that an error in diagnosis is scarcely possible. The nerve is usually damaged in the neck, either by direct pressure of heavy angular objects on the shoulder or by violent muscular effort as in lifting a heavy hammer. Sometimes it is injured by a wound or contusion. In rare cases the paralysis follows exposure to cold. Isolated serratus paralysis is usually one-sided; rarely it is bilateral. It is much more common in men than in women, and is generally on the right side, as seen in Fig. 57? Sometimes the posterior thoracic nerve is paralyzed from disease of its cells of origin in the cord, as in progressive muscular atrophy and infantile paralysis, or crush of the cord. Other muscles than the serratus are then paralyzed and the nature of the affection is plain.

[1] This nerve is derived from the fifth and sixth cervical nerves.

Paralysis of the Supra-Scapular Nerve.[1]—Damage to the supra-scapular nerve causes paralysis of the supra-

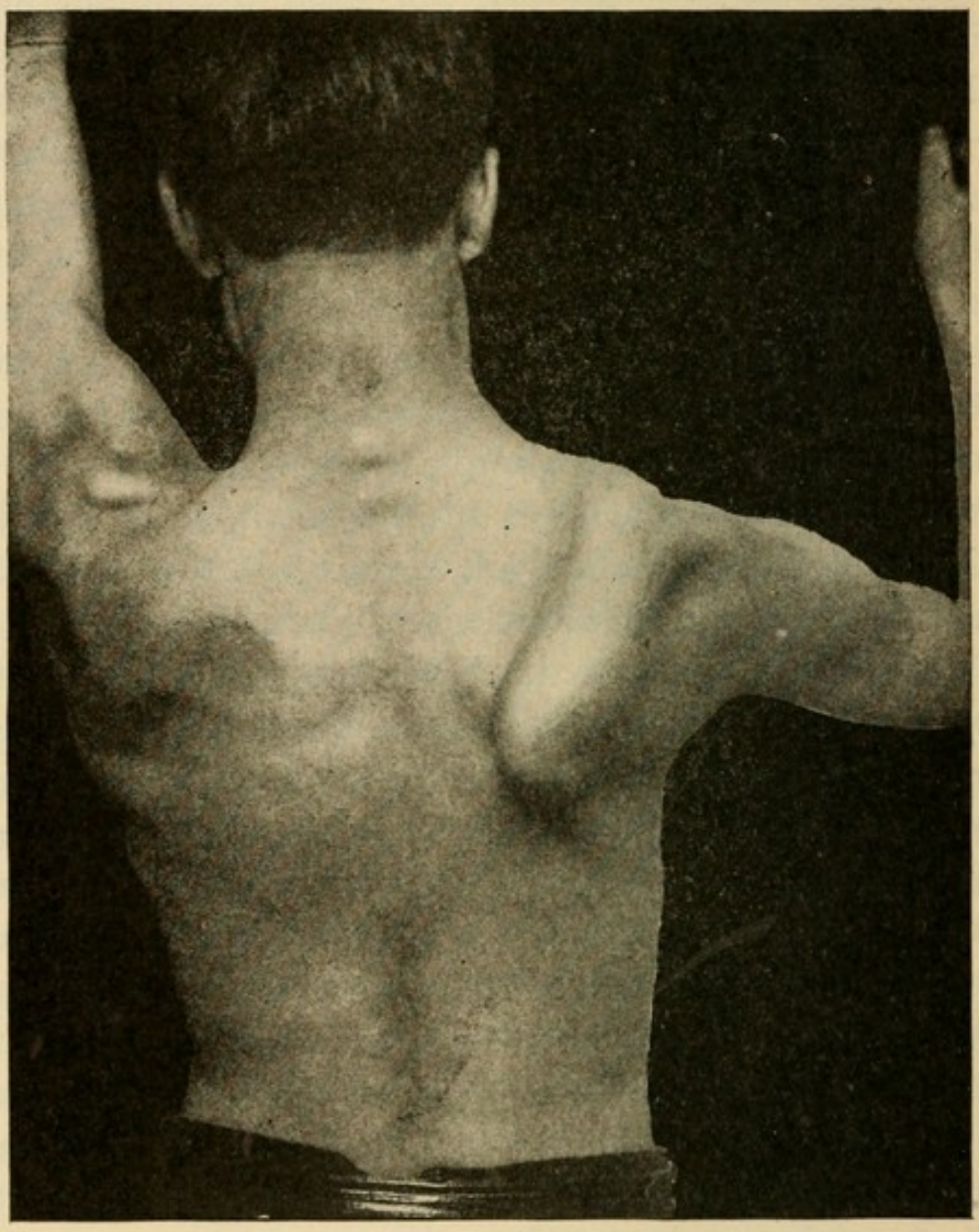


FIG. 57.

Position of shoulder-blades in paralysis of the right serratus magnus during abduction of the arms.

spinatus and infra-spinatus muscles. The paralysis of the former gives rise to no obtrusive symptoms, but

[1] This nerve is derived from the fourth and fifth cervical nerves.

paralysis of the infra-spinatus causes a loss of outward rotation of the humerus. An important effect of this loss is the inability to carry the hand from left to right as in writing.

There is seldom isolated paralysis of the supra-scapular nerve, but it is often affected together with the circumflex in consequence of dislocation of the head of the humerus, and in obstetrical paralysis.

Paralysis of the Circumflex Nerve[1] causes loss of power in the deltoid and teres minor muscles. The paralysis of the former is by far the more important. Its chief sign is inability to raise the arm. In some cases there is loss of sensation on the outside of the upper part of the arm over the muscles. The deltoid wastes, and this alters the contour of the shoulder. After a time trophic changes occur in the shoulder joint (the circumflex sends filaments to the joint), and adhesions may form. Paralysis of the circumflex nerve is easily recognized. It is impossible to confound it with the loss of motion that is seen in anchylosis of the shoulder joint, if it is remembered that in the latter state passive motion of the arm moves the scapula as well as the arm. The circumflex nerve is often injured by falls on the shoulder and by dislocations of the head of the humerus. Rarely it is the seat of "spontaneous" neuritis. It is sometimes paralyzed with other nerves belonging to the brachial plexus (see Brachial Plexus, Diseases of) in a highly characteristic manner.

Paralysis of the Musculo-Cutaneous Nerve (External Cutaneous, Perforans Casserii) causes loss of power in the biceps and brachialis anticus muscles, the effects of which are unmistakable (loss of flexion of elbow—especially marked when the forearm is supinated and the supi-

[1] This nerve is derived from the fifth, sixth, seventh, and eighth cervical nerves.

nator longus cannot act as a flexor). There may or may not be anæsthesia on the outer half of the forearm in front and behind, and over the arm in its lower part and outer side. The musculo-cutaneous is rarely paralyzed by itself.

Paralysis of the Musculo-Spiral Nerve[1] in the vicinity of the brachial plexus causes loss of power in all the extensors of the forearm and wrist and the supinators. Extension of the elbow is impossible, the wrist drops,

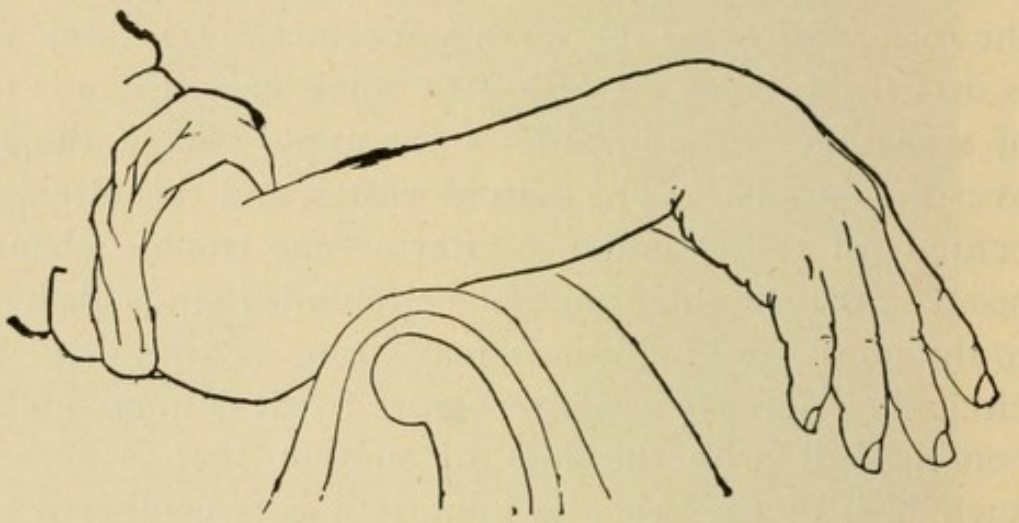


FIG. 58.

Paralysis of the musculo-spiral nerve; maximum extension of wrist and fingers. The extension of the fingers progressively diminishes from the first to the fourth. From a photograph. (Gowers.)

and the fingers are flexed at their distal joints. The fingers can, however, be extended by the interossei and lumbricales if the first phalanges are flexed. Supination, though not entirely lost (the biceps being active), is greatly weakened. After a time the excessive flexion of the carpus leads to undue prominence of the carpal bones and the synovial sacs at the back of the wrist. When the damage to the nerve is serious there is in a few weeks a perceptible diminution in the size of the fore-

[1] This nerve comes from the fifth, sixth, seventh, and eighth cervical nerves.

arm, due to the atrophy of the paralyzed extensors, and the muscles present the RD in various degrees.

If the damage to the nerve is in the middle of the arm the biceps is generally involved. The supinator longus escapes only in rare cases of musculo-spiral paralysis.

The loss of sensation in the parts supplied by the musculo-spiral varies considerably in different cases. In actual division of the nerve above its cutaneous branches there is usually loss of sensation in the outer part of the arm (about one quarter its circumference) from the level of insertion of the deltoid to the external condyle of the humerus, and on the back of the forearm on the outer side above, fading into normal sensation in the lower third of the forearm. The skin on the dorsal surface of the hand is anæsthetic over the thumb and metacarpal bones of the thumb, index, and middle fingers. In many cases of musculo-spiral paralysis there is no anæsthesia either in the hand or arm.

Musculo-spiral paralysis is of frequent occurrence, the course of the nerve exposing it to various kinds of injury. High up the nerve may be damaged by the pressure of a crutch. Indeed, crutch-paralysis is usually due to musculo-spiral injury. The nerve is apt to be torn in cases of fracture of the humerus, and may be pressed upon by callus. The most common cause of the paralysis, however, is pressure on the nerve during sleep. The patient lies on a hard bed or on the floor with the arm under him and receiving his weight. The nerve suffers as it passes around the humerus about the middle of the arm. So often does this occur in patients who have fallen asleep after excess in alcohol that it is known as "Saturday-night paralysis," or "Sunday-morning paralysis," from the times at which the paralytic effects are usually detected.

It is necessary to distinguish musculo-spiral paralysis from some forms of multiple neuritis—notably from lead paralysis and alcoholic neuritis. In lead paralysis the muscles supplied by the musculo-spiral nerve are involved (*vide* Lead Paralysis), but the affection is almost invariably bilateral (though the two sides may suffer very unequally), and the supinator longus muscle is almost always exempted from the palsy. In musculo-spiral paralysis, on the contrary, the paralysis involves only one nerve (in rare cases a cause of musculo-spiral paralysis operates bilaterally), and the supinator longus is almost invariably included in the palsy. Moreover, the onset of lead palsy is gradual and the development of musculo-spiral paralysis is rapid or sudden. The cause of musculo-spiral paralysis is usually readily elicited. The distinction from alcoholic neuritis is usually extremely easy (see Alcoholic Neuritis). The bilateral character of the paralysis, its extensive distribution, and the alcoholic history will prevent error even when the paralysis affects chiefly the upper extremities.

Paralysis of the Median Nerve.[1]—Severe damage to the median nerve above its muscular branches causes loss of power in the flexors of the fingers (excepting the ulnar half of the flexor profundus), in the pronators, in the flexor carpi radialis, in the two outer lumbricales, and in all the muscles of the ball of the thumb except the abductor pollicis and the ulnar half of the flexor brevis pollicis. In consequence of this loss of power the ability to flex and pronate the forearm is greatly diminished but not abolished. Flexion at the wrist to the ulnar side is still possible by the action of the flexor carpi ulnaris ; pronation is feebly performed by permitting the weight of the hand to rotate the forearm after it has been supi-

[1] This nerve arises from all the roots of the brachial plexus.

nated, the supinator longus being capable only of pronating the arm to a position midway between supination and pronation. The thumb is extended and abducted in a characteristic manner, and cannot be brought in contact with the tips of the fingers. The second phalanges can no longer be flexed on the first, and in the first and second

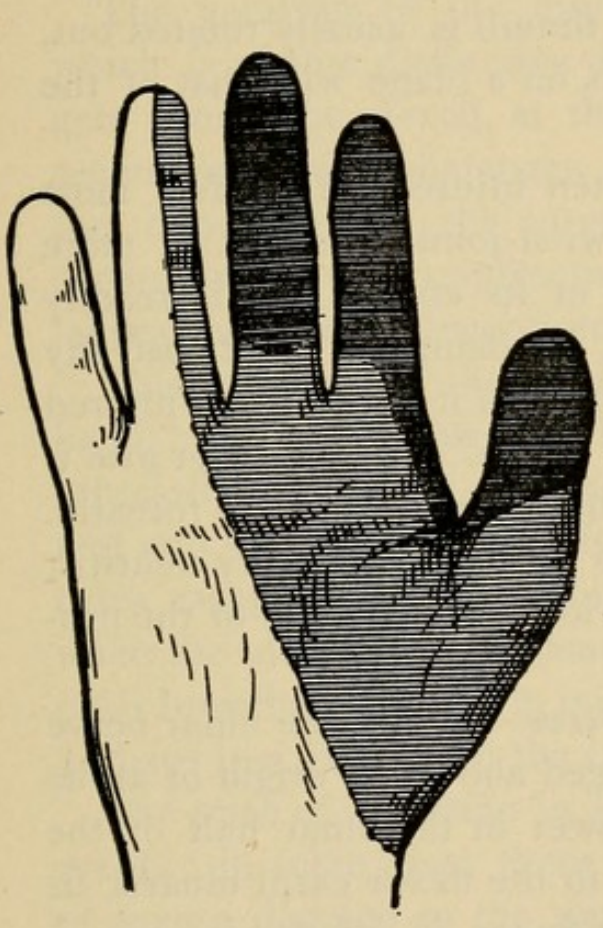


FIG. 55.

Diagram illustrating the area of anæsthesia in a case of injury to the median nerve. (Palmar surface.)

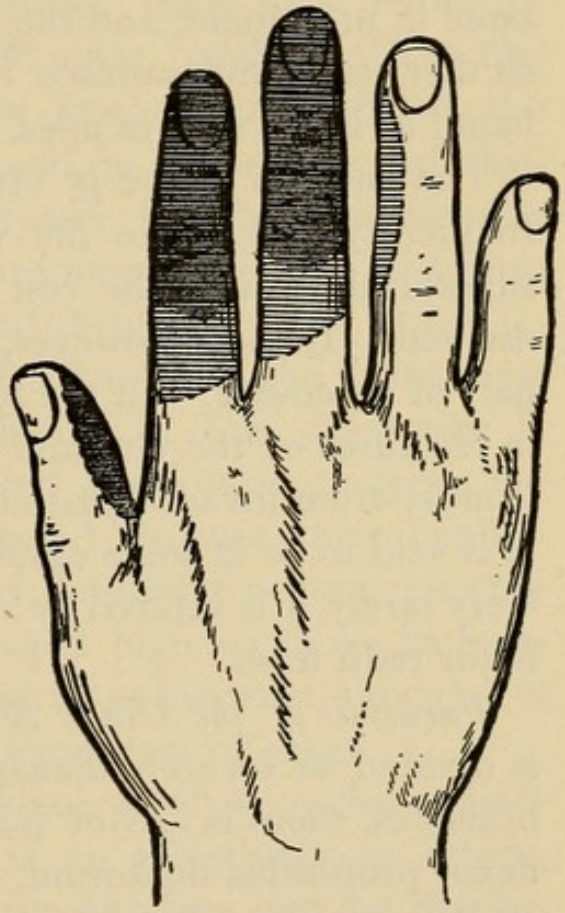


FIG. 56.

Diagram illustrating the area of anæsthesia in a case of injury to the median nerve. (Dorsal surface.)

fingers there is also loss of flexion of the third phalanges. The first phalanges are flexed by the interossei. The characteristic distribution of anæsthesia which is observed in cases of injury to the median nerve is sufficiently well shown in the accompanying diagrams.

The degree and extent of the anæsthesia vary much in

different cases. Sometimes there is no affection of sensation whatever. In a case of severe damage to the median the appearance of the hand and forearm soon becomes highly characteristic. The forearm is much atrophied on the radial side in front, the wrist is inclined to the ulnar side and perhaps hyper-extended, the ball of the thumb is greatly wasted, the head of the metacarpal bone is prominent, and the thumb is usually rotated out, so that its palmar surface is on a plane with that of the hand, as is the case in apes.

The median nerve is often injured. It suffers most frequently just above the wrist-joint, where it is more superficial than in the rest of its course, and is readily divided. It may, however, be damaged in almost any part of its course. In the forearm it is not rarely injured in fractures of the ulna and radius. In the upper arm it is most often invaded just above the bend of the forearm. It is said to be in some cases the seat of primary neuritis. Very rarely it is injured by violent contraction of the pronator radii teres.

Paralysis of the Ulnar Nerve.—When the ulnar nerve is divided or severely damaged above the origin of all its branches, there is loss of power in the ulnar half of the flexor profundus digitorum, in the flexor carpi ulnaris, in all the muscles of the little finger, in all the interossei, in the two ulnar lumbricales, in the abductor pollicis and in the inner head of the flexor brevis pollicis. When this paralysis has lasted some time (three or four weeks or longer), the action of the unparalyzed opposing muscles brings the hand into a very characteristic position. The wrist is slightly bent backwards and to the radial side of the forearm, by the action of the extensor carpi radialis, extensor carpi ulnaris and flexor carpi radialis. The hand is considerably thinner than normal

owing to the wasting of the interossei and the muscles of the little finger, which leave the metacarpal bone of that finger very prominent. There are depressions between the metacarpal bones, but there is a particularly marked depression on the radial side of the metacarpal bone of the index finger on the back of the hand, owing to the wasting of the first dorsal interosseus.

The paralysis of the interossei leads to a deformity which is almost distinctive of ulnar paralysis. The fingers cannot be flexed at the first, or extended at the second and third phalanges, and, in consequence of this, the opponents of the interossei (extensor communis digitorum) by their contracture over-extend the first phalanges and flex the second and third.

This deformity of the hand is known as the "bird-claw hand," or the "claw-like hand."[1] The deformity is especially marked in the third and fourth fingers; the first and second are less affected because their lumbricales escape paralysis. This deformity occurs not only when the ulnar nerve is damaged high up above its muscular branches, but also in injuries at the wrist, though it is perhaps less extreme in the latter class of cases.

The state of sensation in ulnar paralysis varies considerably; in some cases there is no anæsthesia, in others, of severe damage to the nerve, the loss may be that indicated in figures 57 and 58—a typical distribution of anæsthesia.

The ulnar nerve is probably more often damaged than any other spinal nerve. It is frequently injured in wounds of the forearm, especially in wounds at the wrist, where the nerve is superficial. When the nerve is in-

[1] It is seen also in progressive muscular atrophy, where disease affects the cells of origin of the ulnar nerve before those of the musculo-spiral.

jured at the wrist it is generally *above* the origin of the dorsal cutaneous branch. Higher up in the forearm the nerve may be hurt by fractures of the ulna and radius. At the back of the elbow, just external to the olecranon, the nerve is very liable to suffer from wounds, and is occasionally injured by pressure or contusion. Long-continued

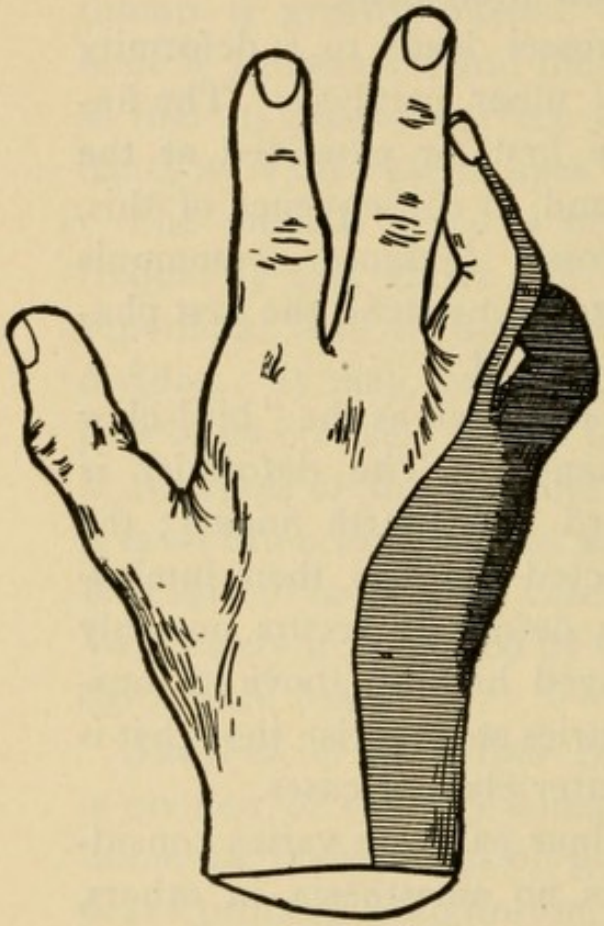


FIG. 57.

Diagram illustrating area of anæsthesia and position of fingers in a case of injury to the ulnar nerve. (Dorsal surface.) After Bowlby.

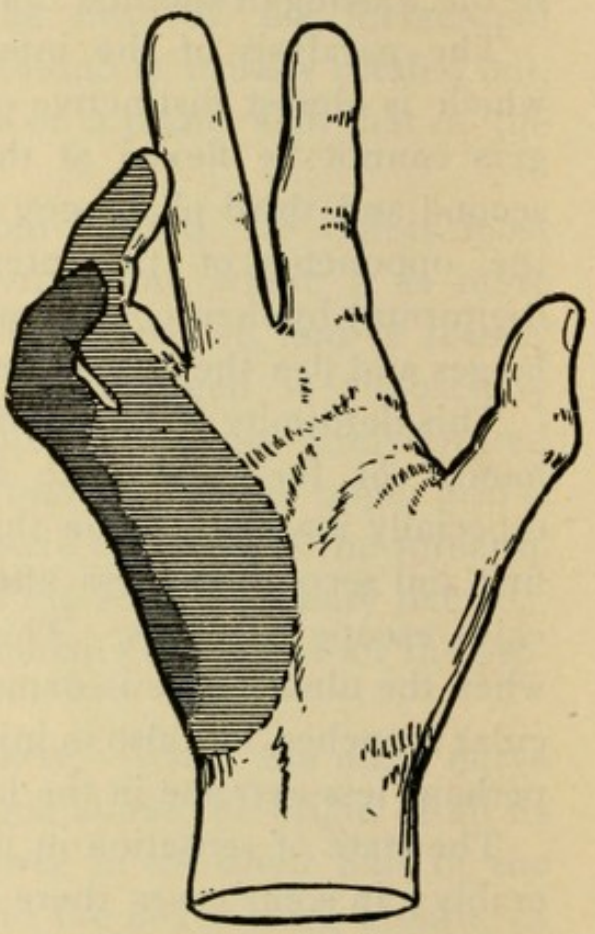


FIG. 58.

Diagram illustrating area of anæsthesia and position of fingers in a case of injury of the ulnar nerve. (Palmar surface.)

flexion at the elbow sometimes suffices to cause ulnar paralysis. Sometimes paralysis arises in this way during sleep. The nerve is very rarely injured in the arm above the elbow. Sometimes the symptoms of an apparently spontaneous ulnar neuritis are observed in persons in reduced health.

The diagnosis of ulnar paralysis is simple. Error may possibly arise in rare cases of disease of the cervical enlargement, in which the ulnar nerve distribution is chiefly affected. Other evidences of spinal cord disease are never wanting in these cases.

Paralysis in the Distribution of Two or More Nerves of the Arm, and due to disease outside the spinal canal, is not very uncommon.

Thus, the median and ulnar nerves not rarely suffer together in fractures of the ulna and radius, and in wounds at the wrist; the musculo-spiral and ulnar, sometimes the musculo-spiral, ulnar and median, in fractures of the humerus; and any or all of the nerves of the brachial plexus (usually including the circumflex) may be compressed or lacerated by dislocation of the humerus (especially sub-coracoid dislocation). Inflammation may ascend one nerve and, reaching the brachial plexus, spread to others ("ascending neuritis," "neuritis migrans"). Thus the ulnar may be implicated by an ascending median neuritis; and conversely, the median may be involved by an ascending ulnar neuritis. The original wounds are usually infected in such cases.

In cases where nerves of the arm, and especially of the forearm, have been injured, and tendons and muscles have been injured with the nerves, it may be difficult to distinguish the effects of the nerve injury and the effects of injury to the tendons and muscles. This is because there is often considerable cicatricial change in muscles and tendons, leading to deformity, which may simulate closely that of nerve injury, and because certain of the most important signs of nerve injury, namely, changes in the irritability of muscles and nerves, may be obscured by the contraction that sets in. Even the most careful attention to every detail in the history and examination

of such cases may not enable the observer to arrive at a correct conclusion as to the extent and situation of the nerve damage.

Non-Traumatic or Primary Brachial Neuritis is an uncommon condition in which the cords of the brachial plexus become the seat of an unevenly distributed inflammatory process. The symptoms vary much in different

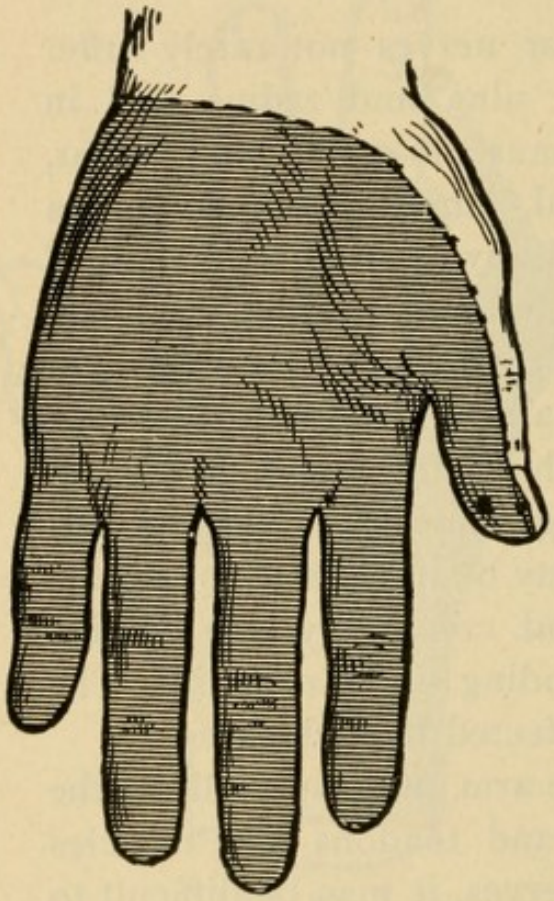


FIG. 59.

Diagram illustrating the area of anæsthesia in a case of injury to the ulnar and median nerves. (Palmar surface.)

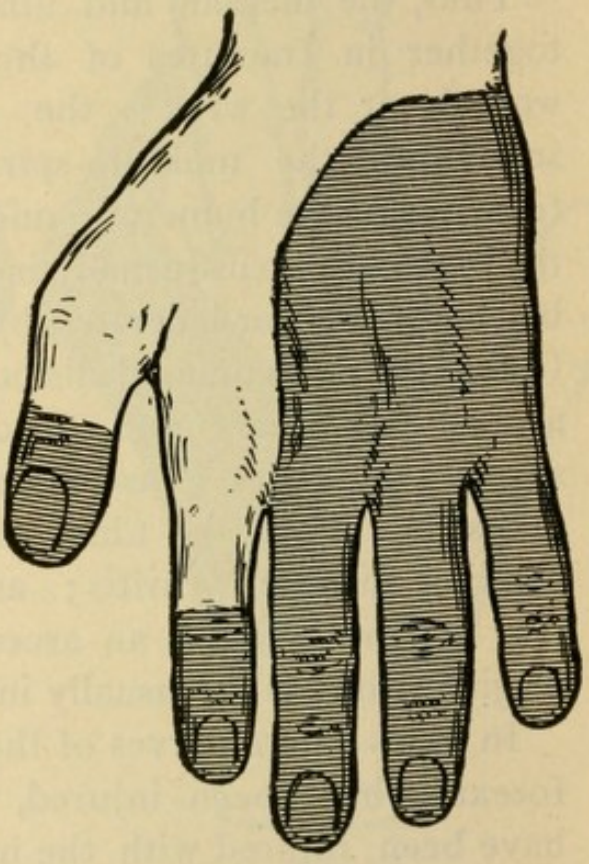


FIG. 60.

Diagram illustrating the area of anæsthesia in a case of injury to the ulnar and median nerves. (Dorsal surface.)

cases, according to the distribution of the neuritis and its intensity. The earliest symptoms are sensory in character, and include pain, paræsthesia and anæsthesia of irregular distribution. The pain is usually referred to the shoulder; sometimes also to the hand and fingers. These sensory symptoms may be followed by gradual

loss of power in certain muscles and muscular atrophy. The muscles suffer unequally and present various grades of RD, but rarely the complete form. Vaso-motor and trophic symptoms may occur but are only rarely very decided. This form of brachial neuritis appears to depend, in some cases, on exposure to cold or over-exertion. Its diagnosis is simple if its occurrence is borne in mind.

Erb's Paralysis (obstetrical palsy).—These names are given to a peculiar form of paralysis dependent on damage principally to the fifth and sixth cervical nerves at one point in the neck, just in front of the edge of the trapezius. The paralysis involves regularly the deltoid, biceps, brachialis anticus and supinator brevis, and supra- and infra-spinati. There is often anæsthesia on the outer side of the arm in the distribution of the circumflex and external cutaneous nerve; there is usually considerable muscular atrophy. The characters of Erb's paralysis differ considerably in adults and young children. In adults the paralysis is often complete, sensory symptoms are marked, and the affection is frequently of long duration. The cause of the paralysis in adults is usually a trauma, but sometimes the condition depends on the presence of a cervical tumor, and occasionally the paralysis develops without known cause.

Erb's paralysis is probably much more often seen in infants than in adults, but even in them it is an uncommon form of paralysis. It always depends, in infants, on undue stretching of the nerve trunks, during delivery, thus causing stretching and rupture of the nerve sheaths and trunk. Hence it is commonly termed "obstetrical paralysis."[1] The severity of the damage to the nerves varies

[1] This term is sometimes applied to the facial paralysis that arises from the pressure of forceps, and to some forms of infantile cerebral palsy. A better name is "brachial birth palsy."

much in different cases. In the slighter cases of injury the paralysis may be unnoticed for some time after the birth of the child. In a well developed case the paralysis can hardly escape detection, and is so peculiar in distribution as to assert its nature. The arm hangs by the side with the forearm in extreme pronation, or some power of elbow flexion may remain. Atrophy of the paralyzed muscles cannot be distinguished until several months after the damage, and may appear very slight even then, as it is masked by the large amount of fat over the muscles. Sensation is rarely impaired, and when it is the impairment may be detected with difficulty. The paralysis in mild cases usually wears away in the course of a few months, sometimes in a few weeks, but in all severe cases some paralysis, atrophy, and deformity usually remain. The peculiar distribution of the paralysis and detection of the lesion of connective tissue overgrowth at Erb's point by means of palpation, and its occurrence during delivery, if this can be established, make the diagnosis clear. Some care must be taken not to confound this condition with some forms of poliomyelitis (*vide* p. 452), or with injuries to the shoulder-joint or muscles. The characteristic electrical reactions in the paralyzed muscles serve to distinguish Erb's paralysis from all conditions outside the nervous system.

The nerves of the lower extremity are, on the whole, less frequently the seat of injury and disease than those of the upper extremity.

Paralysis of the *Obturator Nerve*[1] gives rise to loss of power in the adductors of the thigh, and to defective outward rotation of the thigh. The defective power of adduction renders the patient unable to cross the leg of the paralyzed side over the other leg. The obturator nerve is seldom paralyzed except from damage to the

[1] Derived from the third and fourth lumbar nerves.

lumbar plexus. Occasionally, however, the nerve itself is damaged by pressure against the pelvis during delivery.

Paralysis of the Anterior Crural Nerve causes loss of power and atrophy in the extensors of the knee, and loss of knee-jerk from damage to the reflex arc. When the nerve is damaged within the pelvis the branch to the iliacus muscle is involved and there is impaired[1] power of flexing the hip, as well as loss of extension of the knee. Paralysis of the anterior crural nerve causes also anæsthesia, which involves the entire thigh, with the exception of a strip of variable width along the back of the thigh (supplied by the sacral nerves) and the inner side of the leg and foot. The anterior crural nerve may be damaged in the thigh or groin, may suffer from pressure during parturition, or from dislocation of the hip, or may be involved by disease affecting the lumbar plexus.

Paralysis of the Superior Gluteal Nerve[2] causes loss of abduction and circumduction of the thigh, from paralysis of the gluteus minimus and medius. As an isolated paralysis, apart from affections of the plexuses, it is a very rare condition.

Paralysis of the Sciatic Nerve causes symptoms which vary considerably with the seat of the lesion. Damage to the nerve is usually *below* the upper third of the thigh, and, if severe, gives rise to paralysis of all the muscles below the knee and anæsthesia of the sole and outer side of the foot and the outer side of the leg. The gait in such cases is much like that observed in many cases of poliomyelitis in children. If the lesion is *above* the middle third of the thigh the flexors of the knee and

[1] Flexion is merely *impaired* because the psoas is supplied by other lumbar nerves.

[2] This nerve is derived from the lumbo-sacral cord.

extensors of the hip are included in the paralysis. The sciatic nerve external to the pelvis may be damaged in wounds of the thigh, by disease of the femur, by adjacent tumors, and occasionally by dislocation of the hip. The nerve is often the seat of primary neuritis.

Paralysis of the External Popliteal (Peroneal) Nerve causes loss of power in the tibialis anticus, extensor longus digitorum, extensor brevis digitorum and peronei, in consequence of which there is loss of flexion of the ankle and of extension in the first phalanges of the toes. The patient in such cases has "drop-foot," and in the course of time talipes equinus develops. In cases of severe damage to the nerve there is anæsthesia on the outer half of the front of the leg, and on the greater part of the back of the foot. The external popliteal nerve is very superficial in its course, and passes over the fibula. It is consequently exposed to all kinds of injury—from wounds, from fracture of the fibula, from pressure, etc. It is also sometimes the seat of primary neuritis.

Paralysis of the Internal Popliteal Nerve causes loss of power in the posterior tibial group of muscles (including the tibialis posticus and popliteus) and the long flexors of the toes, and in the muscles of the sole of the foot. Extension of the ankle-joint is impossible, and if the branch to the popliteus is involved there is loss of inward rotation of the leg when it is flexed. When the damage to the nerve is sufficiently severe to cause anæsthesia there is loss of sensation over the outer part and posterior aspect of the lower part of the leg and on the sole of the foot. The posterior tibial nerve is rarely injured except in fractures of both bones of the leg.

The branches of the internal popliteal nerve, the external and internal plantar nerves, are rarely involved alone.

Paralysis of the External Plantar Nerve causes loss of

power in the muscles of the little toe, the flexor accessorius, the interossei, the two outer lumbricales, and the adductor of the big toe. Certain of these muscles (lumbricales and interossei, abductor and flexor minimi digiti) flex the first phalanges, and extend the second and third, an action of much importance in walking, in the propulsion of the body forward just before the foot leaves the ground. The loss of this action is a hindrance in walking, as is the later contracture of the opponents of the interossei, which leads to flexion of the second and third phalanges. The sensory loss in cases of external plantar paralysis includes the skin of the outer half of the sole of the foot and of the little toe, and that of the adjacent half of the fourth toe.

Paralysis of the Internal Plantar Nerve causes loss of power in the short flexor of the toes, the intrinsic muscles of the big toe (with the exception of the adductor), and of the inner lumbricales. It gives rise also to anæsthesia on the inner part of the sole of the foot and the plantar surface of the three inner toes and the adjacent half of the fourth toe.

Paralysis of the Small Sciatic Nerve causes paralysis of the gluteus maximus, with consequent interference with the power of rising from a seat and loss of sensibility in an area of variable size on the posterior surface of the thigh.

The small sciatic nerve is damaged only in disease of the sacral plexus, and is seldom the only nerve involved.

The *Lumbar Plexus* itself may be involved by abdominal tumors, enlarged nodes, or psoas abscess ; and caries of the lumbar spine may cause compression of the nerve-roots that make up the plexus. The plexus is usually irregularly invaded, some nerves suffering more than others. Sensory symptoms of an irritative character are often among the first symptoms of disease of the plexus. There is frequently severe pain in the course of the

genito-crural, ilio-inguinal, and ilio-hypogastric nerves. Later there may be anæsthesia of irregular distribution and loss of power in the distribution of the obturator and anterior crural nerves.

The *Sacral Plexus* is liable to suffer from various forms of pelvic disease, especially pelvic tumors and pelvic inflammation. Occasionally it is compressed during delivery. Sometimes, also, it is the seat of primary neuritis. In other cases it is the seat of neuritis consequent upon extension of inflammation from the sciatic nerve. The symptoms of disease of the sacral plexus are usually at first irritative in character (pain, paræsthesia, etc.); later they are indicative of a destructive process (anæsthesia, paralysis, atrophy, etc.).

The precise distribution of these symptoms varies in different cases, and the various muscular and sensory distributions of the different nerves arising from the plexus may be variously and unequally affected. The manifestations of disease of the plexuses are rarely confined to the distribution of any single nerve arising from them, although a single nerve (as the anterior crural) may for a time be the chief or exclusive seat of the symptoms. Plexus disease is almost invariably *unilateral*, but in rare cases of pressure paralysis and spontaneous neuritis it is *bilateral*. The distinction of lesions of the lumbar and sacral plexuses from other forms of disease, especially cauda-equina lesions and certain forms of non-symptomatic neuralgia, is considered elsewhere. In all cases where plexus disease is suspected a careful rectal exploration is of the utmost importance and may reveal the presence of a pelvic tumor, an aneurism, or an abscess which exerts pressure upon the lumbar or sacral plexus.

Tumors of Peripheral Nerves.—The chief forms of tumors of peripheral nerves have been already

mentioned (see 278). The diagnosis of a neuroma can be made with certainty only when the tumor can be felt. The presence of such a tumor may, however, be suspected in cases where pain, paræsthesia, anæsthesia, and loss of power come on slowly and grow progressively worse, and are limited to the distribution of one nerve-trunk. But there is no way of distinguishing, purely by the symptoms, the effects of damage by a neuroma from the effects of pressure from an adjacent nerve, or even from the effects of neuritis. When the neuroma can be felt there arises the question whether it is a "false" or a "true" neuroma. If there are multiple tumors they are probably of the "true" form. False neuromata are, however, sometimes multiple, but in such cases they are generally of rapid growth. If the tumor appears to grow from the side of the nerve it is probably a "false" neuroma.

The symptoms that characterize **Lesions of the Cauda Equina** in general have been elsewhere touched upon (p. 251).

There remain for consideration here certain clinical forms of cauda disease dependent on the nature of the lesion—namely, compression and crush. Little need be said here regarding crush of the cauda equina. It depends on injury from fracture and dislocation, usually of the upper lumbar vertebræ. The symptoms, which vary in severity in different cases, are of immediate onset, and the nature and position of the surgical affection renders the localization of the damage perfectly evident. The question which arises in some of these cases as to the implication of the conus medullaris has already been discussed. Compression of the cauda may be of rapid or slow development. When of rapid development it always depends on injury — usually an injury which

causes fracture-dislocation of lumbar vertebræ. In such cases there may be both crush and compression of the cauda, and it is impossible to distinguish between these conditions. It is probable that in rare cases the cauda is rapidly compressed by hemorrhage in the absence of fracture-dislocation. When the compression of the cauda is *slow*, the diagnosis of the condition causing it is more difficult than in surgical cases, as all external indications are wanting. The only exception to this is where the compression depends on the pressure of a meningocele in spina bifida, the nature of the case being here sufficiently obtrusive. Slow compression of the cauda usually depends on *tumor* within the spine. The symptoms differ considerably in different cases of tumor, and the number of cases that have been carefully studied is not yet sufficient to enable us to draw conclusions which fit all cases. The following may be regarded as the chief clinical characters of tumor of the cauda equina :

1. Severe pain in the region of the sacrum, persistent and increasing in intensity. Often the pain radiates down the back of the legs. The sacrum may be tender to pressure in the median line, and the spontaneous pain may be greatly increased during movement. This sacral pain is usually the first symptom of a cauda tumor. It may continue or pass away when anæsthesia is developed.

2. The gradual development of anæsthesia of the peculiar distribution already described (p. 253), loss of control over the bladder (often very early) and rectum, gradual development of paralysis and atrophy in the legs, especially below the knee, and gradual but early loss of knee-jerks, perhaps first on one side. Sometimes there are attacks of clonic spasm in the muscles of the legs.

3. The symptoms are always bilateral and are generally highly symmetrical.

The accurate localization of the lesion in the cauda depends on a knowledge of the representation of various sensory and motor structures in the cauda, but an accurate diagnosis is sometimes impossible. The diagnosis of the nature of the tumor is usually impossible. When it is made it is upon general principles. Syphiloma, sarcoma, and cavernous angioma have been met with. Most cases of tumor of the cauda equina are progressive (one to three years), but in rare cases, apparently belonging to this category, they have retrogressed, or remained for a long time stationary.

The points of distinction between tumor of the cauda equina and tumor of the spinal cord do not require special mention. They are sufficiently covered by the points of difference between cauda and cord lesions that have been touched on elsewhere (see Localization). There is, however, one disease of the cord which is liable to be confounded with a tumor of the cauda equina, namely, locomotor ataxia. Sharp radiating pains, paræsthesia, involvement of the bladder, loss of knee-jerks, and impaired gait are common to both. But in locomotor ataxia the pains are inconstant, there is distinct ataxia, and pupillary symptoms are usually present early. The absence of ataxia and pupillary symptoms, and the presence of marked atrophy and distinctive anæsthesia, are sufficient to prevent error if the possibility of confounding the conditions is remembered. There is usually no difficulty in distinguishing a tumor of the cauda from disease of the limb plexuses. In disease of the plexuses the symptoms are generally one-sided. Anæsthesia, if present, is of irregular distribution. Tumor of the cauda equina, however, generally causes bilateral symptoms and the peculiar areas of anæsthesia

so often mentioned. A rectal examination will sometimes clear up the diagnosis. Bilateral lumbo-sacral neuritis of primary origin may for some time cause symptoms that cannot be distinguished from a commencing cauda lesion. On the other hand, the symptoms of tumor may be for a time unilateral. In such cases it is necessary to wait for unequivocal symptoms to appear.

Multiple Neuritis (Polyneuritis—Disseminated Neuritis).—The term "multiple neuritis" is used to designate a large and important class of cases in which several nerves become inflamed either at the same time or in quick succession. Broadly speaking, these cases resemble one another in the occurrence of motor, sensory, and trophic disturbances, but they differ so much in the degree, distribution, and course of their symptoms that it would not be profitable to study them as a single group. All the plans yet proposed for classifying cases of multiple neuritis are imperfect. Perhaps the least objectionable classification is that based on etiology (toxic form, infectious form, spontaneous form, etc.). For the present purpose, however, it is impracticable to adhere to any one classification; it is preferable to study separately in their diagnostic relations the chief clinical types of multiple neuritis as they occur in practice. We may thus distinguish the following forms of neuritis: (1) alcoholic neuritis, (2) diphtheritic neuritis, (3) neuritis from lead poisoning, (4) neuritis from carbonic oxide poisoning, (5) Beriberi, and (6) leprous neuritis.[1] To these varieties of

[1] This list comprises the most important forms of multiple neuritis. Many other kinds of neuritis (dependent on different causes) might be described, but most of these are not sufficiently distinctive or common to require separate discussion. Some of them will, however, be touched on in connection with the diagnosis of the forms above enumerated.

neuritis may be added a condition whose pathology is as yet unsettled, but which probably depends on multiple neuritis—acute ascending paralysis.

Alcoholic Neuritis.—The diagnosis of alcoholic multiple neuritis is based chiefly on a combination of motor symptoms of paraplegic type with obtrusive and rather characteristic sensory symptoms, the onset of the process being subacute, or, as is less often the case, acute.

I. *Sensory Symptoms.* The sensory symptoms of alcoholic neuritis, though they usually develop hand in hand with motor paralysis, are often the earliest indication of the disease. They are chiefly irritative in character and consist at first of paræsthesias—as tingling, sensations of "pins and needles," rheumatoid pains, etc., in the extremities—usually the lower extremities, sometimes the arms. These symptoms are soon succeeded by the following manifestations :

(1) Pain, sharp or burning, rarely dull, in the nerve-trunks of the affected extremities and often in the areas supplied by these nerves. The pain may be very severe. Occasionally it is slight.

(2) Hyperæsthesia (hyperalgesia) of the extremities.

(3) Muscular tenderness. The muscles when pressed are very sensitive. This is a highly characteristic and common symptom of alcoholic neuritis.

(4) Anæsthesia, partial loss of muscular sense and partial loss of pain and temperature sense. The anæsthesia may be confined to irregular areas of skin, or even to the distribution of a single nerve (as the ulnar). Generally it implicates a considerable part of the distal portion of the extremity.

The sensory symptoms of multiple neuritis are symmetrically distributed, the degree of symmetry being often very striking.

II. *Motor Symptoms.*—Sensory symptoms are not long present before loss of power becomes associated with them. The paralysis usually commences in the legs but may begin in and be limited to the arms. Both legs are involved together or in rapid succession. The weakness is especially marked *below the knees* but usually extends to the thigh muscles. Often the paralysis extends to the upper extremities—first to the muscles of the hand, then to the forearm. The extensor groups of muscles are especially affected in both extremities, but often both flexors and extensors suffer in high degree. In the leg

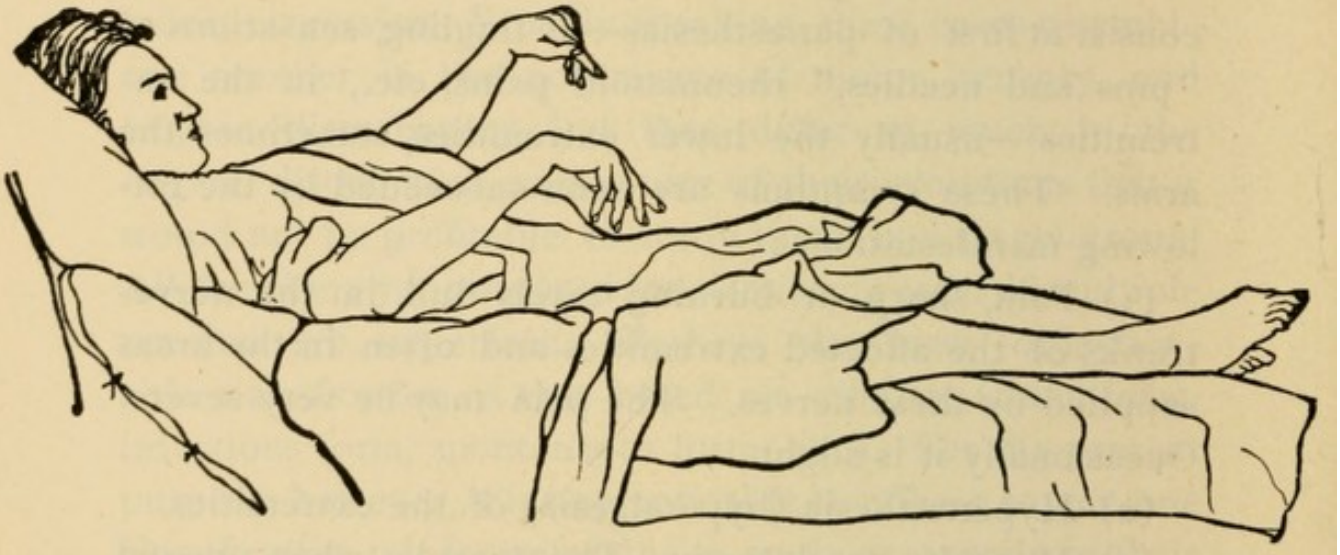


FIG. 65.

Multiple alcoholic neuritis; palsy of extensors of the wrist and flexors of ankle. (Gowers.)

there is "drop-foot," in the upper extremity "wrist-drop." The bilateral drop-foot is as suggestive of alcoholic neuritis as is wrist-drop of lead neuritis. The paralyzed muscles are flaccid, become much atrophied, and show the RD. In rare cases there is double facial paralysis. Paralysis of the sphincters is very rare, unless there is also myelitis.

Associated with these motor and sensory phenomena are often the following conditions:

(1) Inco-ordination, usually slight in degree and confined to the lower extremities. Occasionally the inco-ordination is the most obtrusive symptom of the disease, being greatly out of proportion to the sensory and paralytic phenomena. Such cases may closely resemble locomotor ataxia, and are hence termed "pseudo-tabes."

(2) Loss of myotatic irritability in the paralytic muscles. In the cases where the lower extremities are affected, the knee-jerk is lost early, even when there is only slight loss of power in the extensors of the knee. Cases occasionally occur, however, in which the knee-jerk is not lost at any time in the disease, probably because in these cases the extensors of the knee are not involved.

(3) Cerebral symptoms are seldom wanting in severe cases of multiple neuritis, and are especially constant in women. They comprise active delirium, with illusions and hallucinations, insomnia, loss of memory, fabrications, and general mental failure constituting a distinct psychosis, which may accompany polyneuritis (Korssakow's disease). These symptoms are not usually present until the disease is well established. They may, however, be present from the first, as when multiple neuritis develops during delirium tremens.

(4) Trophic symptoms in the skin and nails of the paralyzed extremities are common, and resemble those observed in simple neuritis.

When the onset of the disease is acute, there may be considerable elevation of temperature during the first weeks of the disease. The duration of the disease varies with the acuteness and severity of the process. In a moderately severe case of subacute development (*i. e.*, a typical case as regards course) the severity of the symptoms increases during a month or six weeks, and

improvement in power does not begin until a month or six weeks more have elapsed. Some chronic cases run a very long and tedious course (one to two years). Except in mild cases, contractures are apt to remain over.

The etiology of cases of alcoholic neuritis is an aid in the diagnosis of the condition, since a history of alcoholic excess is seldom wanting (*vide* p. 293). Such a history is, however, not often necessary for a diagnosis; the distribution of the palsy, the obtrusive and suggestive sensory symptoms and the escape of the sphincters suffice to indicate the nature of the case. But the diagnosis of the condition is not always so simple. There are three conditions with which the disease is readily confounded. These are poliomyelitis, transverse myelitis, and locomotor ataxia.

Paralysis, muscular atrophy, RD, and loss of tendon reflex occur both in alcoholic neuritis and in poliomyelitis. In the latter condition, however, sensory symptoms are absent (except, perhaps, during the onset of the symptoms), the paralysis is unsymmetrical, if not unilateral; there is some retrogression of the paralysis after the onset, especially if this be acute, and the development of the symptoms is fairly rapid if not acute. In alcoholic neuritis, on the other hand, sensory symptoms are obtrusive, the paralysis is usually symmetrical and always progressive, and the onset is usually (though not always) gradual.

The resemblance between some cases of alcoholic neuritis and locomotor ataxia is close. In both conditions there may be inco-ordination, loss of knee-jerks, sharp pains in the lower extremities, defective muscular sense, and great unsteadiness when the eyes are closed and the feet are close together (Romberg's symptom). The chief diagnostic points are the following: In alco-

holic neuritis there are no true lightning pains, the muscles are tender to pressure, there is distinct loss of power, atrophy and some degree of RD, bladder symptoms are absent, and the inco-ordination, if sufficiently marked to be confounded with that of locomotor ataxia, is distinguished by its rapid development in the early course of the disease.

An occasional source of difficulty in diagnosis arises from the circumstance that locomotor ataxia may be complicated with severe neuritis. Whenever unequivocal symptoms of locomotor ataxia are associated with paralysis and muscular atrophy of limited extent, or with limited patches of anæsthesia and analgesia, it is safe to conclude that those symptoms depend on neuritis.[1] But cases occur in which localized sensory and motor disturbances are associated with symptoms that strongly suggest locomotor ataxia, but are nevertheless equivocal. The question arises in such instances : have we to deal with a case of alcoholic multiple neuritis, or with a case of locomotor ataxia, associated with severe neuritis ? If this question cannot be decided by means of the indications that have been already mentioned, it may be necessary to wait and watch the course of the symptoms before reaching a conclusion.

There is seldom any reason for confounding alcoholic neuritis and transverse myelitis. In myelitis involving the lumbar enlargement there is paralysis of motion and sensation in the lower extremities and loss of knee-jerks. But the motor paralysis in such cases shows no tendency to remain confined to the distal portion of the extremities ; the sensory symptoms differ from those of alcoholic neuritis in that they are largely indicative

[1] The gastric and laryngeal crises of tabes probably depend on neuritis, but, of course, their occurrence gives rise to no question as to diagnosis.

of destruction of nerve elements rather than irritation; the sphincters are affected, and often there is a girdle-pain. In myelitis above the lumbar enlargement the knee-jerks are not lost, but are increased,[1] and this feature alone serves to distinguish it from a neuritis involving exclusively the lower extremities.

In rare instances alcoholic neuritis is complicated by myelitis, and the bearing of such an occurrence upon prognosis renders its recognition exceedingly important. The following facts are to be borne in mind in considering the possible existence of a complicating myelitis.

During the time when a case of alcoholic neuritis is increasing in severity we cannot say positively that the lesion will not extend to the cord, and the question of exact localization can hardly be said to have arisen. When, however, the symptoms become stationary, the question may come up. If there is no improvement in the symptoms after they have remained stationary for a period of four months, it is highly probable that the cord is involved, or that the patient is still taking alcohol. Again, if additional symptoms make their appearance after the neuritic process has been stationary for a time, such a progression probably depends on myelitis, provided the continued use of alcohol can be excluded. The symptoms which under these circumstances are particularly significant are an increase of paralysis, rapid muscular atrophy, the replacement of paræsthesia by anæsthesia, loss of control over the sphincters, and the appearance of bedsores.

The relation of alcoholic neuritis to the condition known as acute ascending paralysis is discussed in connection with the latter disease.

[1] In rare cases of acute myelitis above the lumbar enlargement the knee-jerks are temporarily lost.

Cases are not rarely met with in which there are numb ness, tingling, pain, and slight anæsthesia in irregular areas of the hands (acroparesthesia). These cases occur usually in women about the time of the climacteric, especially in those whose general health is for some reason depressed, and whose hands are much exposed to the action of water. The patients are very commonly persons who do not use alcohol to excess. It is only necessary to be aware of the occurrence of this affection, which depends probably on a palmar neuritis, to avoid confusing it with the early stage of alcoholic neuritis.

Lead-Neuritis.—The chief symptoms of neuritis from lead are localized muscular paralysis and atrophy and pains in the limbs. These conditions may be associated, or may occur separately. The most important and characteristic result of lead neuritis is the local paralysis. Generally the paralysis affects the extensor muscles of the wrist and fingers and causes bilateral wrist-drop, a condition highly suggestive of lead neuritis. Usually the paralysis remains confined to the upper extremities, but it sometimes involves the muscles of the legs, especially the long extensors of the toes and peronei. In the arm the deltoid not rarely suffers, and may suffer early. The biceps is also occasionally involved.

A peculiarity of considerable diagnostic importance relates to the distribution of the palsy in the forearm muscles. In a very large proportion of cases of lead palsy two muscles of the forearm are exempt from the loss of power ; these are the supinator longus and the extensor ossis metacarpi pollicis. In the majority of cases of lead palsy from neuritis, the loss of power precedes the wasting of the muscles, and the muscles present the RD. In other words, the character of the palsy closely resembles that observed when a motor nerve has been

involved in a traumatic lesion. But the type of lead paralysis is not always of this kind. Cases occur in which paralysis and atrophy of the muscles develop together, *pari passu*, and in which there is simply a gradual and proportional diminution of the irritability of the muscles to the faradic and galvanic currents. In these cases the paralysis may begin in almost any part of the extremities, but it is especially apt to begin in the small muscles of the hand. This form of local muscular atrophy may be likened to that which is so characteristic of progressive muscular atrophy.

It is important to recognize clearly the existence of these two types of paralysis from lead, for, though intermediate forms occur, the large majority of cases conform to one or other type. Moreover, it is probable that the two types of paralysis are referable to a difference in the seat of the lesion. In the type which resembles the paralysis resulting from an injury of a peripheral nerve, the lesion is almost always confined to the peripheral nerves ; in the type which resembles progressive muscular atrophy, there is often, and perhaps always, involvement of the ganglion-cells of the anterior horns of the spinal cord as well.

Sensory symptoms in lead paralysis are usually slight, and may be entirely absent. Sometimes there is considerable pain in the limbs affected at the time of the onset of the symptoms. Muscular tenderness may be found also. Sometimes there is slight anæsthesia of the skin over the affected muscles. In cases of lead poisoning without paralysis there are often dull or sharp limb pains, dependent on slight neuritis, but not distinctive in character. The reflexes are lost in lead neuritis under the same conditions that cause their loss in alcoholic neuritis.

The diagnosis of lead neuritis is usually easy. From

alcoholic neuritis it is distinguished by the localization of the paralysis to the upper extremities, by the absence or trifling character of sensory symptoms, and by the presence of other indications of lead poisoning, such as colic, the lead line on the gums, and anæmia. Cases of alcoholic neuritis occur, however, in which the paralysis is limited to the upper extremity, and in which the sensory symptoms are slight. In such a case the chief points of distinction from lead neuritis are the involvement of the supinator longus and extensor ossis metacarpi pollicis and a distinct history of alcoholic excess. Sometimes an old-standing case of lead neuritis comes under observation in which there are contractures in the arms and legs, which have developed after slowly progressive paralyses (including wrist-drop and drop-foot), and in which there is no history of lead poisoning. Unless some source of lead poisoning can be traced, the nature of such a case may not be recognized.

The diagnosis from progressive muscular atrophy may be difficult in the cases already mentioned, which resemble this disease in the nature and distribution of the palsy. A history of lead poisoning or the detection of lead in the urine may be necessary before a positive diagnosis is made.

The distinction between lead neuritis and musculospiral paralysis has been already considered (see p. 490).

Arsenical Neuritis.—The symptoms that characterize arsenical neuritis resemble both those of alcoholic neuritis and those of lead neuritis. These symptoms are paralysis of the muscles of the limbs, especially of the extensors of the hand and feet, and inco-ordination, especially in the lower extremities. With these symptoms there are usually marked sensory disturbances, including severe darting pains in the arms and legs and

anæsthesia of the skin over the affected muscles. The knee-jerks are generally lost.

The chief points of distinction between arsenical and lead neuritis are the usual onset of the former after acute arsenical poisoning, the greater severity of the sensory symptoms, and the involvement of the anterior tibial and peroneal groups of muscles in the leg in addition to the extensor paralysis in the upper extremities.

In the cases that depend on chronic arsenic poisoning (as from the careless use of medicinal preparations of arsenic), the cause of the neuritis is usually sufficiently evident. Where there is some doubt as to the nature of the case the presence of arsenic in the urine may determine the diagnosis. The distinction of arsenical from alcoholic neuritis may be very difficult in the absence of a history of alcoholic excess. Here, again, the finding of arsenic in the urine may clear up the nature of the case.

Neuritis from Carbonic Oxide Poisoning.—This is a rare affection, the symptoms of which vary widely in different cases. In most cases there are obtrusive sensory and motor symptoms, and in many cases vasomotor and trophic symptoms are also present. The sensory symptoms of carbonic oxide neuritis are numbness, partial loss of tactile sensibility and sometimes pain in the extremities—usually over the muscles that are the seat of paralysis. The motor symptoms consist of local paralyses of irregular and variable distribution. In some cases the paralysis has the distribution so often observed in alcoholic neuritis, causing double wrist-drop and drop-foot. Sometimes the paralysis is confined to a single nerve territory (ulnar, radial or peroneal paralysis).[1] The paralysis has been known

[1] There is some reason to think that the paralysis in some of these cases was due to pressure on a nerve-trunk during the period of unconsciousness which usually preceded the development of the neuritis.

to be hemiplegic in distribution. In other cases only the lower extremities have been involved. In one case all the muscles of the eyeball, intrinsic and extrinsic, were paralyzed. The vaso-motor and trophic symptoms of carbonic oxide neuritis include redness of the skin, œdema, changes in surface temperature and herpetic eruptions along the course of nerve-trunks.

The recognition of carbonic oxide neuritis is usually easy, because there is generally a distinct history of exposure to the gas, which is developed in considerable quantities whenever coal or charcoal is burned in a space to which oxygen has imperfect access. In cities a common mode of poisoning by carbonic oxide gas is the inhalation of illuminating gas which has escaped into a closed room. A period of unconsciousness usually precedes by a few hours or days the symptoms of neuritis, but the loss of consciousness may be partial, and is sometimes entirely absent. The neuritic symptoms are often preceded or accompanied with pain in the hypocondriac region, shortness of breath, and dyspnœa on exertion. Transient mental symptoms, such as depression of spirits and loss of memory, are of common occurrence in the early stage of the neuritic affection, and are rather characteristic. These symptoms may persist for many months.

Diphtheritic Neuritis.—The symptoms that indicate the presence of diphtheritic neuritis are usually highly characteristic. They consist chiefly of motor phenomena of peculiar distribution. The paralysis is of gradual onset and development in each part and usually involves in succession different and widely separated regions, so that recovery of power often goes on in the parts originally affected while the paralysis is invading other regions. The order in which the paralysis involves the affected

parts varies in different cases. The paralysis usually begins in the palate, the loss of power being shown by the nasal tone of the voice and the partial regurgitation of food. The paralysis may not spread to other parts, but usually it soon involves the ciliary muscle of the eye and gives rise to loss of power of accommodation, with impaired vision for near objects. Very often at the time when accommodation is first impaired the loss of power extends to the limbs. The legs are usually involved before and in greater degree than the arm. The paralysis is generally slight in degree and always bilateral. It is always of the atonic kind, the muscles being flabby and relaxed.

With these characteristic symptoms there is associated a symptom which is so constant as to be of much diagnostic value. This is loss of knee-jerk. The loss occurs early in the disease, and is generally present even when the legs are not paralyzed.

In severe cases of diphtheritic neuritis there are evidences of implication of the vagus—initial retardation of the pulse followed by irregular and rapid heart action. These symptoms have little diagnostic value, as more characteristic evidences of the disease usually precede them by a considerable time.

The sensory symptoms of diphtheritic neuritis are generally slight. Sometimes there are numbness, tingling, hyperæsthesia, and anæsthesia of the skin, usually at the extremities of the limbs that are the seat of paralysis.

The development of paralysis of the palate and later of paralysis of accommodation and progressive weakness of muscles of the lower extremities constitute so distinctive a group of symptoms that their nature is always clear. There is, moreover, almost invariably the history of a throat affection not longer than a month before the onset

of neuritic symptoms, and such a history removes any doubt as to the character of the affection. The diagnosis may be more difficult when the local evidences of diphtheria have been slight and the palate and eye symptoms have been slight and of transient duration. Any leg symptoms which exist in such a case may then appear to indicate disease of the spinal cord. The presence of ataxia and loss of knee-jerk may suggest locomotor ataxia, but the development of these symptoms is too rapid, and there is too little pain and too much weakness in the legs to permit an error to be made. From acute poliomyelitis diphtheritic neuritis is distinguished by the slower onset, the marked symmetry and slighter degree of the paralysis and atrophy, the slighter changes in the electrical irritability of the muscles, and the presence of sensory symptoms.

Beri-beri, Kak-ke.[1]—These names are applied to cases, apparently of multiple neuritis, of widely varying severity and type, which occur in endemic or epidemic form along the coasts of Japan, Borneo, Ceylon, India, on the coast of Brazil, and along the Red Sea. Cases of the disease are occasionally seen in the United States, chiefly in Malayan and Chinese sailors or in travellers. The condition requires only brief notice here.

The chief nervous symptoms of beri-beri are pains in the extremities, numbness, tingling, hyperæsthesia, and anæsthesia, associated with paralysis and muscular atrophy, chiefly of the lower extremities, but sometimes

[1] There is still considerable question as to the cause of beri-beri. At present the most generally accepted view is that it is a contagious disease dependent on the entrance into the body, probably by the lungs or stomach, of a specific micro-organism. There are many facts which suggest that the disease may arise from an animal poison, which occurs in decomposed tunny-fish or in bad rice.

of the extensors of the forearm as well. In mild cases the development of these symptoms is gradual, and recovery occurs usually in less than a month. In severe cases the symptoms are of rapid onset and soon reach a high degree of development. In some cases the nervous symptoms are most conspicuous, but in the majority of cases the disease is of the "hydropic" or "wet" type, in which the nervous symptoms are inconspicuous and a high degree of general œdema, serous effusion into the pleural and peritoneal cavities, and heart failure are the chief manifestations. In other cases still, also of the severe type, very marked nervous symptoms are associated with the symptoms which characterize the hydropic type and suggest a morbid blood-state. Paralysis of the diaphragm, probably from phrenic neuritis, is an early symptom in many of the severe cases of beri-beri.

The distinction of this disease from other forms of multiple neuritis is usually not difficult if the conditions under which it occurs are known, as it is only observed in the United States in persons who have recently been in the countries where the disease is epidemic.[1]

Leprous Neuritis (Anæsthetic Leprosy).—This condition, which was formerly observed only in the countries where leprosy prevails (Sandwich Islands, India, Norway), is now occasionally met with in the United States. The chief manifestations of leprous neuritis are anæsthesia, muscular atrophy and trophic changes, especially marked in the most distal parts of the extremities and of very gradual development. The anæsthesia is preceded almost invariably by the appearance of a patch of hyperæsthesia, commonly on the hand or foot. In these

[1] A disease resembling closely some forms of beri-beri has been observed in Massachusetts fishermen who have returned from fishing trips, during which they have been on a diet chiefly of fish.

hyperæsthetic areas there is usually more or less spontaneous pain of a burning character. Very gradually, usually after the lapse of many months, the hyperæsthesia is replaced by anæsthesia. In some cases the loss of sensibility corresponds closely to the distribution of one or more nerve trunks; more often the loss does not correspond accurately to nerve distribution. When anæsthesia is established there is usually some weakness and atrophy in the muscles of the part involved, the flexors of the hands and feet become the seat of contractions, and in consequence of the resulting deformities the patient becomes unable to use his hands or to walk. Both hands and both feet are usually thus involved in from four to six years after the first symptoms of the disease. When the muscular atrophy is advanced other trophic changes make their apearance. An ulcer appears usually near the base of the big toe. This ulcer remains small, but after a time involves the joint of the toe. Then the bones of the toe become necrosed and the part eventually separates. In a similar manner the fingers and the remaining toes are gradually involved.

The symptoms that have been described characterize the anæsthetic form of leprosy. Very often these symptoms are associated with the appearance of small hard nodules on various parts of the body, and especially the face, and with fever (so-called "tubercular" form of leprosy) a combination of symptoms known as the "mixed" form of leprosy.

The distinction of anæsthetic leprosy from syringomyelia has been already touched upon (p. 464).

Acute Ascending Paralysis (Landry's Paralysis).—The designation acute ascending paralysis is employed both in a restricted sense and in a more general sense. In

consequence of this difference in usage some confusion has arisen.

Acute ascending paralysis, in the restricted sense, is a disease characterized by : (1) the rapid development of ascending paralysis (legs, then arms, diaphragm, neck, and sometimes palate) with flaccidity of the muscles involved ; (2) loss of reflex action (including knee-jerks) in the muscles paralyzed ; (3) the absence of fever ; (4) the absence of a considerable degree of pain or sensory loss ; and by (5) the absence of muscular atrophy and RD in the cases which live long enough to have made the development of these conditions possible. As to the character of the lesion which gives rise to these symptoms, it is sufficient to say that nothing whatever is at present known, and that there is as yet no sufficient reason for regarding any of the cases of this type as dependent on multiple neuritis.[1]

In the wider sense of the designation, acute ascending paralysis includes, in addition to the well defined group of cases just mentioned, cases in which the loss of motor power has the same rapid development and ascending course as in those cases, but in which there are marked sensory symptoms (pain, paræsthesia, anæsthesia), RD, muscular atrophy, and, in some cases, affections of the sphincters, and fever.[2] There is good reason to believe that many of the cases which belong in this second group

[1] There is some reason to think that these cases may depend on the action of a toxic substance in the blood upon the fibrillary terminations of the upper segment of the motor tract, and upon the motor ganglion-cells of the cord with which these are connected. The relation of some cases to acute specific diseases, and the enlargement of the spleen and lymph nodes sometimes observed, suggest the agency of a toxic substance.

[2] The propriety of including these cases under Landry's paralysis is rather doubtful, but has been unquestioned by some writers of excellent repute.

are instances of multiple neuritis,[1] and it is certainly true that there occur undoubted cases of alcoholic neuritis, diphtheritic neuritis, and beri-beri, in which the symptoms are rapidly ascending in progress and run a quickly fatal course. It is sometimes very difficult to say whether a case is one of Landry's paralysis in the strict sense, or one of the cases that depend on a multiple neuritis. The points to be especially studied in such a case are the sensory symptoms, the state of nutrition of the muscles, and the electrical reactions. Another point of some importance is the fact that in multiple neuritis bulbar symptoms are usually slight or absent, while in acute ascending paralysis they are almost constant. Extensive poliomyelitis, involving the lumbar, cervical, and bulbar centres may simulate acute ascending paralysis, but the early appearance of muscular atrophy and RD, and the absence of sensory symptoms sufficiently indicate the nature of the condition in such cases.

Cases of acute ascending paralysis differ considerably in the rate of their progress. In many cases they end fatally within a week. Sometimes they run their course and terminate fatally within two days. In other instances the disease runs a course of two, three, or four weeks. Most cases that have been reported as instances of recovery from acute ascending paralysis have probably been cases of multiple neuritis or myelitis.

[1] It has been thought that some of these cases are instances of myelitis of a peculiar type, but there is little to support the view.

CHAPTER VI.

THE DISTINCTION OF FUNCTIONAL AND ORGANIC DISEASE.

THE distinction of functional and organic disease of the nervous system constitutes a general diagnostic problem of the highest importance. The object of correct diagnosis being accurate prognosis and rational treatment, it needs no argument to show how necessary is the clear separation of processes that involve actual destruction or alteration of tissue, and limited power of repair, from those which consist in disorders of function and nutrition with practically unlimited possibilities of recovery.

The terms "functional disease" and "organic disease" are commonly employed in the case of the nervous system, as with other parts, in an antithetical sense. We picture to ourselves, on the one hand, a class of morbid conditions in which the symptoms depend on demonstrable structural changes in nerve-elements, or their vascular and connective-tissue belongings, and, on the other, a category of cases in which the symptoms are due to disorders of function alone, without correlative changes in structure. But such separation of categories is, in part at least, erroneous. A functional disease is strictly speaking a derangement of function without a pre-existing alteration in nutrition. The cases that actually belong in this class are probably very few, and

are separated by a wide gap from the class of undoubted organic disease. In this gap lies a large class of cases in which alteration in the nutrition of nerve-elements may be assumed to underlie disordered function. In some cases these nutritional changes are slight, in others they are profound, and it is readily to be conceived that a state is often reached in which the nutritional alterations reach a point where slight alterations in structure make their appearance. There is every conceivable gradation, it should seem, between the disease that is purely functional and the marked changes of organic disease, and experience teaches that many of the functional disorders of one generation are classed with organic diseases in the next, under the influence of improved microscopical technique. The antithetical terms "organic disease" and "functional disease" may designate with sufficient accuracy the extreme types of disease, but do not cover satisfactorily the transitional territory between, and their inadequacy must be borne in mind when they are employed in the wide and rather loose sense which convenience and custom sanction.

It is designed in this chapter to touch upon the salient features of the various forms of functional (and nutritional) disease of the nervous system in their relation to diagnosis, special stress being laid on the distinction of forms which simulate organic disease.

The great type of functional disease of the nervous system is hysteria, and we may well commence with a consideration of its characteristics.

Hysteria.—The word hysteria is employed to designate a morbid state of the nervous system which is especially common in the female sex during the period of adolescence and early adult life, and which consists, broadly speaking, in a primary derangement of the

highest cerebral centres, through which lower centres, both in the brain and in the cord, may become more or less extensively involved in a secondary manner. The variety of these secondary derangements of lower centres give to hysterical disorders a latitude of range which constitutes one of the most striking features of a remarkable malady. In the category of the symptoms of hysteria is included almost every symptom known to organic disease. Here are found paralyses, spasm and contracture, convulsive seizures, states of disordered consciousness, as stupor and coma, mental disorders, disturbances in gait without paralysis, headache and pain of almost every kind and distribution, speech disorders, and the most varied disturbances of general and special sensibility.

These varied forms of hysterical manifestation may, in their various clinical combinations, present a close resemblance to certain forms of organic disease. There are, however, certain broad characters that are commonly possessed by the hysterical conditions, which often serve in making the distinction from organic disease.

First, there is the circumstance that hysterical disorders very commonly develop after *emotional disturbance*, as after fright, grief, or anger. Often, also, the onset of the symptoms follows immediately on an injury which is slight in degree and out of all proportion to the extent and apparent gravity of the consecutive symptoms. A careful inquiry in such cases will generally show that with this slight trauma was associated a considerable degree of emotional disturbance. In some cases the hysterical symptoms come on after the patient has witnessed similar or remotely analogous symptoms in another person—as, for example, where hysterical

tremor develops in a patient who has lived with a case of chorea.

Secondly, there is the fact that hysterical symptoms often increase distinctly on attention. During examination many symptoms may become aggravated. When such increase under attention occurs it is a highly significant sign; but it is frequently absent, and hence its absence has little negative significance.

Thirdly, the symptoms of hysteria are frequently unsettled. They may vary, not only under attention, but without apparent cause. Symptoms of one kind may disappear suddenly or rapidly, to be followed by others of a different character,—in fact, of a character so different that they could not be referred to the same organic process. Thus the hemi-anæsthesia which is right-sided one day is left-sided the next; or, to take a less uncommon instance, a paralysis which is complete, becomes within a few hours partial in degree. Again, anæsthesia and hyperalgesia may rapidly alternate with one another. This mutability in the character of the symptoms is highly distinctive of hysteria.

But the most important element in the diagnosis between hysteria and organic disease is the fact that differences actually exist, in the majority of instances, between symptoms that are hysterical and the corresponding and similar symptoms of organic origin.

As examples of these differences, we may take different types of paralysis: the monoplegic type, the paraplegic type, and the hemiplegic type, and compare the functional (hysterical) form with the organic.

An hysterical monoplegia may involve either arm or leg—more frequently the former,—and may be of sudden or gradual onset. The paralysis may be complete or partial; it is rarely complete from the onset. The

paralysis has a segmental distribution,—*i.e.*, the distribution characteristic of paralysis of cerebral origin; the muscles of the hand are involved, or the muscles of the hand and forearm, or of the hand, forearm, and arm. This segmental character of the paralysis distinguishes it (generally speaking) from nerve-palsies, where the paralysis corresponds to definite nerve distribution, and from spinal paralysis, in which functionally related groups of muscles are involved.

If the paralyzed muscles are contractured, this contracture presents a characteristic which is distinctive of all hysterical contractures, namely, that when an effort is made to reduce the contracture, the resistance experienced is not uniform, but varies from instant to instant and gives the impression of spasmodic voluntary resistance.

This is a very important point of distinction between hysterical and cerebral (organic) paralyses.

In an hysterical monoplegia the paralyzed muscles never undergo any considerable degree of atrophy. The limb may, indeed, grow flabby, and in time undergo such slight diminution in bulk as occurs from disuse; but there is no such wasting as is regularly seen in the paralyses of spinal and neural origin. The electrical reactions are at first normal, and may be normal throughout, but in many instances the slight wasting from disease is associated with slight alterations in the irritability of the muscles (*vide* page 173). The RD is never observed even in its partial forms.

Hysterical monoplegias may be unassociated with any alteration in the sensibility of the skin overlying the paralyzed muscles; but often there are sensory changes. When present, these sensory changes are often distinctive in character.

The most important general characteristic of the anæsthesias and analgesias of hysterical monoplegias is their segmental distribution. Instead of corresponding to the distribution of one or more nerve trunks, as is the case with the anæsthesias and analgesias of peripheral and spinal origin, they correspond in general to the segments of the limb affected—hand, hand and forearm, or hand, forearm, and arm—if it be the upper extremity. In respect to this segmental distribution the hysterical anæsthesias resemble the anæsthesias that result from organic cerebral disease. There is, however, one important point of difference which is, as a rule, available. In the anæsthesia that accompanies an hysterical monoplegia the loss of sensibility extends to a certain level on the limb, and then ends abruptly, but in the loss of sensibility that accompanies a monoplegia due to a cerebral lesion, the sensory loss (which is commonly slight in degree and limited in extent) fades gradually from the periphery towards the central end of the limb. These points of distinction will serve to distinguish even the cases of hysterical monoplegia in which the general indications of hysteria are equivocal.

Under the designation "hysterical paraplegia" are included several conditions, which, though closely allied, should be clearly distinguished. The first class of cases comprises those in which there is a purely cerebral derangement of control over the lower extremities—a paralysis of volition, without even secondary functional derangement of the spinal cord. The central functions of the cord are preserved; the reflexes, superficial and deep, are normal; there is no affection of the sphincters, and the muscles show little or no wasting. But the patient cannot move her legs, or can move them only slightly; only rarely is the loss absolute. When the

patient makes an effort to overcome resistance, the group of muscles chiefly concerned in the act is antagonized in a hesitating or jerky manner by the opposing muscles, and the resulting movement is irregular and jerky. Sometimes when an attempt is made to obtain the knee-jerk, a contraction of the extensors of the knee is prevented by a semi-voluntary contraction of the antagonistic flexors. This may create the erroneous impression that no knee-jerk is present.

In some of these cases of hysterical paraplegia there is some loss of sensibility of the legs, but it is usually slight in degree. It is scarcely conceivable that such a case should be confounded with an organic affection of the cord, but with cases of the second class of "hysterical paraplegias" such an error may be more readily made.

The important characteristic of the cases of the second class is that they present distinct evidence of disordered function of the spinal cord. In addition to considerable or slight loss of power in the lower extremities, there is an increase in the myotatic irritability of the leg muscles. The knee-jerks are increased, and may be obtained by striking the quadriceps tendon above the patella. In rare cases there may be obtained a true ankle clonus, but more often there is merely a semi-voluntary jerky spasm, known as the "spurious clonus." There may be retention of urine, but never incontinence, either of urine or fæces. There is often some spinal pain and tenderness in the lumbar region, but never a cincture pain. The legs may be flabby, but never waste, and never present the RD. Sensibility is only slightly or not at all affected. Attention to these points of distinction from paraplegia of organic origin will prevent an error of diagnosis.

The last variety of hysterical paraplegia to be mentioned differs little in its symptoms from that just described. But it differs in its history in this important respect, that the symptoms of disordered function of the cord are merely aggravated by the development of the hysterical state, and are preceded by a considerable period in which the patients have had slight symptoms of spinal cord disturbance, which are *not* due to hysteria. These symptoms are slight weakness in the legs, fatigue on slight exertion, spinal pain, and flabby muscles, occurring usually in women in depressed health from any cause (but especially prolonged anæmia and debilitating febrile diseases). When hysterical symptoms develop in such women, the existent spinal symptoms ("spinal weakness") determine the direction of development of the hysterical symptoms, and hysterical paraplegia results.

Hysterical hemiplegia may be readily distinguished from hemiplegia of organic origin by the following characters: In hysterical hemiplegia the face is never involved; in organic hemiplegia the face rarely escapes, unless the arm and leg are very slightly involved or the lesion is in the pons or medulla. The contractures of hysterical hemiplegia have the characters noted in connection with hysterical monoplegias (p. 531). The skin reflexes are generally normal, never lessened, as at the onset of organic hemiplegia. The knee-jerks are usually equal, not increased on the paralyzed side. There is never an ankle clonus on the paralyzed side unless there is great contracture of the posterior tibial group, but a "spurious clonus" is sometimes obtained when there is no contracture.

In hysterical hemiplegia the loss of power is never absolute, as it so often is in the organic paralysis, and

the leg is not rarely more affected than the arm. When progression is possible, it is different in hysterical and organic hemiplegia. In the former, the affected leg is brought nearly straight forward from behind, the toes trailing on the ground; in the organic paralysis the palsied leg is advanced with a marked lateral movement, the foot being thrown out in such a way as to describe the arc of a circle. In most cases of hysterical hemiplegia there is anæsthesia of the paralyzed side. It may involve the whole of one side, including the face (hemianæsthesia), or may affect chiefly the arm or the leg. In cases of hemianæsthesia the special senses are implicated in the peculiar manner described elsewhere (p. 127). The onset of hysterical hemiplegia is often rapid, but is never sudden, as is the case with vascular lesions that cause hemiplegia. It is, moreover, never accompanied with loss of consciousness.

The occasional cases of bilateral hysterical hemiplegia, with bilateral or one-sided anæsthesia, cannot be confounded with bilateral organic hemiplegia if the facts relating to the distinction of hysterical hemiplegia are borne in mind.

The most common form of hysterical paralysis is adductor palsy of the larynx, with resulting aphonia. It is important and easy to distinguish this form of palsy from organic laryngeal palsies. Its characters are highly distinctive: there is loss of voice, but, notwithstanding this, the cough is in most instances perfect. In all other laryngeal palsies loss of phonation is associated with loss of cough. In local affections of the larynx, such as laryngitis, there is always an effort to phonate, with resulting hoarseness. Inspection of the larynx in a case of hysterical aphonia shows that the cords move normally during respiration, but that they are not

approximated when the patient attempts to speak. Very rarely abductor paralysis of the vocal cords occurs as an hysterical affection, with laryngeal symptoms like those of the organic form of the same paralysis. The diagnosis here depends on evidence of an associated hysterical affection.

A form of hysterical disturbance known as astasia-abasia is liable to be confounded with inco-ordination due to organic disease. This condition, when typically developed, consists of an inability to stand without swaying violently from side to side, and of an inability to walk without a side-to-side staggering motion, due to the alternate and violent contraction of antagonistic muscles. There is no ataxia, strictly speaking. If the patient be steadied in the erect position the toes are readily guided into contact with neighboring objects. The upper extremities are entirely free from irregularity of movement, and when the patient lies, control over the lower extremities is perfect. Sometimes the defect is hemiplegic in distribution. The knee-jerks are often increased, and the condition is often associated with unmistakable signs of hysteria. These characters serve to separate the condition from the inco-ordination of spinal and cerebellar disease. In its fully developed form and as the dominant symptom of hysteria, this condition is rare. A slighter grade of the disturbance with other hysterical manifestations is common. It appears not to depend on cutaneous anæsthesia.

Hysterical internal strabismus may be confounded with organic paralysis of ocular muscles, unless it is remembered that it is due to spasm, and that the absence of paralysis may be demonstrated by testing the muscles of each eye with the other closed. Hysterical ptosis, when single, is easily detected by asking the patient to look up,

when the orbicularis spasm on which it depends becomes exaggerated in order to prevent the lid from moving up. When double, the head is thrown back when the patient is requested to look up.

The convulsive paroxysms of hysteria are distinguished by their associations from the convulsions of acute organic disease and by their general distribution, irregular character, and tendency to extensor spasm, from the locally commencing seizures that characterize chronic focal lesions of the brain.

There are other hysterical conditions besides those mentioned here that have been confounded with organic disease of the nervous system, but it is believed that the points of distinction given here and in the chapter on symptomatology will suffice in the great majority of instances for the separation of the hysterical from the organic conditions. But certain general facts regarding conditions that appear to be of hysterical origin remain to be emphasized. The first step in the diagnosis of any condition in which there is a suspicion or distinct evidence of hysteria is to look most carefully for symptoms that indicate the presence of organic disease, and not until such disease has been excluded is it safe to assume that the hysterical manifestations present constitute the primary disease. Many women who are the subjects of organic disease of the nervous system (as multiple sclerosis and certain other organic brain diseases) suffer also from hysteria, the occurrence of which is determined by the presence of the organic disease. It is therefore evident that until the possibility of a primary organic condition has been eliminated, even the most pronounced signs of hysteria in a woman of the age at which hysteria prevails should not be allowed weight as to the nature of the primary disease. Close attention to this rule will prevent

many errors in diagnosis. When, as occasionally occurs, the symptoms appear distinctive of neither hysteria nor organic disease, and are to all appearances quite indecisive, a diagnosis, if possible, must be based on a knowledge of the differences (which almost always exist) in the minute characters and associations of the symptoms common to the two different states.

The Traumatic Neurosis or Psycho-Neurosis.[1] —It is generally acknowledged that many forms of injury that are accompanied, at the time of their infliction, with a considerable degree of mental shock (railroad injuries especially), are followed, after a variable period, by nervous symptoms varying widely in character in different cases, but agreeing in this, that they indicate a more or less general disturbance of cerebral function. The symptoms in some of these cases suggest cerebral disturbance of the most purely functional type. In fact, these cases possess the characters that have been described as distinctive of hysteria, and such have been very appropriately designated "traumatic hysteria." Often the subjects are males who have some neurotic predispositions. In some cases the symptoms are such as indicate purely general cerebral disturbance; in others local symptoms, as monoplegic or local sensory disturbance, are especially obtrusive. But even in these cases the cerebral and functional nature of the symptoms is plainly shown by

[1] Actual simulation of symptoms of a traumatic neurosis is very rare, but voluntary exaggeration of symptoms actually present is relatively common. It is, however, not often encountered in hysteria. In neurasthenics the making the most of symptoms is not only an important part of the disease, but is inseparable from it. It is often difficult to say whether a patient exaggerates in spite of himself or intentionally. Some help may be gained from information obtained from outside sources.

the nature of the local symptoms and by their association with more general hysterical manifestations. The criteria of distinction from organic disease are, of course, the same in these cases as those that have been described in the preceding section (*vide* Hysteria).

But there remains a large and important class of cases in which the symptoms following injury and mental fright (shock) are such as to suggest, by their character and permanency, a graver condition than can properly be included even under the broad term hysteria. There is good reason to believe that the symptoms in these cases cannot be referred to a mere disturbance of function, but depend on nutritional alterations in nervous elements, which vary in degree in different cases, and which may even consist, in some instances, in fine structural changes at present beyond the cognizance of histology. The cases belonging to this class present important differences in the severity of their symptoms, and fade imperceptibly, on the one hand, into the severer forms of traumatic hysteria, and, on the other, into the milder forms of diffuse and focal organic disease of the brain.

The term "traumatic neurosis" has been and is still used to designate this class of cases, but its use is open to the objection that it implies a distinct and specific form of cerebral disturbance, whereas it must be owned that the cases included in the term may and probably do differ considerably in their pathology. But the name is convenient, and the objection to its use is much reduced if it be borne in mind that it probably does not designate a pathological entity. Those who wish to emphasize the characteristic and obtrusive psychical element, use the term "traumatic psycho-neurosis."

The traumatic neurosis[1] regularly presents symptoms which cannot be purely functional in character, and it appears an unwarrantable extension of the term hysteria to make it include forms of disease that are so stable in symptomatology that they must depend on decided nutritional alterations in nervous tissues. The "traumatic neurosis" is as much entitled to a separation from hysteria as are the numerous cases of chorea that follow fright. But it must be owned, even by the most zealous advocates of the traumatic neurosis, that there are cases on the borderland between hysteria and the traumatic neurosis, which it is difficult to classify, and about whose classification there is room for a just difference of opinion. There are also cases in which hysterical symptoms are mixed with symptoms of the traumatic neurosis, which cause great difficulties in diagnosis. It must be owned, furthermore, that the entire subject of traumatic hysteria and the traumatic neurosis is beset with difficulties and is likely for a long time to come to afford opportunity for the expression of widely different views.

After a brief description of the main clinical features of the traumatic neurosis will be presented the main points of distinction from hysteria.

[1] Some writers contend that the traumatic neurosis is merely a combination of hysteria and neurasthenia. They hold that trauma acts in these cases by causing a kind of mental over-strain, the short duration of the influence being compensated by its intensity. There can be little doubt that trauma may lead to the development of combined hysterical and neurasthenic symptoms, but this, we take it, does not constitute the traumatic neurosis. The severity of the symptoms in most cases of the traumatic neurosis, and their rapid onset in many instances, would serve, it should seem, to completely separate at least these cases from any that may reasonably be termed neurasthenia.

The symptoms of the traumatic neurosis come on within a short time of the injury and fright, or, as happens with equal frequency, they do not appear for weeks or months afterwards.

In a considerable proportion of cases the patient becomes unconscious for a short period (few minutes) after the injury, or passes temporarily into a state of mental confusion. Much more rarely the period of shock lasts many hours or even days. Sometimes acute insanity (hallucinatory type) follows immediately on the injury. In most cases there are no immediate symptoms. The patient seems perfectly well for several days—is able to work as usual, etc. Then symptoms, at first slight and perhaps overlooked, make their appearance, and with the lapse of time (weeks or months) become gradually more obtrusive. Sometimes the original surgical injury occupies the sole attention of the patient and his physician for weeks or months, the symptoms of the neurosis meanwhile developing insidiously.

The first distinct symptoms are usually purely subjective in character, and include the following:

Pain, especially at or near the seat of injury, and when there is no local injury, but only general concussion, especially in the lower dorsal, lumbar, and sacral regions. The pain is increased by movement in the part affected, and leads to some limitation of motion.

In addition to pain, which is almost always observed, there are usually restlessness, general irritability, and very often an expression of anxiety. The mental changes grow rapidly or slowly more marked, until they develop into a state of hypochondriacal depression, associated commonly with considerable irritability. Insomnia is a frequent and early symptom. It is rarely absolute, but the patient has an unquiet sleep, broken by frequent cries due to bad dreams. General tremor, attacks of syncope, and occa-

sional convulsions may be among the earlier manifestations of the disease. The intellectual faculties remain unaltered or only slightly changed. Some form of motor defect is of almost constant occurrence. The loss of power is usually partial and often very slight. The distribution of the palsy is determined partly by the seat of the injury, partly by other circumstances. All four extremities may be slightly paretic, or there may be more marked paralysis of hemi-, para- or monoplegic distribution. These paretic conditions have many of the characters of the paralyses of hysteria which have been already mentioned —there is little or no atrophy, and the electrical reactions are normal or show only slight quantitative changes. When the paralyses are monoplegic they show a distinct tendency to segmental distribution, never the distribution which corresponds to peripheral nerves or spinal segments. Occasionally a considerable degree of atrophy is observed in the paralyzed muscles, with distinct histological changes in the muscle-fibres.

Sensory symptoms are common and highly characteristic. Very often there is partial or complete hemianæsthesia, like that observed in hysteria, with alterations in the special senses on the same side, especially concentric limitation of the field of vision, for colors and for white.

A variety of disturbances of speech are observed, but there is never aphasia. Most often there is simple slowing of speech ; not rarely the patient brings out his words in an irregular and uncertain manner, apparently owing to mental excitement. The knee-jerks are very often increased, never lost. Occasionally a true ankle clonus is obtained. The pupillary reflex is usually normal, but is sometimes lost. The functions of the bladder and rectum are not commonly involved, but there is sometimes retention of urine, as in hysteria. Loss of sexual desire

is very common. Œdema and cyanosis of the paralyzed extremities are not rarely observed.

One of the most important of the objective symptoms of the traumatic neurosis is tachycardia. In most patients the heart's action is habitually more rapid than normal (90–130), and on excitement may reach 130–140–150 or 160 beats per minute. Sometimes this condition leads to actual cardiac hypertrophy and dilatation.

As a rule, the patients who suffer from the condition described grow progressively worse, by the addition of one symptom to another, and by the gradual or rapid advance of those that first appear.

After a variable period the condition either slowly improves or settles into a chronic state, with slight alterations for the better or worse. The prognosis as regards life is good ; as regards recovery it is bad. Among the most persistent and unchanging symptoms are the mental alterations.

The injury that gives rise to the traumatic neurosis varies much. There may be a slight or considerable local injury, or there may be merely a general concussion. The gravity of the disease appears to bear no fixed relation to the severity of the injury. It is, however, greatly influenced by the mental shock that constantly accompanies these cases. Indeed, it is to this regular accompaniment of the trauma that the peculiar character of the disease appears to be due. That severe mental shock is the essential element in the causation of the neurosis is shown by the occasional occurrence of cases of typical character in which the mental shock has been profound but physical injury could not be detected.

Among the criteria that serve for the distinction of the traumatic neurosis from traumatic hysteria are the following :

I. *The Greater Fixity of the Symptoms* which the two

conditions have in common. This is especially noticeable in the sensory symptoms. The hemianæsthesia of the traumatic neurosis is subject to relatively slight variations in distribution and intensity; there is never a transfer or a rapid change in the degree of anæsthesia. This greater fixity is also very striking in the case of the mental symptoms. There are, indeed, changes, but these are not considerable within short periods of time, and the general nature of the mental disturbance remains the same, whilst in hysteria it may vary much. A similar but less striking difference in the permanence of symptoms is noted in the motor and vaso-motor disturbances.

II. The traumatic neurosis generally occurs in persons who have no recognizable neurotic predisposition, and who have been otherwise healthy. It is very rare, on the contrary, to find cases of hysteria of equal gravity, in which a neurotic predisposition is not traceable or even obtrusive. This is an exceedingly important etiological point of distinction.

III. *Differences in the Symptoms Themselves.*—While there is a strong general resemblance between many of the symptoms of the two conditions, there are important differences in the grouping, detail, frequency of occurrence and character of certain symptoms. Thus in the traumatic neurosis the mental change is a state of hypochondriacal depression, a dominant and characteristic condition. In hysteria a continuous change of this nature is very rare. In the traumatic neurosis, insomnia, in some degree, is an important symptom, and one of whose existence there are objective proofs. If present in traumatic hysteria, it is rarely a prominent symptom. Distinct muscular atrophy, histological alterations in the muscle-fibres, and œdema of the paralyzed extremities are more often observed in the traumatic

neurosis than in traumatic hysteria. True ankle clonus is not uncommon in the traumatic neurosis; in hysteria it is rare. An important and common objective condition in the traumatic neurosis is tachycardia. It is highly characteristic of the condition, and is not a *continuous* symptom in cases of pure hysteria. Generally speaking the symptoms of the traumatic neurosis are symptoms of a graver character than those of hysteria, and suggest more distinct nutritional changes in the nerve elements. Indeed some cases of the traumatic neurosis show conditions that can be referred only to anatomical alterations within the nervous system (optic-neuritis, optic atrophy). The recognition of gross organic lesions, which may be combined with the symptoms of the traumatic neurosis, is of course a simple matter. But it is no easy task, indeed it is often impossible, to state whether a given symptom or combination of symptoms is due to advanced nutritional changes or to slight organic changes. It is important, however, to try to form some conception in each case of traumatic neurosis of the degree of the nutritional changes in the nervous system. The great point to remember is that we have to deal with a nutritional disease of the nervous system, which in its graver forms imperceptibly shades into slight organic conditions (optic neuritis, optic atrophy, central degenerative changes, as shown by loss of pupillary reaction, etc.), which for purposes of prognosis it is extremely important to detect. The detection of such conditions can be accomplished only by making a systematic search for the slightest unequivocal sign of organic disease. It is unnecessary to repeat here what these signs are (see Paralysis, Atrophy, Electrical Reactions, Ophthalmoscopic Changes, Convulsions, Abnormal Reflex Action, etc.).

Neurasthenia[1] is a nutritional disease of the brain due to disturbance of the general nutrition of the body, resulting especially from defective assimilation of food. One of the most important immediate elements in the production of the nervous symptoms is a vaso-motor instability which leads to continual alterations in the blood-supply of the brain. Associated with this condition of vaso-motor instability is probably an alteration in the quality of the cerebral blood-supply, but of this there is no actual knowledge.

The indirect causes of the disease are numerous, but the most important are anxiety, grief, fright, prolonged emotional excitement, and sexual excesses and masturbation. Not rarely neurasthenia is associated with hysteria and with the traumatic neurosis.

The chief symptoms and signs of neurasthenia are general debility, defective memory, slight diminution in will power, abnormal irritability, mental depression, incapacity for prolonged mental or physical exertion, insomnia or abnormal somnolence, headache (often occipital),[2] peculiar cephalic sensations (p. 140), asthenopia, palpitation, tachycardia, fibrillary contractions, blepharospasm, various dyspeptic troubles, night sweats, and concentrated urine.[3] While many of these symptoms are of cerebral

[1] No form of nervous disease is of such common occurrence as some degree of neurasthenia.

[2] The headache of neurasthenia is never severe, and is exceedingly apt to be temporarily diminished after the ingestion of food.

[3] There are few cases of neurasthenia which do not show in the urine or fæces some indication of defective metabolism. The conditions found vary considerably. The fæces often contain excessive amounts of urobilin or some related substance. The urine is usually concentrated and of small volume (600-1000 c.c. in 24 hours). Frequently there is an excessive excretion of phosphoric acid (P_2O_5), and an alteration in the quantitative relation of urea and uric acid. In

origin, many others are due to visceral disturbances, but the latter may be entirely absent. Not rarely there is associated with neurasthenic symptoms that form of mental depression which is due to false ideas of bodily ailments such as might conceivably exist, viz., hypochondriasis.

The distinction of neurasthenia from other forms of functional disease gives rise to no difficulties, but it is important to remember how often it occurs with such diseases. Only occasionally is there difficulty in distinguishing between neurasthenia and organic disease. The cases that resemble general paralysis of the insane are elsewhere considered (*vide* p. 400). Fibrillary contractions may be very persistent, and may be localized for days to special groups of muscles. There is no danger in confounding such cases with progressive muscular atrophy if the associations of neurasthenia are borne in mind. Careful inquiry will usually show that the fibrillary contractions have at some previous time been general in distribution. Persistent headache may be associated with local spasm, such as might suggest a focal brain lesion, but any error may be avoided by remembering that the local spasm of neurasthenia does not recur for more than a few days in the same place, and that the associations of neurasthenia and those of a focal lesion are quite different.

Epilepsy.—Epilepsy is a chronic functional (nutritional) disease of the central nervous system (brain), characterized by the recurrence at irregular intervals of

health the relation of the uric acid to the urea excreted varies between 1 to 45 and 1 to 60 in adults. In neurasthenia (as well as some other conditions) the relation is often 1 to 40, 1 to 35, or 1 to 30. Indican is often present in pathological quantities, especially in cases of sexual neurasthenia. Oxalate of lime is often present in excess in the urine.

convulsive seizures of a certain type, or by the sudden loss or impairment of consciousness, or by a combination of these conditions.

For clinical purposes it is convenient to classify the different forms of epilepsy under the following heads :

1. Common spasmodic epilepsy ("grand mal," "major seizures").

2. Attacks consisting of brief loss of consciousness, with or without slight muscular spasm, or (rarely) of slight muscular spasm, or a sudden sensation without actual loss of consciousness ("petit mal," "minor seizures," improperly called "epileptic vertigo").

3. Attacks consisting of apparently volitional and co-ordinated motor or mental actions during a state of modified consciousness. The mental state during these attacks differs both from normal consciousness and from the unconsciousness that usually occurs in epileptic attacks ("psychical epilepsy," "psychical epileptic equivalent"—so-called because the paroxysms may take the place of typical minor seizures).

These different forms of epilepsy may be variously combined in the same individual. The most frequent combination is that of grand mal and petit mal.

The diagnosis of "grand mal," from organic disease of the brain is always a most important problem, and sometimes an exceedingly difficult one. A diagnosis has to be made from cases of acute cerebral disease (vascular lesions) and from cases of chronic cerebral disease. In epilepsy the convulsions are general, tonic spasm is succeeded by clonic spasm, the duration of the attack is short (less than five minutes), the attack is succeeded by a period of sleep in which the patient may be partially aroused, there is no local paralysis (except in rare cases of unilateral spasm where the weakness is transient),

there is often a history of repeated attacks of a similar character, and there is often an aura. In vascular lesions of the brain, on the contrary, there may be no convulsions, and, if present, they are chiefly unilateral and clonic ; loss of consciousness is usually prolonged, there is hemiplegic weakness with changes in reflex action on the same side, and if the attack is not a first one the number of preceding attacks is small (one or two).

In the diagnosis of epilepsy and chronic brain disease a distinction must be made between active brain disease and old retrogressive lesions. The convulsions of chronic active brain disease (tumor, abscess, chronic meningitis) usually have a local commencement, and are often partial in their extension (*vide* p. 82). This is a most important point of distinction from idiopathic epilepsy ; but it must be remembered that it is not an absolute distinction, for the convulsions of epilepsy sometimes have a local commencement, and the convulsions of organic brain disease may have a general and not a local commencement. Other features than the convulsions themselves have therefore to be considered. It is essential to make a careful inquiry for and to exclude all evidences of organic disease, especially severe and persistent headache, persistent hemiplegic or monoplegic weakness, vomiting without gastric cause, optic neuritis, and paralysis of cranial nerves.

Certain forms of retrogressive, non-active brain disease, as atrophic tumors (tubercular), old cases of so-called polioencephalitis and meningeal hemorrhage in infancy, and old patches of softening, may give rise to convulsive seizures closely resembling attacks of grand mal. Often these forms of disease cause permanent hemiplegia, and

when this is the case the diagnosis is simple. But many of the cases of hemiplegia of infancy recover largely from the paralysis. In any case, therefore, in which convulsions date from infancy, and especially if they are unilateral, careful search must be made for signs of an old hemiplegia. The most important of these signs are *slight* hemiplegic weakness, hemiplegic rigidity, increased myotatic irritability, and slight retardation of growth on the weaker side. The diagnosis, in many instances, depends on these objective features of the case, as the original hemiplegia, if slight, is often overlooked. It is important to note that the first recurrence of convulsions may occur a long time (years) after the convulsions that attended the onset.

In seeking to discover the cause of the paroxysms in any case of grand mal, it is important to study the possible influence of injury to the head. A not inconsiderable number of cases of grand mal follow closely, in their development, upon blows or falls on the head. Many of these cases are indistinguishable by their symptoms from the so-called idiopathic cases of epilepsy. Their recognition is especially important, because in some instances it is possible to relieve them by operation. In many cases local changes are found in the meninges. It is important, also, to recognize the influence which disordered nutrition exerts in some cases of apparently idiopathic epilepsy in determining the occurrence of paroxysms.[1]

[1] The most important evidence of such nutritional disorder is obtained by a study of the urine. In some cases of idiopathic epilepsy (perhaps a considerable proportion) there are distinct evidences of abnormal putrefactive processes in the small intestine. The occurrence of such processes is shown, in a general way, by a disturbance

The distinction of grand mal and the convulsions of hysteria is usually easy. In typical epileptic seizures there is no apparent cause for the seizure, there is often an epigastric or one-sided aura, the onset is abrupt and is often accompanied with a cry, the seizure itself consists of tonic spasm followed rapidly by clonic spasm, frequently the tongue is bitten, the pupils are widely dilated, and the face is pallid or livid at the onset (an important sign), micturition is common, the patient never talks during the seizure, and the duration of the entire attack is never more than a few minutes. These features contrast sharply with hysteroid convulsive seizures, in

of the relation which normally exists between the preformed and the combined or ethereal sulphates in the urine (10–1). In the cases to which reference is made, this relation is habitually changed, so that there is present an excessive proportion of the ethereal sulphates (7–1, 5–1, or even 3–1). The form in which the ethereal sulphates occur in the urine of such cases appears to vary in different instances. Usually they consist in large part of indoxyl sulphate, which is estimated as indigo blue. In other cases they may be represented chiefly by phenol (carbolic acid). It is not, of course, claimed that the presence of these substances is pathognomonic of epilepsy. They are known to occur in excess in most cases of chronic Bright's disease, and probably occur in many other states associated with derangement of intestinal function. This circumstance does not, however, diminish the significance of their presence in excess in certain cases of epilepsy, for it is a striking fact that when these substances are present *they bear a direct relation to the occurrence of the paroxysms; i.e.*, though habitually present in excess they become greatly increased just before the occurrence of the paroxysms, and diminish immediately thereafter. Moreover, in some instances these substances have been diminished by means of intestinal antiseptics, and so long as these have been thus effective, paroxysms have been staved off. It has been claimed that the occurrence of epileptic paroxysms is related to the defective elimination of uric acid. In a large series of unpublished observations which bear upon this question, we have found no support for this hypothesis.

which emotion commonly determines the attack, there is no true aura, the onset is not rarely gradual, there is no cry at the onset, the attack itself consists of periods of prolonged rigidity, and wild, apparently purposive, movements with or without opisthotonos, the pupils never dilate, micturition never occurs, if there is biting it is indiscriminate (hands, lips, tongue, people, and things), talking and groaning are common, and the duration of the attack is much longer than in epilepsy. In epilepsy the attack is followed by stupor; in hysteria the mental state is clear immediately after the attack ceases.

Nocturnal attacks of grand mal may go long unrecognized. The most trustworthy signs of such attacks are a feeling of soreness in the limbs, general headache, and a sense of exhaustion, minute hemorrhages under the skin of the face and neck and in the conjunctivæ (almost pathognomonic), and, in adults, the occasional wetting of the bed. The distinction of grand mal from the convulsions of uræmia and lead poisoning depends on the recognition of the causes of the latter.

There is no organic disease of the nervous system which is likely to be confounded with petit mal. The minor attacks may generally be distinguished from attacks of syncope by their sudden onset and termination, and their occurrence without evident exciting cause (such as usually exists in syncope). Faintness, dizziness, or palpitation may occur in either condition before the attack, but the occurrence of a true aura (visual, olfactory, auditory, visceral) shows the attack to be epileptic. Similarly, slight mental confusion after the attack, micturition and slight rigidity or tremor during the attack, strongly suggest the epileptic nature of the seizure.

A small number of cases of petit mal are distinguished by the occurrence of a vertiginous sensation, and must

not be confounded with true vertigo. In true vertigo there is rarely loss of consciousness, and the dizziness persists, while in petit mal consciousness is generally lost or clouded, and the vertiginous sensation passes quickly away. Aural vertigo (due to labyrinthine disease) (*vide* p. 164.) can hardly be confounded with petit mal, if it is remembered that aural vertigo is characterized by persistent dizziness after the attacks, tinnitus aurium, and often by persistent and defective hearing, none of which conditions occur in epilepsy.

Migraine (Hemicrania—"Sick Headache," "Bilious Headache," "Blind Headache").—Migraine is a paroxysmal nervous disorder of which the most characteristic feature is headache (often one-sided). Nausea and vomiting frequently accompany the pain in the head. In a large number of cases sensory disturbances of one kind or another are associated with the headache, and in exceptional instances these sensory symptoms are the chief elements in the paroxysm, the headache being slight or even entirely absent. Rarely there are motor disturbances in the limbs at the time of the paroxysms.

Of the sensory accompaniments of migraine the most important are the disturbances of vision, which are present in some form in more than half the cases. These manifestations are usually unilateral, and consist in some cases of amblyopia, in others of hemianopsia, and in others of a bright moving spot on one side of the visual field (scintillating scotoma, an early symptom of an attack). The disturbances of speech which not rarely accompany paroxysms of migraine are usually of the nature of sensory aphasia (especially partial word-deafness). When sensory phenomena involve the limbs, they consist, like the visual symptoms, of manifestations of combined sensory irritation and sensory loss: there is tingling suc-

ceeded by numbness and perhaps anæsthesia (fingers most often involved, leg rarely, always unilaterally). When motor symptoms occur, they consist of transient unilateral weakness (arm, especially fingers), and very rarely of slight spasm. Muscles supplied by the third nerve are sometimes paralyzed for a considerable period (weeks—see p. 389).

It occasionally happens that migraine is confounded with organic brain disease. The cases in which this is most likely to happen are those in which migraine is associated with ophthalmoplegia ("ophthalmic migraine"). In these cases the palsy (always one-sided, ptosis, strabismus) comes on acutely with the pain of migraine, but persists for days or weeks with gradual improvement and ultimate recovery, to recur at some future paroxysm of migraine. In acute ophthalmoplegia from nuclear hemorrhage, the symptoms of hemorrhage (apoplexy, etc.) are so obtrusive that no confusion can occur, but from syphilitic ophthalmoplegia the distinction is not always so easy, for the onset of the palsy may be rapid and attended with headache, and there may be spontaneous recovery with subsequent recurrence of the symptoms. The diagnosis is to be made by close attention to the symptoms and character of the onset. In migraine the palsy coincides with a paroxysm which is usually distinctive in character, and the pain ceases or diminishes when the ophthalmoplegia comes on. In syphilitic ophthalmoplegia the palsy is never sudden, and headache (if present) precedes and follows the ocular symptoms.

Cases of migraine of the ophthalmic variety appear to be not rarely followed by general paralysis of the insane. The recognition of the latter disease is sufficiently simple if its connection with these cases of migraine be

known. The symptoms of ordinary migraine recurring after the onset of general paralysis may give rise to error, in that they may be confounded with the paroxysmal attacks of that disease. Mental symptoms resembling superficially the symptoms of general paralysis (defective memory, confusion of thought, hesitation in speech) may occur after severe paroxysms of migraine, but are distinguished from them by their transient duration and non-progressive course. In most cases of migraine an exciting cause can be traced for each paroxysm (overfatigue, dietetic indiscretion, ocular fatigue, sexual excess, or several of these factors combined), and this is sometimes an aid in determining the nature of a paroxysm (as in the diagnosis between migraine and petit mal).[1]

Chorea.—Chorea is a disease especially of childhood and the adolescent period of life, characterized by spontaneous spasmodic and irregular movement (especially

[1] There are probably few typical paroxysms of migraine which are not associated for the time being with evidences of disordered metabolism. The nature of the disturbance seems to be different in different cases. The urine excreted just previous to the paroxysm is usually highly concentrated and may contain a large excess of phosphoric acid. Whether there is any abnormality in the excretion of uric acid in migraine, as some have claimed, is as yet unsettled. It is improbable that there is any constant relation between uric acid excretion and migraine.

In one case of recurrent paroxysmal vomiting, perhaps allied to migraine, there was a striking diminution in the excretion of uric acid during a two days' period of persistent and uncontrollable vomiting. In the long intervals (several months) between the attacks, the patient, a boy of eight, enjoyed excellent health, and the relation between the uric acid and urea excreted was normal (about 1–60); during the attack it was as follows: first day, 1–159; second day, 1–135. Immediately after the attack the relation again became normal.

of face and arm) and by inco-ordination of voluntary movement. With these symptoms are associated very frequently slight loss of power in the parts affected by spasm, and slight, sometimes considerable, mental change. We have observed that an excessive excretion of uric acid is a highly constant condition in cases of pronounced chorea. In the cases that have been studied in relation to this point it has been observed that the excretion of uric acid has reverted to normal in proportion as the symptoms have been controlled by treatment. In many cases of chorea the urine has a peculiar and rather characteristic red (somewhat eosine-like) color, probably not due to urobilin. Another rather common feature of chorea urines is a diminution of the chlorides in relation to the urea. Almost all chorea urines are of high specific gravity (1024–1030) and of very small volume in the twenty-four hours. The disease is usually of limited duration (six weeks to six months), but occasionally lasts (usually in slight degree) for years. The chief immediate cause of chorea is emotion, especially fright.[1]

The diagnosis in the large majority of cases of chorea can hardly be said to present any difficulties. In atypical cases there is sometimes the possibility of confusion with organic disease. Thus, in those cases in which

[1] The frequent association of endocarditis or rheumatism, or both, with chorea, must be borne in mind. The relation between these conditions is not understood, but it is certain that this association is too frequent to be explained by coincidence. It seems probable, upon the whole, that both the chorea and the endocarditis which precedes or accompanies it are dependent upon a blood-state identical with or allied to that of rheumatism. The excessive excretion of uric acid in chorea, which we have noted, is of interest in this connection. The theory that chorea is due to the obstruction (by emboli washed from cardiac valves) of the small vessels supplying the motor structures of the brain has little to commend it.

there is considerable weakness and very little spasm an organic cerebral paralysis may be thought of. But the associated symptoms of a chronic focal lesion (headache, optic neuritis) are absent, the paralysis is limited to one arm, and close attention will show slight choreic spasm and inco-ordination in the affected limb and perhaps in other parts. When the legs are chiefly affected in chorea, there may be difficulty in walking and standing, but confusion with spastic paraplegia need not arise, for in this disease spasmodic movements are conspicuous only on voluntary motion, and an ankle clonus is always obtainable.

Not very rarely the movements of chorea are unilateral (hemi-chorea), and such cases may be confounded with so-called "post-hemiplegic chorea" (mobile spasm, athetoid movements). These movements, however, are usually slow (occasionally they are rapid) and rhythmical, persist during sleep, and contrast with the quick irregular movements of chorea, which cease during sleep. There is also usually more or less rigidity in the parts that are the seat of the post-hemiplegic movements. The history or evidence of preceding hemiplegia of course fixes the diagnosis. Hysterical patients sometimes develop true chorea; in other cases they develop symptoms which while resembling chorea must be distinguished from it (hysterical chorea). In order to justify a diagnosis of hysterical chorea there must be distinct evidence that the attack arose from imitation. The spasmodic movements of hysterical chorea may be indistinguishable from those of chorea, but sometimes they have a rhythmical character, and often they are more violent and abrupt than those of chorea.

Hereditary Chorea (Huntington's Chorea) is an obscure form of disease characterized by spontaneous ir-

regular spasmodic movements resembling closely those of ordinary chorea. In other respects it differs greatly from typical chorea, for it is a disease especially of adult life, is distinctly hereditary (often through several generations), and affects many members of a family, runs a progressive course, shows no special relation to cardiac disease or to rheumatism, never develops from chorea, and is generally characterized by mental failure and eventually by dementia. The disease is to be distinguished from general paralysis, which it may resemble in its mental symptoms, and from bilateral post-hemiplegic athetoid movements. From post-hemiplegic disorders of movement, the absence of rigidity and the irregular and wild character of the movements, together with the history, serve for a distinction. There are, however, cases of bilateral athetosis without any history of preceding acute cerebral disease which it may be difficult to distinguish from hereditary chorea, and which are regarded by some as possibly atypical forms of the latter disease.

Paralysis Agitans (Parkinson's Disease—Shaking Palsy).—Paralysis agitans is a nutritional disease of the central nervous system in the second half of life, and is distinguished by the presence of three cardinal symptoms—tremor, muscular weakness, and rigidity. The disease is chronic and in most instances progressive. The tremor, which is characterized by the fact that it continues during rest, usually precedes the weakness and rigidity, which come on together. Usually the symptoms begin in the upper extremity (hand), extend to the leg of the same side, and if the extension continues, to the arm and then to the leg of the opposite side. The extension of the disease is thus hemiplegic.

Typical, well-marked cases of paralysis agitans present no difficulty in diagnosis. Cases occur, however, in

which weakness and rigidity of the limbs come on without tremor and without the characteristic attitude and facial expression of paralysis agitans, namely, a bending forward of the head and upper part of the spine and a fixed anxious expression. If in such cases there is considerable mental weakness, the diagnosis from organic brain disease may be difficult until the appearance of tremor. An important point of distinction from slight hemiplegia is that the knee-jerks are not increased on one side as in hemiplegia.

The legs are sometimes chiefly affected in paralysis agitans, and in such cases the suspicion of spinal cord disease may arise, but will be dispelled by the detection of slight tremor in the arms and by the fixity of gaze.

The diagnosis from insular sclerosis is always easy if the following facts be remembered: In insular sclerosis the tremor occurs only on movement (intention tremor), and is wider in range and more irregular than that of paralysis agitans. Insular sclerosis rarely commences after forty, paralysis agitans rarely before thirty. The head is regularly involved in the tremor of insular sclerosis; in paralysis agitans it is only occasionally affected. Nystagmus is usually present in insular sclerosis; in paralysis agitans it never occurs. The speech disturbances of the two diseases are different. In paralysis agitans the voice is monotonous and without emotion and expression, and the words are often run together; in insular sclerosis the syllables are unduly separated as a rule. The fundus is normal in paralysis agitans; in insular sclerosis there is often slight ophthalmoscopic change (slight optic nerve atrophy). Furthermore, the peculiar attitude, fixity of facial expression, and muscular rigidity of paralysis agitans are all absent in insular sclerosis.

Post-hemiplegic tremor is fine, while the tremor of par-

alysis agitans is coarse. In post-hemiplegic tremor the carriage and facial expression are normal and a history of hemiplegia is usually available. These points serve to prevent error in diagnosis.

Paralysis agitans is to be distinguished from "senile tremor," a fine tremor of the limbs and head which is

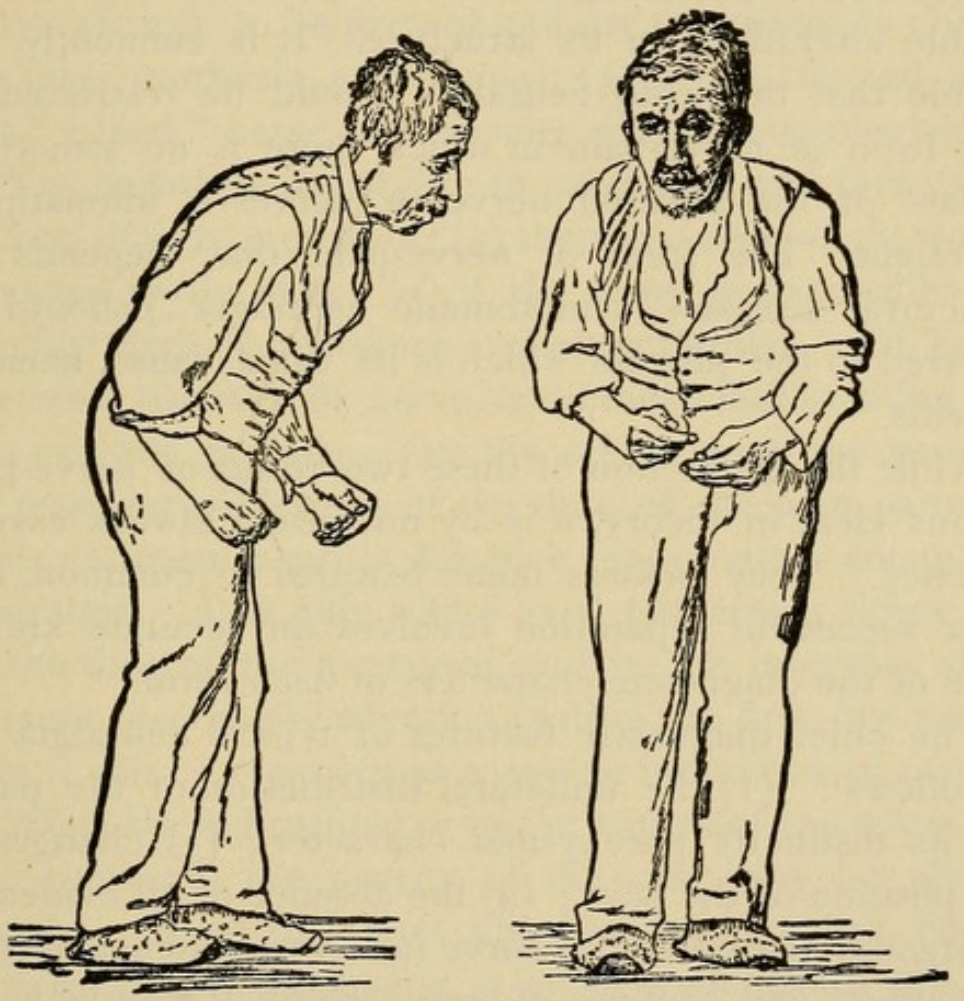


FIG. 66.

Paralysis agitans. (After St. Leger.) (Gowers.)

seen in many aged persons. The tremor is finer than that of paralysis agitans, affects the head early, involves both upper extremities about the same time and is unassociated with the rigidity which is so characteristic a feature of paralysis agitans. These features serve for a distinction from paralysis agitans, but there are cases

intermediate in character between the two conditions which it is impossible to classify.

Neuralgia.—The word neuralgia is merely a euphemism for nerve-pain, and nerve-pain may occur under two very different conditions, namely, as the result of structural irritative disease of a nerve, and as the result of purely nutritional changes in a nerve without any demonstrable alterations in its structure. It is eminently desirable that the word neuralgia should be restricted to that form of nerve-pain in which there is no structural disease of the affected nerve or nerves—"idiopathic" neuralgia. The form of nerve-pain that depends on structural disease ("symptomatic neuralgia") should be referred to the process which is its usual cause, namely, neuritis.

While the distinction of these two forms of nerve-pain is thus clear in theory, it is by no means always easy in practice. They possess many features in common, and their successful separation involves an accurate knowledge of the diagnostic characters of each form.

The chief diagnostic features of typical neuralgia are as follows: (1) the unilateral distribution of the pain; (2) its distinctly paroxysmal character; (3) changes in the position of the pain; (4) the absence of all evidences of organic disease of the nerve involved (absence of local tenderness and swelling of nerve where the nerve is accessible).

In some cases of neuralgia the pain is not migratory, but fixed. Such neuralgia must be distinguished both from the fixed pain of pressure neuritis and from that of slight spontaneous neuritis. Where the nerve-pain depends on pressure (bone disease, tumor), there are almost certainly other symptoms of such pressure, and

the pain steadily progresses in intensity and suggests its dependence on a progressive cause.

The pain of neuralgia is sometimes indistinguishable from that of slight neuritis. Usually, however, a distinction can be made and is based on the following facts: (1) the pain of neuritis is continuous or nearly so; (2) changes in the sensibility of the skin supplied by the nerve are apt to be present and are persistent in character (hyperæsthesia, anæsthesia); (3) if the affected nerve is a "mixed" nerve, the muscles which it supplies become flabby, and their irritability to galvanism and faradism is at first slightly increased; (4) there are often slight trophic changes in the skin; (5) if the nerve is accessible it is usually tender to pressure and often swollen. Of course the mere absence of nerve tenderness and swelling does not exclude neuritis. On the other hand the presence of *transient* tenderness at the time of the pain paroxysm does not mean neuritis, for such tenderness is common in neuralgia. It is only where local tenderness exists both at the time of the paroxysm and in the intervals, and is present *early* in the affection (within the first few weeks), that it can be regarded as a *certain* indication of neuritis.

When the indications as to the nature of the nerve pain are equivocal the history of the case may be allowed weight. Thus the fact that the patient has been a sufferer from nerve pains in other parts makes the neuralgic nature of the affection probable. The etiological factors may also be given some importance. Thus, a family history of neuralgia or of functional nervous disease, a history of a period of general debility before the development of the pain, or a knowledge of the fact that the nerve pains are often related to mental influences, would each of them increase the probability that the trouble was functional and not organic.

Various organic affections of the nervous system are apt to be confounded with neuralgia. We may touch briefly upon some of the errors in diagnosis that are especially liable to arise.

In caries of the upper cervical vertebræ, there are often severe neuralgiform pains in the region of the greater and lesser occipital nerves during the early stage of the disease. Usually the pain is one-sided. The distinction of these cases from cervico-occipital neuralgia may cause some difficulty unless it be borne in mind that the pain of caries is aggravated by motion or jarring and that such pain is associated with spasm in the deep cervical muscles on the side of the pain.

Neuralgiform pains are common in disease of the abdominal and thoracic viscera. Whenever, therefore, there is unilateral neuralgic pain in the trunk, a careful physical examination must be made of the organs near the seat of pain. In some cases of thoracic disease the pain does not correspond to the seat of the disease, but is "reflected" to distant parts. Thus an aneurism of the aorta may cause little local pain, but may give rise to severe neuralgiform pain in the arm. In ordinary intercostal neuralgia there are tender points at certain characteristic situations in the course of the nerve—at a point corresponding to the emergence of the nerve from the intervertebral foramen, at a point near the middle line in front, and at a point in the axillary region about midway between these. These points of tenderness are seldom found in the neuralgias from disease of internal organs. The "lightning pains" of tabes are sometimes experienced in the trunk, especially in its lower part. These pains are, however, of such short duration and so shifting in seat that they are not likely to be confounded with neuralgia. Moreover, they are associated usually with pains in the legs, with disturbances

of sensibility, and, usually, with loss of knee-jerk or loss of the light-reflex of the iris.

The trunk may be the seat of severe neuralgiform pains in the early stage of a tumor of the spinal cord. The severity and constancy of these pains are their characteristic features, and should always excite suspicion of organic disease. Moreover, the pain is usually increased by a lateral or rotary movement of the spine, a feature most suggestive of organic disease. In the cases where there is evidence of pressure upon the cord, there is, of course, no diagnostic difficulty, but it often happens that the neuralgiform pains precede the development of such symptoms by a considerable period. In disease of the vertebral column there is often intense unilateral pain, but this pain is usually much increased by any movement of the trunk.

The diagnosis of *sciatica* may conveniently be considered here. The term sciatica is applied to any condition characterized by pain in the region of the sciatic nerve, whether the pain be due to a primary affection of the nerve, or whether it depends on damage to the nerve from causes outside it. It is well to restrict the word to the primary affections of the nerve, sciatic neuralgia and sciatic neuritis. The chief point of distinction between sciatic neuralgia and sciatic neuritis is that in the latter condition there is *persistent* tenderness of the sciatic nerve.

The pain of sciatica is sometimes felt near the hip-joint, but it is almost impossible to confound the pains of hip-disease with those of sciatica. In sciatica the pain extends far down the thigh in the line of the sciatic nerve, and is increased in this distribution when the nerve is put on the stretch, and there is usually some tenderness (at least at times) in the nerve. When sciatic pain is

secondary to pressure on the sacral plexus by pelvic or abdominal tumors there is usually little tenderness of the nerve in proportion to the degree of pain referred to it. This condition should excite suspicion of organic pelvic disease and lead to a rectal examination. It is important to remember that primary sciatica is practically never bilateral.

Primary neuralgia in the distribution of the anterior crural nerve is rare. Neuralgiform pain in this situation is likely to depend on pressure upon the lumbar plexus, or upon neuritis of this plexus secondary to a sciatic neuritis. In either case there is some wasting of the anterior thigh muscles and slight or considerable changes in their electrical irritability. In many cases there is, moreover, loss of knee-jerk. None of these conditions are ever present in neuralgia.

Occupation Neuroses.—It is here convenient to touch very briefly on the diagnosis of that class of affections which is characterized by the occurrence of certain motor and sensory symptoms (especially spasm and pain) as the consequence of frequently repeated muscular acts appertaining usually to the occupation of the patient.

The most important general characters of these "occupation neuroses," as they are called, are as follows: (1) The symptoms above referred to occur in the part which is concerned in the habitual muscular action. (2) This habitual muscular action is interfered with and perhaps effectually prevented by the symptoms. (3) The most common symptom is *spasm* in the parts affected ("cramp"; hence "writer's cramp," "telegraphist's cramp," etc.). (4) With this spasm, or unassociated with it, occurs pain, referred to the muscles, bones, joints, or nerves, and excited, *at first*, only by the special muscular action.

The chief forms of occupation neuroses are the following: Writer's cramp, piano-forte player's cramp, telegraphist's cramp, needle-women's cramp, and violin player's neurosis. But almost any occupation that involves often repeated special muscular acts occasionally makes a victim of an occupation neurosis. In most cases of occupation neurosis there is a combination of spasm and pain; but very often one of these symptoms predominates, and it is accordingly convenient to distinguish a motor or spasmodic form and a sensory or neuralgic form of occupation neurosis.

The occupation neuroses seldom occur in persons in perfect health. They develop usually in persons who have become debilitated from some cause, and are distinctly neurasthenic. Often a neuropathic heredity can be traced. A period of anxiety not rarely precedes the development of the neurosis, and sometimes its development is related to a local disease or injury. It seems probable that these varied influences operate by producing a functional derangement in the nervous centres (especially the cerebral centres) concerned in the particular act whose frequent repetition has excited the local affection.

The diagnosis of the occupation neurosis is not usually difficult. The relation between a given affection and the action on which it depends is generally sufficiently obtrusive. In the case of the most common of these neuroses —writer's cramp—confusion may at times arise with paralytic affections (chiefly hemiplegia of gradual onset) in which the interference with writing is thought to depend on the neurosis. Knowledge of one fact suffices to make the distinction of writer's cramp from all other forms of disease. It is this: In writer's cramp *the symptoms are at first brought on only by the act of writing*. In

all other affections (paralysis, paralysis agitans, tremors, etc.), the symptoms are *not* limited to the act of writing, but may be observed in all other acts that require the use of the affected parts. This diagnostic feature applies, *mutatis mutandis*, to the diagnosis of the less common forms of occupation neuroses.

Delirium Tremens (Acute Alcoholism, Alcoholic Delirium).—Delirium tremens is an acute nutritional affection of the brain, involving especially the nerve-elements of the cortex. The disease runs a definite course, and has a strong tendency to subside spontaneously. It is practically confined to drunkards, and usually follows a prolonged "spree."

The symptoms of delirium tremens are of rather rapid development and usually attain their full development in the course of a few days. The early symptoms are partial insomnia, bad dreams, restlessness, mental depression, and loss of appetite. When the symptoms are fully developed, there are distressing and rather characteristic hallucinations and illusions, especially of sight, both at night and during the day. Very often the patient sees snakes or spiders crawling on his bed or on the walls, or sees grinning faces of men or devils. The false sense impressions give rise to delusions of a fearful character. Associated with this mental condition is a characteristic irregular tremor of rather wide range, which occurs on movement. The temperature is almost always elevated, the degree of fever being proportioned to the severity of the disease.

The diagnosis of delirium tremens seldom presents any difficulty. From acute meningitis it is distinguished by the absence of headache, spasm, and paralysis. From ordinary symptomatic delirium it is readily distinguished by the great prominence of the terrifying visual halluci-

nations and by the presence of tremor. Sometimes delirium tremens is confounded with general paralysis of the insane or with acute mania. The horrifying hallucinations of delirium tremens cannot be mistaken for the exalted mental and physical activity of acute mania. The characteristic irregular tremor of delirium tremens is absent in acute mania. Considerable irregular tremor may, however, occur in general paralysis ; but the busy anxiety and visual hallucinations of alcoholic delirium make a strong contrast with the unnatural complacency and delusions of grandeur that characterize the organic cerebral affection.

Cerebral Concussion.—A certain degree of cerebral concussion is an accompaniment of many forms of injury to the head, and its symptoms are then lost in those of the trauma. Numerous cases occur, however, in which the only symptoms are such as must be referred to violent concussion of the brain. It is probable that in some of these cases of violent concussion the symptoms depend, in part at least, on the occurrence of minute foci of hemorrhage into the substance of the brain (sub-cortical white substance, sometimes mid-brain and pons). This fact removes these cases from the category of strictly functional disease, but the character of the symptoms is such as suggests functional rather than organic disease, and justifies their consideration here.

The symptoms of cerebral concussion vary much. The blow or fall which causes the concussion gives rise almost immediately to loss of consciousness, or to a confused mental state. In some cases the loss of consciousness is associated almost immediately with irritative phenomena of a cortical nature—unilateral and bilateral clonic spasm, increased reflex irritability, increase in myotic irritability, and often ankle clonus. The pupils are often contracted,

the pulse is slowed, and there may be repeated vomiting. Nystagmus is a common symptom. It is often very rapid, and the oscillations may be vertical as well as horizontal. Rapidly changing strabismus often occurs; internal strabismus alternates with external strabismus. In young children nystagmus, rotary spasm of the muscles of the neck, and strabismus often follow relatively slight concussions (falls from chairs or cribs) without loss of consciousness. These irritative symptoms are usually of short duration (few hours or days), and gradually wear away. In adults cerebral symptoms may persist for several weeks (headache, mental confusion, slight spasm, slow pulse). A slight elevation of temperature occurs in some cases and lasts several days.

The diagnosis from organic disease is not always easy if the history of a concussion is not known. The irritative symptoms (unilateral and general spasm, strabismus, vomiting, slow pulse,) and slight fever may suggest meningitis. The spasm in concussion is often more violent than that of meningitis, and more continuous. The symptoms are also more rapidly changeable. In short, the irritative symptoms are more intensely acute. In concussion the symptoms come on immediately after the injury and reach their maximum in a short time. They also rapidly subside. In meningitis the symptoms more slowly reach their maximum, there is more fever, and delirium precedes stupor. When the irritative symptoms are not so marked, there may be a just suspicion of commencing meningitis for several days. Thus headache, somnolence, mental confusion, slight fever, a slow pulse, and slight spasm may last many days after a concussion, and then gradually clear up.

It is exceedingly important to distinguish between cerebral concussion and extradural hemorrhage. The

two conditions may co-exist. The distinction rests chiefly on the fact that in extradural hemorrhage there are distinct evidences of hemiplegia with or without spasm, while in concussion, paralysis, if present, is slight, and on the fact that the symptoms of extradurul hemorrhage are preceded by a lucid interval, and do not come on suddenly. In extradural hemorrhage, again, there is usually evidence of fracture.

In young children, the significance of nystagmus, or rotary spasm and nystagmus, following immediately on an injury is sufficiently plain.

Malingering.—Malingering is the simulation of a morbid condition which has no basis whatever in fact, and in the case of nervous diseases is not very uncommon. Malingering must be carefully distinguished from the mere exaggeration of a morbid condition, which is very common indeed, especially in hysterical conditions.

Many cases of malingering are so patent that they would not impose on a novice, others require much patience and knowledge for their detection, and others still may deceive the best educated physician. The attempt to feign organic disease, or diseases with decidedly objective signs, is not very common, and need never be successful. But functional diseases, of which the symptoms are chiefly subjective, are often successfully simulated. Thus pain of every variety and distribution may be successfully feigned, but it is more difficult to feign convulsive seizures or paralysis. There is good reason to think that loss of sensibility to pain may be so successfully simulated, even under the severest tests by the faradic brush, the needle, etc.

In any case where malingering is suspected, especial attention should be given the objective signs that may

exist, for the purpose of comparing these, as regards detail and grouping, with forms of actual disease which they resemble. Close attention will always reveal important though perhaps slight differences. Thus while a malingerer may imitate closely the character of seizures of grand mal, examination at the beginning of the paroxysm will show that the vaso-motor symptoms—facial pallor and dilatation of the pupil—do not occur. An almost universal tendency is for malingerers to overdo their part—both in the feigning of objective symptoms and in complaints about subjective states.

But while a diagnosis may often be made from the symptoms themselves, it is commonly desirable or necessary to obtain information from other sources. The history of the patient should be obtained with regard especially to his character, his social and his financial condition. It is especially important to know what incentive there may be for malingering, whether, for example, in alleged traumatic cases, the patient expects to recover damages (as in railroad cases), whether the patient is poor and shiftless or independent and thrifty. An exceedingly important thing, where elaborate malingering is suspected, is to find out whether the patient has had opportunities for the observation of the symptoms of nervous diseases, whether he has lived in hospitals, or has been about from dispensary to dispensary, and has been repeatedly examined for nervous symptoms. Thus some symptoms, as hemianæsthesia, hemianopsia, restriction of the visual field, etc., are not likely to be feigned, but may, nevertheless, be successfully feigned by an intelligent patient who has discovered, by experience at hospitals and dispensaries, the significance attached to these signs. A single unequivocal symptom or sign of organic disease should be allowed great weight

if found. Of such symptoms and signs the most important are local muscular atrophy, changes in electrical reaction (RD), ankle clonus, increase or loss of knee-jerk on one side, slight optic neuritis or atrophy, incontinence of urine or fæces, and paralysis of cranial nerves. Insomnia is a symptom which cannot long be successfully feigned. Its persistence when the patient believes himself to be free from observation is significant of at least functional disturbance.

When the symptoms of actual disease, functional or organic, are exaggerated by a patient, it is exceedingly difficult to estimate the degree of exaggeration, unless this be very extreme. A close watch for feigned symptoms for a considerable period of time may furnish data for the estimation of the degree of exaggeration, but there are many cases in which the best observers would differ in their judgment on this point.

CHAPTER VII.

EXAMINATION OF THE PATIENT.

In the clinical study of nervous diseases it is essential for accuracy and rapidity in diagnosis to employ a systematic method of examining patients.

Inasmuch as the forms of nervous diseases vary so much in character, no one method of examination can always be adhered to rigidly, but this does not destroy the feasibility of using a more or less definite system of study of the cases. In the following pages a general method of procedure is indicated which may be modified or curtailed to suit individual diseases. When a patient is first seen by the physician it is often eminently desirable to make a preliminary rapid survey of the case. Such examination sometimes enables the physician to make a provisional diagnosis for immediate treatment and gain intelligent guidance for subsequent more elaborate and special examination. A preliminary examination should cover the essential points of an extended scheme; a detailed record of the latter is shown in a composite case which is reproduced here to illustrate the points in the examination. In recording the clinical findings of the examination intelligible abbreviations should be used wherever possible, but the use of symbols is to be discouraged for obvious reasons.

The diagnosis must rest largely, at least in most cases,

upon the careful objective or direct examination of the patient for the detection of the signs and symptoms of disease. The history of the patient and of the development of his disease as gathered from the patient or his friends is of course of great importance, but the diagnostic indications that are obtained in this way are to be used in subordination to the objective signs of disease about to be detailed. Where the indications derived from the two sources conflict, preference is always to be given to conclusions that are obtained from an examination of the patient. In short, an effort should constantly be made to make the diagnosis of organic nervous diseases as purely objective as possible, the history being used chiefly in subordination to the physical signs for the confirmation and correction of the diagnosis based upon them.

The history properly begins with a concise statement of the most obtrusive subjective symptoms and their duration: in short, the main symptoms of which the patient complains.

The Family History should contain the cause of death of the patient's immediate relatives, particularly as regards the influence of alcohol, syphilis, tuberculosis, insanity, epilepsy or other nervous diseases in the parents.

The Personal History should be taken and recorded in the chronological order as far as possible, beginning with the patient's intra-uterine life, birth, the accidents and morbid incidents attending the same, diseases of infancy, habits, etc. Evidence of infantile rickets should be carefully searched for as they often play an important rôle in deciding the vulnerability of an individual to nervous disease in after life, particularly as to degenerative diseases, such as epilepsy and allied conditions.

Present Illness.—Data relating to the present illness

should also be arranged in the chronological order of their occurrence, following which a short résumé of the present condition or status of the patient should be given. It should include both the physical and mental state. The principal neurological facts of a mental inquiry concern states of alienation, exaltation, perversion, depression or defect, and whether there be stupor, delirium or coma. The state of memory, the presence of vertigo, convulsions, headache, epileptic or apoplectic attacks, vomitings, etc., should all be inquired into.

The *Physical Examination* is begun by examining the sensory and motor functions of the cranial nerves; this is done by taking each one in its numerical order.

I. *Olfactory.*—Inasmuch as a loss of the sense of smell is less frequently due to a lesion of the nerve than to disease of the mucous membrane, the latter cause must be first excluded. Persons who have lost the sense of smell often complain of a loss of taste only, as that is the more serious deprivation. In testing this sense substances such as assafœtida, musk, peppermint and cloves should be employed, as they act on the sense of smell only; moreover they are easily identified and named. Only a small portion of the testing substance should be used on the bottle stopper or on a piece of cotton wool and applied only for a short time as excessive stimulation may deaden or paralyze a weakened or imperfect sense before the entire examination is finished. The test should be applied to either nostril alternately, the patient being required to recognize and name the test substance. Many persons, even though such common substances as here mentioned be employed, are able to recognize only an odor without naming it, but practical ends will be subserved if this be so, as a

knowledge that the nerve impulses are transmitted is quite sufficient.

II. *Optic Nerve.*—The examination of the optic nerve is restricted from a neurological view-point to (*a*) test of the acuteness of vision, (*b*) the fields of vision, (*c*) state of the optic disc. The acuteness is tested either for distant or near vision, or both, the former by the use of Snellen's test type at a distance of twenty feet (six metres) since rays of light from that distance are practically parallel. The acuteness of vision is expressed by a fraction, the numerator of which corresponds to the number of feet separating the patient from the chart (twenty), and the denominator to the number indicating the distances at which the smallest letters seen should be read by the normal eye. Thus if the patient's sight is normal, his acuteness of vision will equal $\frac{20}{20}$; this would be expressed for the right eye R V = $\frac{20}{20}$. If vision is less than $\frac{20}{200}$, the distance of the chart from the patient should be reduced. If the largest letter is seen at eight feet, R V = $\frac{8}{200}$. If the top letter cannot be read, the distance at which fingers can be correctly counted is recorded, for example, R V = fingers at one foot, or, R V = fingers at seven inches. If even less vision is present the hand is moved before the eyes; if such movements are appreciated a *perception of hand movement* is recorded.

If still less vision is present, alternately shading and exposing the eye discloses whether there is a *perception of light* (*P. L.*) *or not.* This last test may also be tried in a dark room by throwing light upon the eye by means of an ophthalmoscopic mirror. Each eye is tested separately, one eye being covered with a card or an opaque disc. Jaeger's test type is often used for testing near vision.

The extent of the visual fields is best measured by means of the perimeter, the use of which is described in text-books on ophthalmology, but for practical purposes the hand, candle or blackboard test may be employed. Only the first two lend themselves for immediate and rapid use.

For the hand test the patient is turned with his back to the light, the examiner facing at a distance of about two feet. After covering one eye, the patient is directed to fix constantly the eye of the examiner which is opposite. The hand of the examiner is then moved from various parts of the periphery inward, and the patient indicates when he sees the fingers. In this way the examiner compares the patient's visual fields with his own. If both be normal, the patient and examiner must see the fingers simultaneously. This method is simple and rapid and is sufficiently accurate for detecting gross changes in the field. If the patient is not able to see the hand, a lighted candle may be used in a dark room in the same manner. Contraction defects in the visual fields when on one side of the periphery are indicated as the temporal, nasal, upper or lower contraction, or they may be designated as in the superior, inferior, internal or external quadrants. The presence of peripheral and positive scotoma are only detected by the examiner passing the hand through all points of the visual fields. The blind spot is a physiological scotoma.

An ophthalmoscopic examination of the fundus should be made in all cases where organic disease of the central nervous system is suspected, especially where the symptoms point to a cerebral lesion. The direct method of examination is most generally useful but should be supplemented when practicable by the indirect method. The technique of such examination, including the

method ordinarily used for measuring the discs (in case there is marked swelling), is described in text-books on ophthalmology.

III., IV. and VI. *Motor Oculi, Patheticus and Abducens.*—Ordinarily the functions of the third, fourth and sixth nerves are examined together, as they control all the movements of the eye. The state of the ocular muscles is disclosed by requiring the patient to follow the finger first from the right to the left side and *vice versa*, then up and down, and finally by having the patient fix the examiner's index finger as it is moved gradually toward the nose (convergence). The free hand of the examiner should assist the patient in fixing his head in the anatomical position throughout the entire examination. A loss of power in any muscle is proven by (*a*) limitation of movement, (*b*) non-compensation of visual axes, (*c*) secondary deviation of the sound eye, (*d*) erroneous projection, (*e*) double vision and (*f*) the oblique position of the head.

The limitation of movement (*a*) is always in the direction of the action of the paralyzed muscles; thus if the right external rectus (sixth nerve) is paralyzed there would be limitation of movement in the right eye in an attempted movement of that eye toward the right side. (*b*) A non-correspondence of the visual axes is noted as an internal or external strabismus in one or both eyes. In the normal state the visual axes are parallel and remain so in all associated movements of the two eyes.

(*c*) Secondary deviation is the most delicate test of a slight paralysis in any single eye muscle and is never present in any form of ordinary squint. When present this symptom is shown if the sound eye is prevented from seeing the object by employing the hand or a piece of cardboard as a screen and the patient fixes the object with the

affected eye, and the object be moved in the direction of the action of the paralyzed muscle; the sound eye will move still farther in this direction, hence the primary deviation in the visual axes is increased and becomes at once noticeable. This is called "secondary deviation." Its existence can be still further proven by subsequently

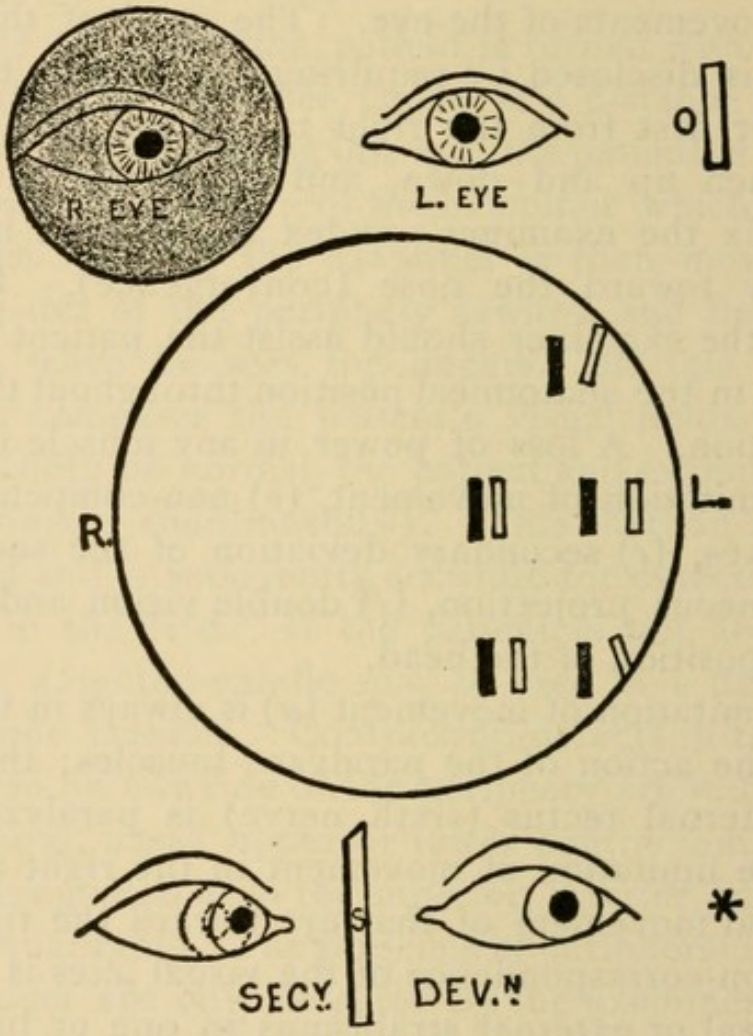


FIG. 67.

Paralysis of the left external rectus; colored glass over right eye; primary deviation on looking towards an object (o) on the left; position of double images; secondary deviation of the right eye when the screen, s, obstructing the fixation of * by this eye, compels fixation by the weak muscle. When the screens are moved, the right eye, in fixing, moves back to the position of the dotted outline of the cornea. (In the diagram of diplopia the highest white (false) image should have been represented a little lower than the true image.) (Gowers.)

covering the paralyzed eye and requiring the patient to fix the object with the sound eye, which then must move back to its former position.

In ordinary squint, deviation occurs in all movements and not in special movement as in paralytic strabismus. In ordinary strabismus the deviation is the same whether the patient fixes with one eye or the other; in paralytic strabismus fixation with the normal eye evokes the primary and that with the affected eye secondary deviation; the two are similar in direction but opposite in character. The primary is a defect and the secondary is an excess of movement.

(*d*) Erroneous projection.—The paralyzed eye doe not see objects in their correct location. The false projection is due to a markedly increased innervation conveyed to the nerve supplying the paralyzed muscle in an effort to force it to act; this gives the patient an erroneous idea of the position of the object. Erroneous projection is really a mental error, an error of judgment, and can be demonstrated by closing the patient's sound eye and requiring him to grasp quickly with thumb and finger an object held in front. The grasp is directed to the side of the object corresponding to the paralyzed muscle. The degree of false projection will be in proportion

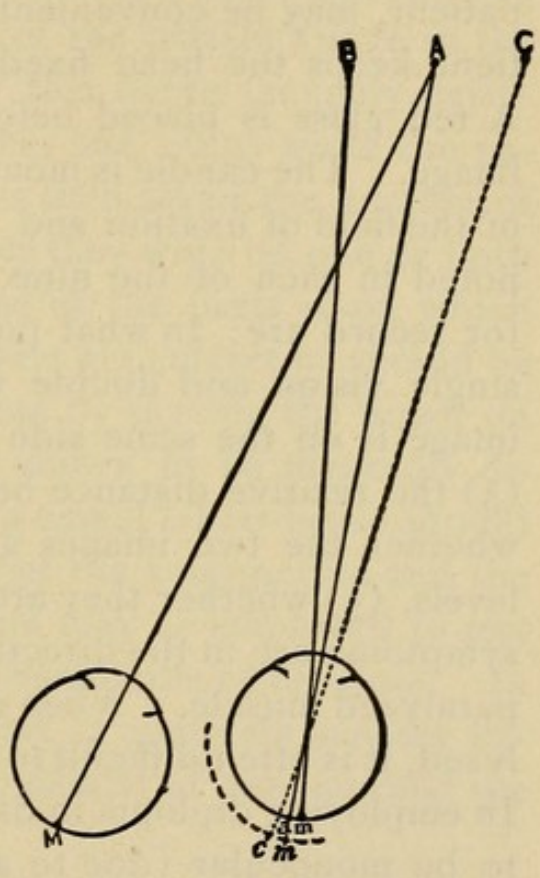


FIG. 68.

Diagram to illustrate the production of diplopia by the erroneous projection. In consequence of weakness of the right external rectus, the image of A formed at the macula, M, of the left eye, falls within the macula of the right eye at *a*. But the effort corresponds to that necessary to bring the macula to *m* (in the broken arc). Hence B, whose image is at *m*, seems to occupy the position of A, and the image formed at *a* to the left of *m* is projected to the other side of A, and seems to occupy the position C, as if it were formed at *c* to the left of *m*. (Gowers.)

to the amount of paralysis; it is also increased in proportion as the paralyzed muscle is required to act. Record is made of the distance between the false and true image.

(*e*) Double vision is detected by means of moving a lighted candle in different positions of the field of fixation. A diagram consisting of two horizontal and vertical lines forming nine spaces held ten feet from the patient, may be conveniently used for record. The patient keeps the head fixed and merely moves the eyes. A red glass is placed before one eye to distinguish its image. The candle is moved about in different positions of the field of fixation and the nature of the diplopia is noted in each of the nine spaces. The data required for record are: In what portion of the field are there (1) single vision and double vision, (2) whether the false image is on the same side as the weak eye or crossed, (3) the relative distance between the double images, (4) whether the two images are on the same or different levels, (5) whether they are erect or inclined. All three symptoms are in the direction of the normal action of the paralyzed muscle. When more than one muscle is paralyzed, it is often difficult to detect the exact combination. In employing diplopia in diagnosis it must first be proven to be monocular (due to a dislocation of the lens), and that it ceases on using one eye only. It is easy to remember that when the prolonged ocular axes are not crossed the diplopia is crossed.

(*f*) There is a characteristic position of the head for every variety of ocular paralysis, which should be noted in each case. The patient turns the head toward the side of the paralyzed muscle and in the direction which the muscle would move the eye if acting. This is done to diminish or correct the double vision. The character

and degree of nystagmus is noted when present in testing for ocular palsy.

V. *Trifacial Nerve.*—Inasmuch as disease of the sensory portion of this nerve is first shown by a loss of the sense of touch, perception and localization of this sensation are the only tests made, particularly if such are negative in results. For this purpose two pieces of cotton wool should be used and touched simultaneously to the same parts of both sides of the patient's face in the three areas of distribution of this nerve (roughly designated the forehead, cheekbone, and chin) while the patient's eyes are closed. He is then asked the number of times he was touched, whether they were on one or both sides of the face and the name of the parts upon which he was touched. If the answers are correct he should be required to localize these spots by placing the finger on them. In case sensibility is shown to be markedly diminished or lost by this test, a camel's-hair brush should be used to test the sensibility of the conjunctiva and the tongue, while snuff or ammonia may be employed to test the irritability of the mucous membrane of the nose. In case touch-sensibility is altered, a complete sensory examination should follow (for details of this method see sensory examination).

Inasmuch as the diminution or loss of the sense of taste on the posterior as well as the anterior aspect of the tongue is counted a disease of the fifth nerve (see Fig. oo, in Chap. I.), this sense should be examined at this stage. For this purpose the following colorless solid substances are used as they do not appeal to any other than the taste sense; sugar, citric acid, salt, and quinine all combine the taste sense of bitter, sweet, sour, and salt; these substances are especially selected as they do not excite the sense of flavor as the latter is appreciated

by the olfactory nerve. Bitter and sweet are tested for at the back, while sour and salt are tested for at the tip and edges of the tongue. Minute areas are not to be tested as there are spots in which one quality can be perceived and not another. In the normal individual there are often differences in the relative power of detecting various substances. The sense is much more acute in the young than in the old. Solids are used in preference to solutions as they enable one to limit their action more easily to the spots examined. A little time should be allowed after the application of the solid for the powder to be dissolved and slight friction usually facilitates this latter process. The tongue should be kept constantly protruded during the test and a prearranged signal be given by the patient as soon as the substance is tasted. The voltaic current is the most accurate means of testing this sense as it directly influences the nerve endings. It may be applied by winding two insulated wires together with exposed ends of a few millimetres apart. A current of one or two cells causes a metallic taste whenever the function is intact. More cells should not be used because pain is then produced and obscures the sensation. In suspected fracture of the base of the skull in which the chorda tympani is involved, the three regions, the front of the tongue, the back of the tongue, and the palate, should be especially tested by this means.

The resulting loss of power in the muscles of the motor portion of the fifth nerve may be recognized by placing the fingers on the masseter and temporal muscles of both sides and requesting the patient to bring the teeth forcibly together as in the act of biting. The feebleness or absence of contraction on the affected side is then evident. If the loss in one muscle is slight the affected muscle may contract a little slower and later than the

others. Paralysis of the external pterygoids is detected (1) by the inability to move the jaw toward the unaffected side, (2) and by deviation toward the paralyzed side when the lower jaw is depressed.

In paralysis of the internal pterygoids the lower jaw cannot be thrust forward. In case there is only slight diminution in power in any one of these muscles, its degree of loss may be disclosed by opposing any one of these physiological actions. The downward movement of the jaw may be somewhat limited after the occurrence of atrophy and secondary shortening of the paralyzed muscles of mastication.

Trophic and spasmodic disorders in the distribution of this nerve should also be noted. In neuralgic states either of functional or organic basis, the painful points and areas of pain-radiation should be recorded. Unilateral furring and accumulation of food in the cheek should be looked for in case there is a lack of masticatory power on the weakened or anæsthetic side.

VII. *Facial.*—The seventh is the motor nerve of the face and is engaged in all voluntary and emotional acts. Its examination consists in requiring the patient to place each muscle in action. He elevates the eyebrows, frowns, closes the eyes firmly, shows the teeth, whistles, puffs the cheeks, and smiles. The difference in voluntary and emotional acts should be noted. In the state of rest, comparison of the two sides may be made for the absence of unilateral muscle tone. Secondary overaction should be looked for in facial paralysis after partial voluntary power returns. It is marked at rest, but slight movement intensifies it while considerable movement will disclose the true nature of the muscular defect. An associated overaction may also be shown in the affected side in asking the patient to close the eye in strong con-

traction, the zygomatici on the paralyzed side will then overact. It may also be excited by reflex action through the fifth nerve (Chvostek's reflex). The character and degree of spasm if present should be examined, first at rest and then during emotional and voluntary movements.

VIII. *Acoustic.*—Inasmuch as disturbance of the function of the auditory nerve is much more frequently due to disease of the ear than to disease of the nerve trunk or centre, examination must be undertaken to exclude the former in making a neurological examination: we are only concerned with alterations of the latter. All accumulations of impacted wax should be removed. The hearing is most frequently tested by the watch held at varying distances from the ear. The use of the tuning fork is most accurate. The patient closes the eyes, or, the examiner's hand is placed so that the patient cannot see the progress of the test. In case of deafness by means of transmission through the air, the watch may be heard on contact with the external ear. If air transmission is not perceived, the integrity of the nerve trunk and centre may be shown by bone conduction. This is tested by placing the fork or watch over the zygoma, mastoid process, or at the root of the nose. In case of slight deafness in which the tuning-fork can still be heard through the air, slight defect in the meatus or middle ear can be demonstrated by noting that the tuning fork is heard for a longer time through bone conduction than through the air. When vibrations have ceased to be appreciated through the air, the fork should be placed quickly against the mastoid process and the vibrations although diminished in intensity will still be appreciated.

Lowered conductivity of the labyrinth is proven if the vibrations from the fork are not intensified by closing the

external meatus. Unobservant persons, however, often fail to note the increase. Galton's whistle is a valuable instrument for testing the slighter grades of nerve deafness as the sound is high-pitched and of short duration. Deafness to all its sounds is pathological. The high-pitched short sound of the watch is also of considerable value. The antitragus may be pressed against the external meatus, the watch may be held near the mastoid or zygoma, but not against it, and then pressed firmly against the bone. If the watch is heard better in contact than when not in contact, the function of the labyrinth is not impaired. If it is not heard better in contact, further observation may be made with the tuning-fork. If the sound is heard through the bone but not so loudly as normal, we cannot infer disease of the labyrinth or nerve, because simple anchylosis of the stapes will reduce bone conduction, although no disease of the middle ear will abolish it. After fifty years of age the watch is often not heard by bone conduction, yet there may be no other evidence of impaired nerve function. The loss is due to change in the labyrinth due to age. Under normal conditions, bone deafness is rare in the first half of life. If such defect is unilateral, the rule still holds good for any age. If Galton's whistle indicates bilateral deafness through the air in advanced life, it is pathological. Bone conduction indicates the functional state of internal ear and nerve and eliminates morbid states of the conducting media, the meatus and tympanic cavity. Vibrations pass directly through the bone to the labyrinth. Perversions or elaborations of simple sounds into those of definite words usually depend upon changes most frequently in some form of insanity or epilepsy.

Electrical tests to discover the power of conductivity of the nerve should not be employed as they cause

distressing vertigo; their results are variable and do not always aid in differential diagnosis between diseases of middle ear and nerve. There are no tests to distinguish between disease of the labyrinth and nerve. Differential aid is furnished, however, by the associated symptoms.

Hyperacusis is determined by the ease with which the high notes of Galton's whistle are heard. Paralysis of the stapedius muscle from disease of the facial nerve may be shown if low notes of the whistle are heard with undue loudness. Pulsating sounds may be diagnosed as vascular if they are found to be synchronous with the pulse, and compression of the carotids causes their modification or cessation.

IX. *Glosso-pharyngeal Nerve.*—Paralysis or weakening of this nerve is shown in requesting the patient to swallow liquids and noting their regurgitation through the nose, owing to imperfect action of the upper muscles of the pharynx innervated by this nerve. The superior larynx should also be tested for loss of sensibility as shown in the abolition of the pharyngeal reflex by tickling the throat. There are no accurate means of detecting a unilateral impairment of the nerve. It is also of little clinical importance.

X. *Vagus and Accessory Part.*—Examination of this nerve resolves itself for practical purposes into testing the muscle action of the pharynx, larynx, thorax, heart, and gastro-intestinal tract. (1) The paralysis of pharyngeal muscles is usually detected by the inability to swallow. Slight disability in swallowing occurs in a unilateral affection of the nerves if no physical obstruction to the act exists. (2) Paralysis of the larynx may be detected by changes in or loss of the voice; an impediment to the entrance of air or an impairment in the closure of the glottis may be shown in the act of cough-

ing. The defective movements may also be directly observed by laryngoscopy. Complete paralysis is shown by an absence of vocal sounds but deep inspiration may still cause stridor.

If the paralysis is complete and bilateral, the ordinary explosive cough is changed to a sudden rush of air through the glottis. One cord is usually unaffected if there is a low-pitched hoarse voice. Slight bilateral paralysis is demonstrated if the cords can be used in phonation and cough although stridor be present on deep inspiration. Furthermore, there will be no expiratory stridor and expiration will be much shorter than inspiration. In laryngeal paralysis the movement of the larynx is much greater than ever occurs in tracheal stenosis. These symptoms when present enable one to make positive a diagnosis of paralysis of the larynx. In unilateral affection of the vocal cords these symptoms may be slight or absent and only a laryngoscopic examination will reveal the paralytic state.

Hysterical adduction palsy is always bilateral, and expiration is noiseless although inspiratory stridor may occur while speech is unaffected; this combination of symptoms differentiates it from spasm of the glottis. Phonic paralysis of hysteria is recognized in adductor paralysis, and in that the patient is able to cough but is not able to speak. The patient is also usually able to sing but not able to speak in a whisper. A less explosive cough is usually evidence of organic palsy. Entire loss of cough and voice suggests bilateral palsy of grave import, but loss of phonation without loss of speech indicates unimportant adduction palsy. Loud inspiratory stridor without loss of voice means adduction palsy also. Loss of cough without loss of voice suggests paralysis of one cord. The following scheme will aid in routine

clinical examining and recording where there are symptoms of laryngeal paralysis.

SYMPTOMS.	LESION.
No voice; no cough; stridor only on deep inspiration.	= Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	= Total unilateral palsy.
Voice little changed; cough normal, inspiration difficult and long with loud stridor.	= Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	= Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	= Adductor palsy.

Alterations in the pulmonary, cardiac, and gastric branches of the vagus are to be diagnosticated only in the absence of organic lesions sufficient to account for the symptoms present, organic visceral disease being excluded. Retarded respiration, accelerated or retarded action of the heart, and vomiting are the more or less positive signs of a nerve disease. The seat of disease of the vagus is to be determined by the distribution of symptoms and their peculiar associated disturbances. Examination discloses much less frequently a disease of the trunk of the the nerve than of the roots and branches.

XI. *Spinal Accessory* (*spinal portion*).—Paralysis of the sterno-mastoid may be detected by the absence of the normal prominence of the muscles, in movement of the head from side to side which may be measured by the observer resisting the volitional movement. Unilateral defect in rotation is always to the side opposite to the

paralysis. There will be no deviation of the head at rest in simple unilateral paralysis if the opposite unopposed muscle does not produce deviation by secondary contraction. Complete paralysis and wasting in the trapezius may be demonstated by putting the muscle at tension in straightening the shoulders behind, the examiner opposing the movement. The shallowness of the concave curve of the outer side of the neck in unilateral defect is brought out prominently by deep respiration. Bilateral palsy of these two sets of muscles is shown in their inability to maintain the head in the upright posture. If both sterno-cleido muscles are impaired the head tends to fall back, while if the trapezii are both affected the head readily falls forward on the chest. It falls to one side or the other according to the inequality of the paralysis of the unilateral two sets of muscles. The marked inequality in the elevation of the palate is best seen in paralysis when the patient utters a long "ah."

XII. *Hypoglossal.*—Paralysis of the tongue is shown, when it lies at rest in the normal position in the mouth, by the increased height on the paralyzed side, due to defect in the posterior fibres of the hypoglossus. A deficiency in the muscle power on the paralyzed side is shown by pressing the finger against the cheek in which the tongue is thrust. On protrusion, however, the tongue is deflected toward the paralyzed side, due to overaction of the unopposed genio-glossus. The amount of deviation on protruding the tongue should be judged by an imaginary line drawn vertically between the upper two central incisors, as the nose often suffers some deflection to the right or left, either from natural asymmetry or injury. The tongue lies motionless in complete bilateral paralysis and cannot be protruded. Slight de-

flection of the tongue on protrusion may not be counted pathological in the absence of other symptoms of impairment of the twelfth nerve.

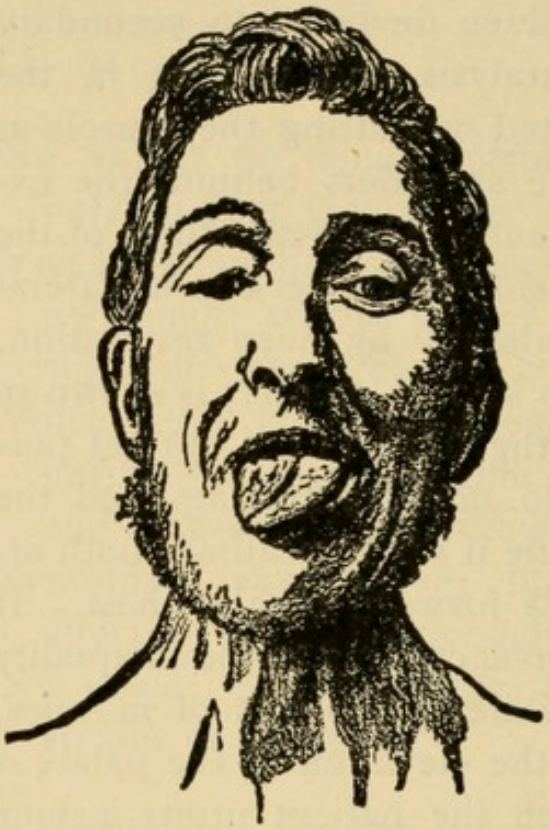


FIG. 69.

Paralysis and wasting of the right half of the tongue, due to disease of the hypoglossal nerve. (Gowers.)

Bilateral palsy interferes not only with articulation but seriously affects the processes of mastication and deglutition. Imperfect mastication may be noted by the inability to keep the food between the teeth. The beginning of the act of swallowing is defective in paralysis of the tongue.

Atrophy in the muscles of the tongue causes the mucous membrane to be thrown into irregular longitudinal folds, which is to be distinguished from geographical tongue, a functional affection.

Defects of articulation are shown in a paralysis of the lips (orbicularis oris is probably supplied by the twelfth) by requiring the patient to pronounce *p*, *b*, and *m*, which, owing to imperfect closure of the lips, are transformed into *f* and *v*. In palsy of the tongue the linguals *l* and *t* are imperfectly pronounced and tend to become *g* and *th*. In palsy of the palate, the defect not only gives an undue nasal resonance to the voice but impairs the explosive labials *p* and *b*, which are transformed into the continuous labial *m*. In mild disorders of the bulbar

mechanism, the patient may be asked to pronounce subordinate consonants and syllables; not only imperfect enunciation irrespective of their character will then be noted, but the syllables themselves often run together or are elided. Sometimes consonants are omitted, or easier sounds are substituted, or consonants are unintentionally repeated. R is especially a source of exact trial as in pronouncing the phrase, "third riding artillery brigade." It is a classic test in developing the paretic type of speech. In other spastic states of the muscles of articulation a staccato or scanning speech may be demonstrated.

Examination of Trunk and Extremities.—In conducting the motor examination of the trunk and extremities, the patient is placed in the anatomical position standing or lying down. The general bony framework, its development, its departures from the normal, from the age, sex, and station in life, and the presence of positive deformities are noted. The examiner must carry an approximate standard of the normal in mind if the lesion is symmetrically bilateral, otherwise a comparison of the two sides may be made. Allowance must be made, however, in comparing the muscular size and power of the two sides in unilateral lesions for the possible presence of hypertrophy on the sound side due to the necessity of increased use of that side. The general state of muscular development, as firmness, softness, etc., is determined by feeling the muscle tissue at rest and in moderate and strong contraction. The degree and completeness of atrophy or hypertrophy is noted in the same manner. Comparative measurements are not usually necessary for practical clinical purposes; the eye should be trained so as not to depend upon such aids for diagnosis. However, when employed as supplemental data to an examination, the following are of common use:

Measurements of the length of arm: (from the acromian process of the clavicle to the end of the middle finger).

Length of leg: (from anterior superior spinous process of ilium to the inner malleolus of the ankle).

Circumference of arm: (at the middle or largest part of the biceps).

Circumference of forearm: (at its maximum of measurement just below the bend of the elbow).

Circumference of wrist.

Circumference of thigh: (*a*, at the gluteal fold; *b*, at the minimum just above the patella).

Circumference of leg: (at the largest part of calf muscle).

Circumference of leg: (at its smallest point just above the ankle).

The measurements should be taken with a steel tape equipped with an automatic reel, as the tension of the spring can be relied upon for drawing the steel band uniformly tight in all comparative measurements. Physiological variation in size of the two sides of the body must be discounted in comparative measurements. Variations of one half to three-quarter inch may be within normal limits in the absence of other positive data of a pathological lesion. Care must be exercised to have the parts held in exactly the same position and degree of muscular tension while comparative measurements are being made. All variations from the normal position should be noted.

Defects in Muscular Power.—The muscular strength of a segment of a limb or a single muscle is best judged by the examiner resisting its action. Examination should be made in a systematic manner from the proximal to distal parts of the extremity. The examiner may

often experience no resistance, yet muscular contractions are sufficiently intact to move the limbs, particularly when the parts are in certain advantageous positions in which muscular action is greatly assisted by gravity. The character and firmness of the muscular contractions are determined by the examiner placing the free hand upon the laboring parts. The actual measurement of muscle power of the upper extremity is confined for practical purposes to determining the relative power of the hand grasp by the instrument known as the dynamometer. The patient grasps the instrument with the hand uppermost, and the arm is freely extended from the trunk, avoiding all artificial support for the extremity, while the instrument is in process of registration. The relative power in health between the right and left hand is in the ratio of 5 to 4 respectively. The rule is reversed in left-handed individuals. For ordinary purposes the patient grasps the hands of the examiner. In examining for cerebral diseases such as hemiplegia, defects in segmental movements are noted, such as that of adduction, abduction, flexion, and extension. This is done because *movements* are represented in the brain and not *muscles*, as in the spinal cord. In disease of the latter, as that of antero-poliomyelitis, for example, the examination of each muscle or group of muscles is necessary.

Actual paralysis is recorded, as to (*a*) range and (*b*) completeness, and whether such paralysis is of the flaccid or spastic type. If the latter exists and cannot be forcibly overcome by ordinary effort, the degree of deformity remaining is noted as permanent contracture. If the paralyzed muscles have shortened, as in old hemiplegias, the state is noted as positive contracture, but if the unopposed muscular antagonists have produced the contrac-

ture-deformity as in old spinal palsies, it is then recorded as negative contracture. A condition of diminished muscle tone (hypotonia) as in tabes is shown in the extreme extension of the joints by passive means.

Tremors are detected while the muscle is at rest and in action. Fine tremors are often made coarse when muscles are placed at tension. Indeed, tremor is often detected for the first time when the muscle is placed at a certain degree of tension. The latter form of tremor, however, should not be confounded with the quaking, coarse tremor of large parts or a segment, such as occurs in the finger or hand, called intention tremor, or, better still, *action tremor*. The latter is seen only when muscles are at increased tension. Tremors need to be noted as to their range (fine or coarse), time (slow or fast), and variation in regularity and equality.

Spasm of muscle is not only noted as tonic or clonic, but its degree of bodily involvement, the excursion or range of muscle movement, duration, and rapidity should be recorded. It is essential that the hand shall be placed on the parts showing tremor or spasm to accurately detect the nature and quality of motor disorders. Simple inspection often gives incomplete or deceptive knowledge.

The degree and nature of inco-ordination or ataxia of the upper extremities is best tested by requiring the patient to touch suddenly with the finger or with a penci different parts of the body with the eyes open and then closed. The direction of movements depends upon sensory elements. It should, however, be remembered that inco-ordination interferes with voluntary movements and *vice versa*. Standing ataxia or dis-equilibration in the lower extremities is noted when the patient attempts to stand with his eyes closed and his feet close together so as to reduce the base of support. The patient in mild de-

grees of ataxy may also be made to stand upon one foot alone. A great many apparently normal persons show considerable sway, laterally and antero-posteriorly under these tests, and all show some. Neurasthenics are apt to show wide variations. If the patient falls or tends to fall under these tests, it is called static ataxy or Romberg's symptom. Ataxy in the gait is brought on by the patient walking a chalk line or a crack in the floor by what is known as "heel and-toe gait." At the end of the line or crack the patient should be required to turn sharply at command and retrace his steps. Slight degree of defective equilibration is often shown only in the act of turning. Inco-ordination or ataxy is usually increased if the patient walk with the eyes closed.

In examining the muscles of the trunk, especially when compression paraplegia occurs at the lumbar segment, the rising of the umbilicus should be noted. This is determined by the examiner holding the finger at the level of the umbilicus and requesting the patient to sit up. The umbilicus will be seen to rise from 1 to 3 inches according to the degree of paralysis of the rectus abdominis.

The spine should be examined for deformity and tenderness. The latter is tested by laying the flat hand or fist over the vertebra in which disease is suspected, then strike this forcibly with the other fist.

Reflexes.—For convenience and thoroughness of examination the following superficial and deep reflexes are examined in the order given.

SUPERFICIAL.	DEEP.
Conjunctival and corneal.	Pupillary.
Cilio-spinal.	Trigemino-facial.
Scapular.	Jaw jerk.
Abdominal and epigastric.	Pectoral.

Cremasteric.	Elbow—biceps and triceps.
Gluteal.	Wrist — supinator and ulnar.
	Knee-jerks.
	Achilles.
	Front tap.
	Babinski.

The conjunctival and corneal reflexes are tested by contact of a twisted bit of cotton-wool. The eyelashes should not be touched in making the examination.

The cilio-spinal is obtained by pinching the skin at the nape of the neck at which the pupils should dilate. The scapular is shown by stroking the skin between the shoulder-blades; the response is noted by the inner-lower angles of the shoulder approximating the spinal column. A quick sharp stroke of the finger-nail or the end of a match along the free border of the ribs brings out the abdominal reflex. The epigastric reflex is demonstrated by stroking the hypogastrium. The same sort of stroke on the inner side of the thigh in man shows a sharp retraction of the testicle of the same side. The gluteal reflex is produced by irritating the skin just above the gluteal fold. By stroking the sole of the foot it is retracted, which demonstrates the plantar reflex.

Inasmuch as the deep reflexes are of greater clinical significance, every precaution should be taken to exclude error in making the necessary tests. In ordinary tests the pupillary reflex is obtained by causing the patient to face a window in such a manner that there shall be an equal lighting of both eyes, while the examiner alternately and conjointly exposes and screens the eyes with his hands.

If the pupils dilate and contract in dark and light respectively, the pupil is said to show *light action*, if not, *light inaction*. The test for accommodation is made by asking the patient to fix first some near and then some far object while the light upon the two objects remains the same; if the pupils respond, the test is indicated as *far, and near action.*

The preservation of far and near action while there is *light inaction* is the condition known as the Argyll-Robertson pupil. In case there is doubt whether accommodation to distance may cause the slight change in the pupils during the light-action test, the patient should be required to fix definitely upon some objective point, the examiner's nose, for example, in a dimly lighted room, and a candle or match may then be brought from behind the patient's head to a position in front of the face so the rays of light may fall upon the pupil. In this way one may definitely exclude the possibility of far and near action confusing the light test. The reflected light from a mirror may be also used instead of a match or candle; the pupillary accommodation to distance can thus be absolutely excluded.

The jaw jerk is shown by tapping the finger which depresses the lower jaw during slight muscular tension.

The biceps jerk is obtained by placing the forearm at a slight angle with the arm and tapping the finger laid on the biceps tendon. The triceps jerk is obtained by a direct tap on the muscle tendon above the olecranon. The arm is partly supported by the examiner, the forearm being allowed to hang loosely. The supinator jerk is obtained by a direct tap on the muscle midway between the elbow and wrist while the forearm is moderately flexed. The ulnar jerk may be shown by striking the tendon of the extensor carpi ulnaris, or the ulnar bone

itself, along the shaft where the latter becomes subcutaneous

The presence or absence of the knee-jerk, the most important muscle jerk, is determined by having the knee so flexed that the quadriceps femoris is gently extended. If then the patellar tendon is struck, the quadriceps contracts and the lower leg is jerked forward if free to move. (Fig. 70.) The blow is given over the space above

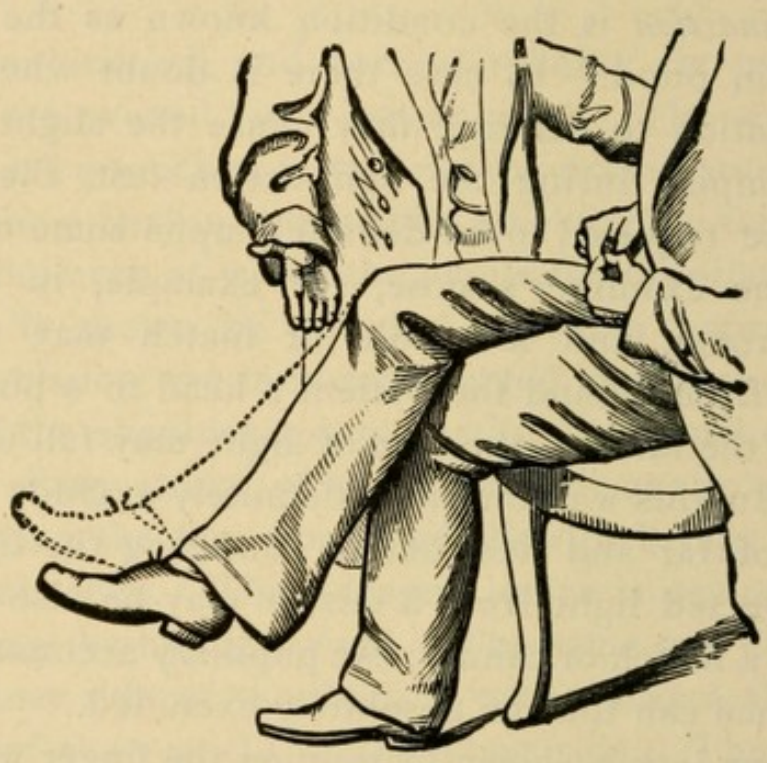


FIG. 70.

The knee-jerk. The dotted line indicates the movement which follows the blow on the patellar tendon. (Gowers.)

the tibia where the tendon can yield if there is a sudden increase in the tension of the muscle. The most convenient position is with the knee to be tested flexed nearly but not quite at a right angle, by being placed over the other knee as the person sits. If the leg to be tested is stout, its tension in this position may be too great to permit of any movement. In such a case the examiner may place his arm beneath the patient's

thigh just above the knee and rest his hand on the patient's other knee. Muscular relaxation is sometimes more readily obtained when the leg hangs vertically or the foot rests on the floor, and the contraction be appreciated by the hand placed directly on the muscle; the effect of the recoil and of the muscular contraction must then be carefully distinguished. If the bent fingers of the hand are hooked together and the eyes closed, the movement is increased in degree—termed reinforcement. It is essential that the flexors also should be free from voluntary contraction. This contraction can be ascertained by feeling the hamstring tendons, and pressure on these by the fingers often helps to secure relaxation. The blow may be given by the side of the hand, a percussion hammer specially devised for this purpose or a continental stethoscope with an india-rubber edge to the ear-piece. When the jerk is doubtful the skin should be bared.



FIG. 71.

The knee-jerk. Method of obtaining it when it is not readily produced in the ordinary way. (Gowers.)

The same contraction may also be obtained by other modes of suddenly increasing the muscle tension, especially when the excitability is abnormal in degree. A blow on the tibia may be effective. While the patient is in the recumbent position with the muscles relaxed the patella may be displaced downward by a finger placed

across the upper edge; tap the finger so as to suddenly displace the finger in the direction of tension; this excites the contraction of the muscles even where there is but slightly increased excitability. If the muscle tension be maintained after the tap and there be great excitability of the muscle clonus, repeated rhythmic jerks may persist while the tension lasts. Similar contraction may be induced in other muscles susceptible of the same treatment. The Achilles jerk is brought out by gently pressing the foot up, thus increasing the tension of the calf muscles; the tendon is then tapped, the muscle contracts and causes slight extensor movement of the foot.

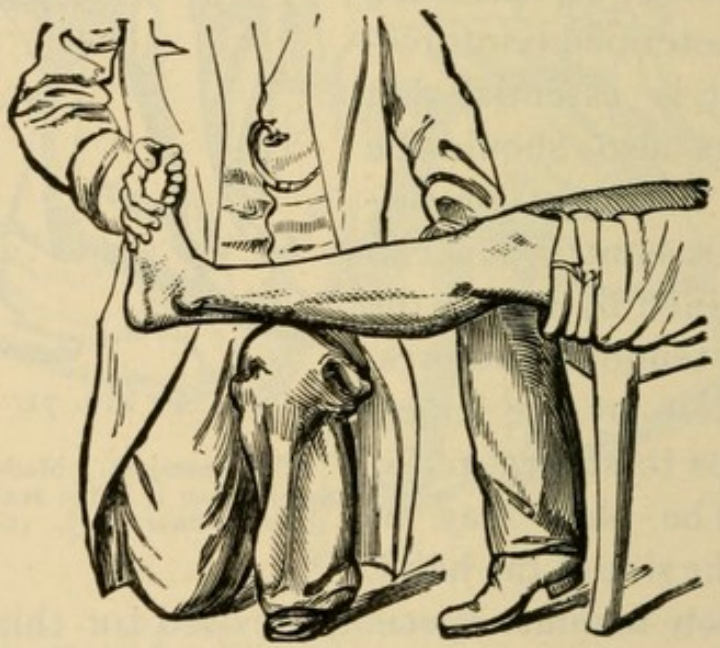


FIG. 72.

Method of eliciting the foot-clonus. (Gowers.)

(Fig. 72.) A tap on the side of the tendon is equally effective, but if the tendon be so firmly supported as not to permit the foot to yield, the hand must be placed on the calf muscles to appreciate the contraction. The cause of the suddenly increased muscle tension is the stimulation and not the blow on the tendon itself. If

the excitability of the calf muscles be excessive the test for the Achilles jerk may excite an ankle clonus.

The front tap is best tested by smartly tapping the tibialis anticus with the hammer or stethoscope while the foot is flexed dorsally by the observer. The ankle should rest on the examiner's knee or on the edge of a stool placed slightly lower than the chair upon which the patient sits, or with the leg extended if the patient is in bed. The reflex consists in plantar flexion of the foot.

In examining for Babinski's toe-phenomenon all possible agents complicating the response must be excluded; the foot should be warm, the leg partly flexed to aid muscular relaxation of the parts and to avoid the occurrence of voluntary movements while the test is being made. The outer sole of the foot is stroked more or less slowly by the thumb-nail or a piece of match. If this pathological sign be absent, the plantar flexion of the big toe is observed, but if present, the toe is dorsally flexed, or the flexor longus proprius tendon is seen to flick up notwithstanding the muscle action is not sufficient to actually move the big toe. The movements of the other toes should be disregarded. They may or may not coincide in the big toe movements.

Electrical Examination.—In making the electrical examination the sponges should be kept wet by frequent dipping in a weak solution of common table salt or bicarbonate of soda. When examination for reaction of degeneration by means of polar changes is made, as a matter of convenience, one pole (indifferent electrode) should consist of a large flat sponge pad applied over the sternum, at the sacrum, or nape of the neck. The patient must fully relax the muscles to be tested. The exciting electrode should have a small surface to concentrate the current. The amount of electricity passing should be

gauged by a galvanometer, as in a reversal of the poles frequently less current passes during the first two or three applications of the isolated shocks. A decision that a polar change exists should be inferred only when there is an equal amperage to both positive and negative current at the point of test. In the milder stages of nerve degeneration the *slowness* of *muscular contraction* is noted as a slight sign of R. D. in the absence of positive polar change. In children and in sensitive individuals it is preferable to use the isolated induced shock as it is less painful. Before actually applying the current the examiner should first receive the current upon his own person for the sake of safety to the patient and to minimize the patient's sense of fear of the proposed test. Faradization should be first tried and if the reaction is normal galvanism is unnecessary. In case there is a diminished or lost response to faradism (although not necessary for diagnosis), galvanism may be employed for the purpose of prognosis. The minimum of galvanic current to which an electric response may be obtained should always be employed. Many times when there is a slight but equal diminution of responses to both poles, the degree of departure from the normal can be determined by comparing the same parts of the sound side with those diseased, or in case of a bilateral lesion an analogous part in a sound extremity may be used for comparison. The designation "lost" as applied to an absent response means a negative test to that current which can be borne by the patient without great pain. Even under such circumstances a response might still be gained under anæsthesia, but for practical purposes such a procedure is never necessary. Fig. 73 and Fig. 74 show the motor points of the inner and outer side of upper extremity, while Fig. 75 and Fig. 76 are the

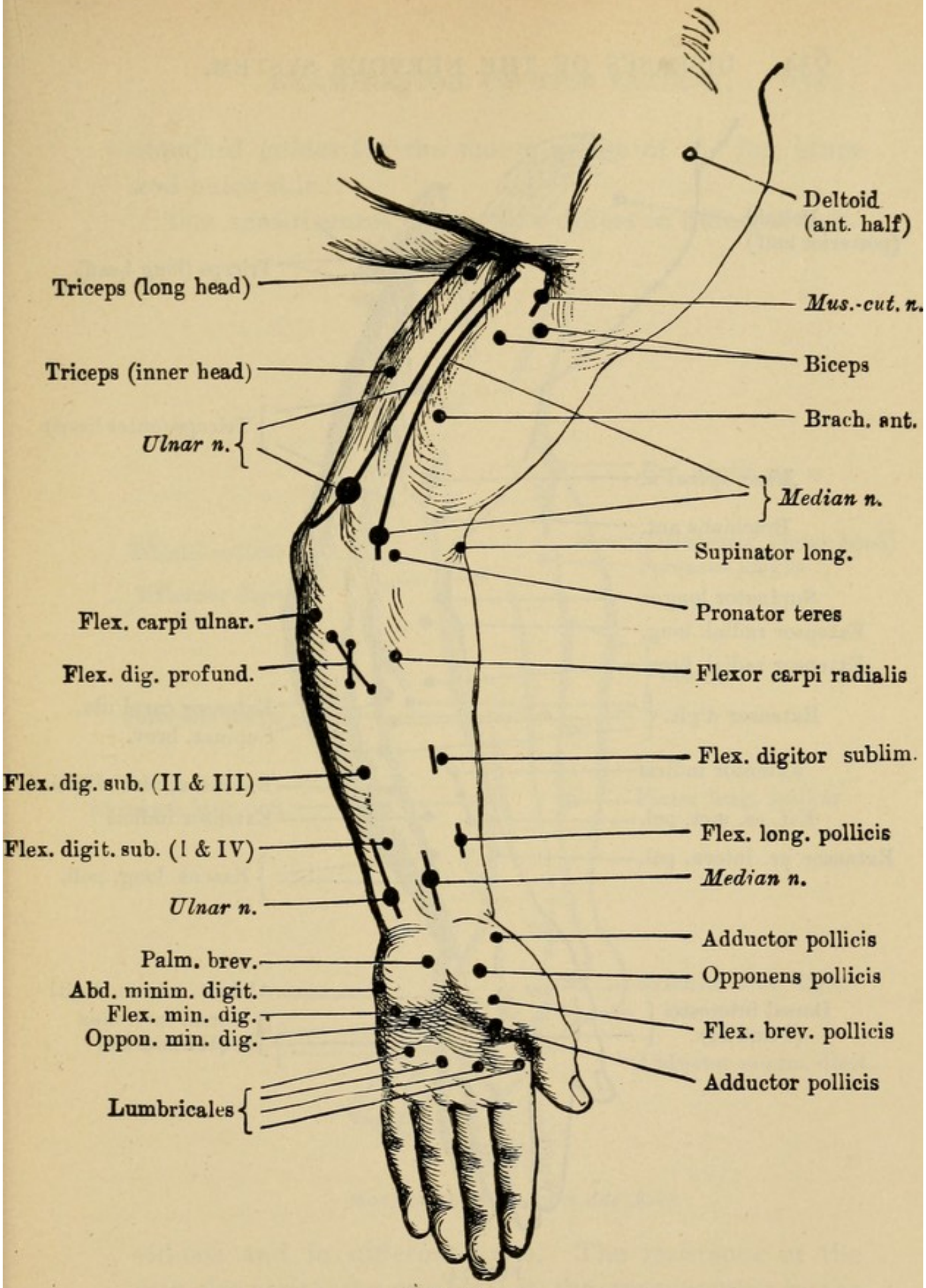


FIG. 73.

Motor points for the arm, inner side (from Erb). The points at which the muscles and nerves can most effectively be stimulated.

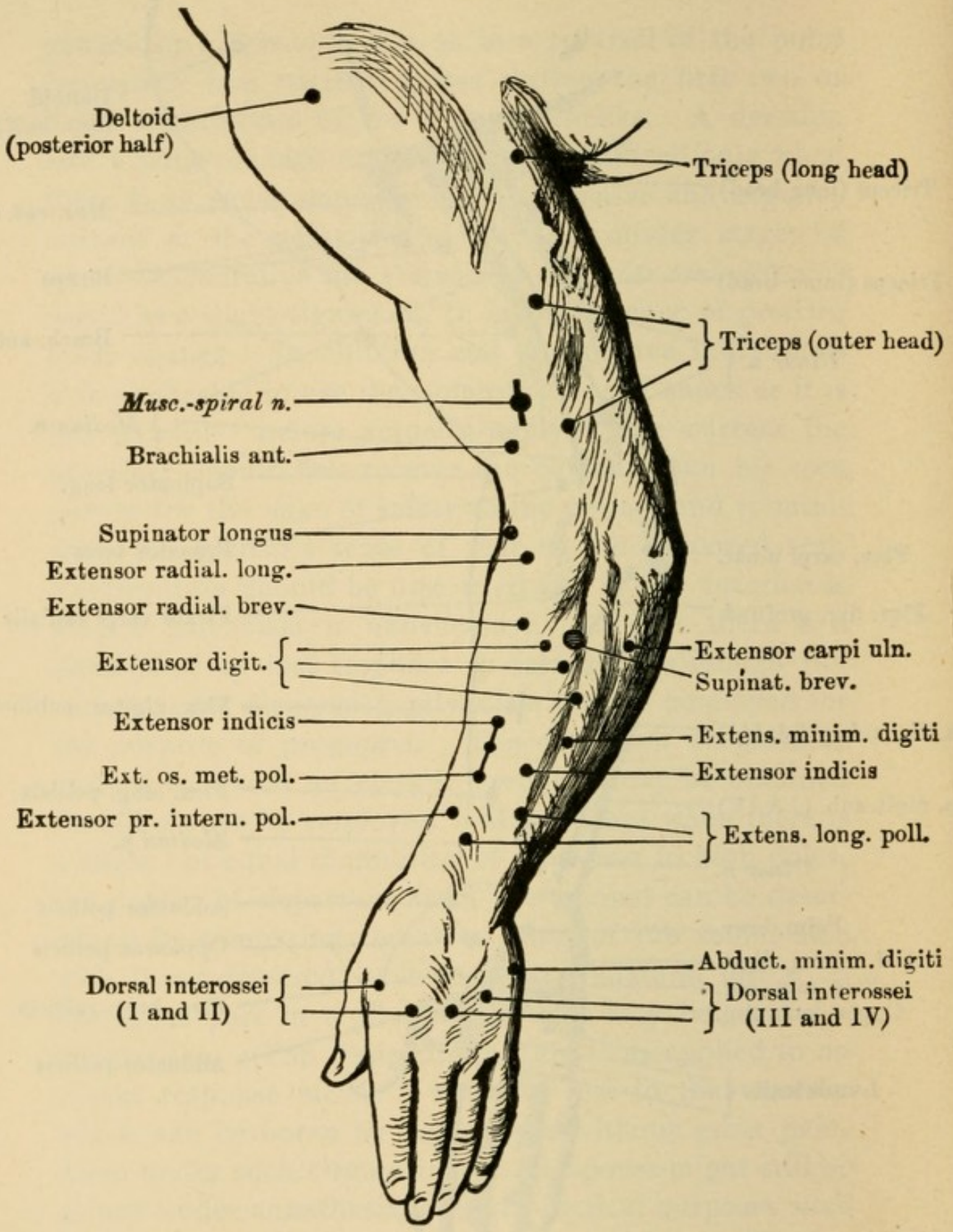


FIG. 74.

Motor points for the arm, outer side (Erb.)

standard guides for the motor points of the leg, inner and outer side.

The sensitiveness of the skin differs in different indi-

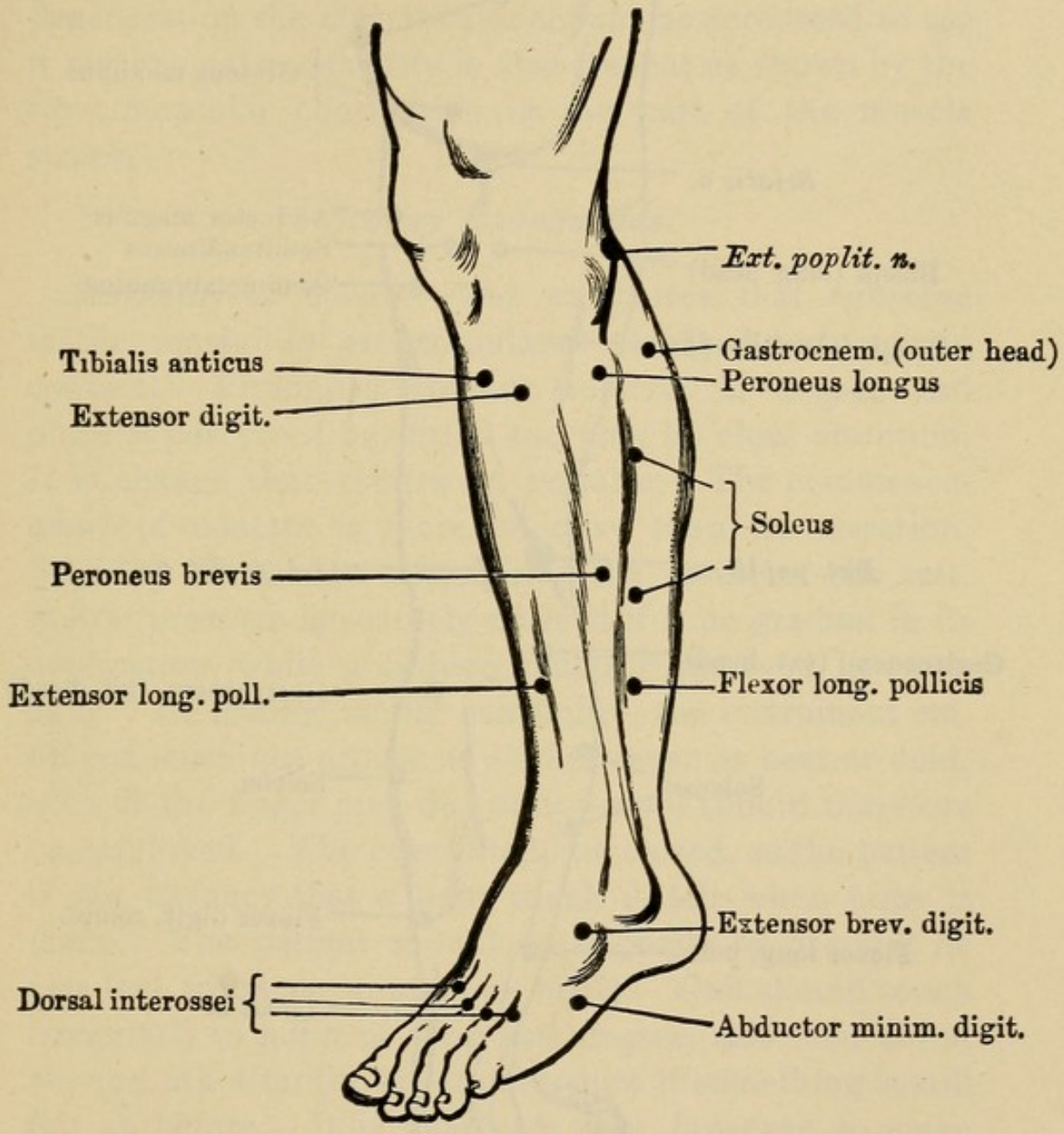


FIG. 75.

Motor points of leg, outer side (Erb).

viduals and in different parts. The resistance of the skin also varies; the greater it is, the stronger must be the current used to secure the action on the nerves below.

Hence the importance of ascertaining what current is actually passing by use of the galvanometer. It may be

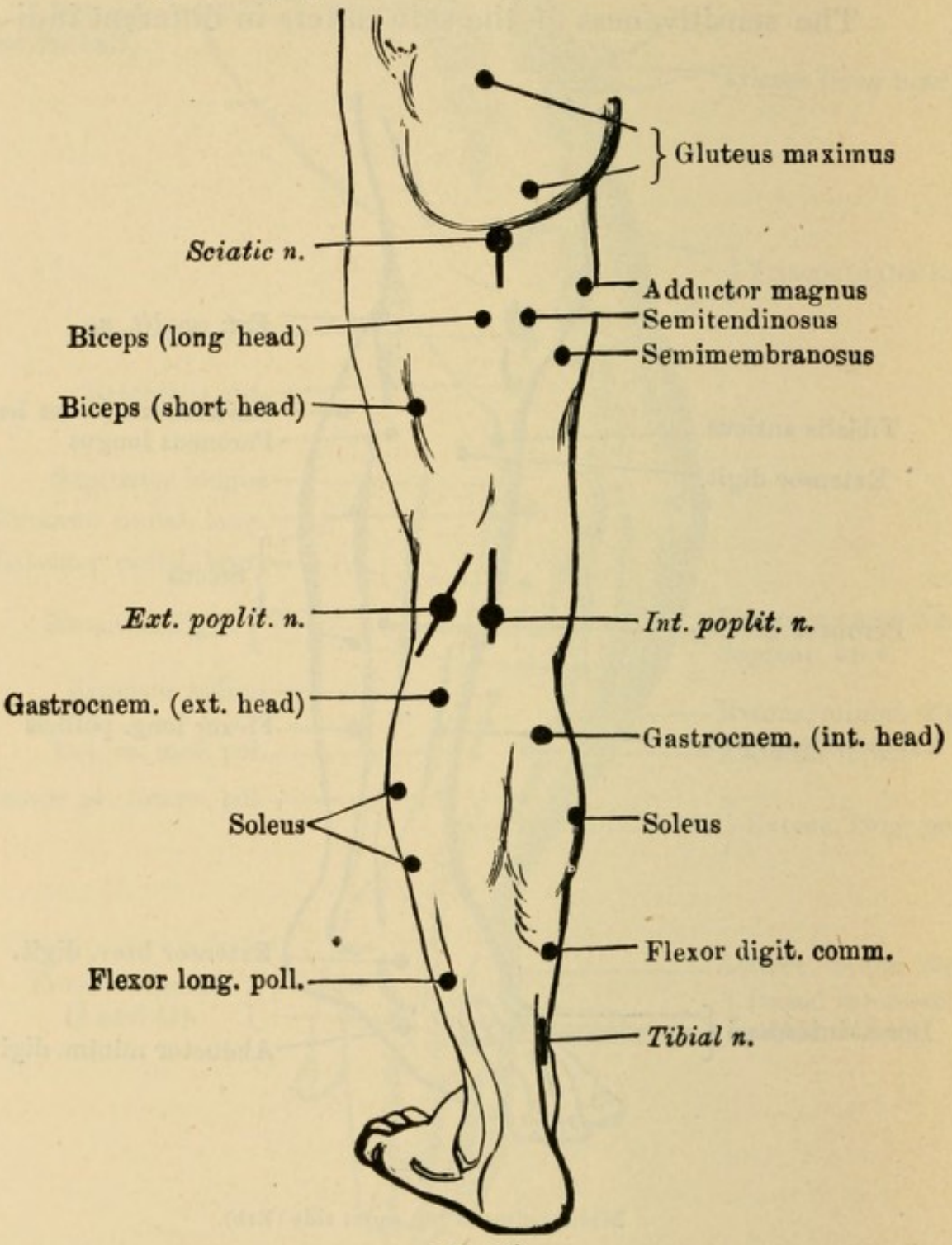


FIG. 76.

Motor points, back of thigh and leg (Erb).

used once to gain information as to the significance of

the number of cells that constitute the evidence of the difference between the two sides, when the sensitiveness to the pain which is caused prevents the use of this instrument in all measurements. In complete reaction of degeneration the muscles also should be percussed to see if mechanical excitability is also present as shown by the slow muscular contraction in the part of the muscle struck.

Sensory Examination.

Sensibility to Touch.—The structures that subserve tactile sensibility are stimulated chiefly by change in contact. Prolonged contact uniform in degree and place is perceived but little and only by close attention. It is change that excites an impulse. The commencement of contact is more effective than its cessation. Rapidity of change determines the result of the test. A firm pressure is scarcely noticed if it be gradual in its application, while a sudden touch is observed however light. In testing tactile sensibility, the instrument employed must not arouse any other sense as heat or cold, such as the finger may do; cotton-wool should therefore be employed. The eyes should be closed, as the patient is apt to fancy that a light touch is felt when none is made. The patient should occasionally be asked if he can feel when no contact is made. One should touch irregularly in point of time and degree, and then not at all, and ask after the latter procedure if something is still felt as before. It is nearly as easy, however, to make the test too fine as too gross. If the skin be thick, sensibility of the skin is normally low, and light touch is often unappreciated in the healthy under such conditions. The test employed may be tried on the examiner himself, which affords a normal standard always at hand. The term

"sensibility to touch" should be used instead of "anæsthesia," as the latter is often inaccurately used. The patient should not only be asked if he feels a touch, but should be required to name the part touched and be able to localize it with the finger. The part touched should be constantly varied, so the patient may not become schooled to the examination. The acuteness of touch sensibility is subject to a certain degree of measurement, as the distance apart at which two points are disconnected is nearly the same in different individuals in the same part of the body, although it varies much in different parts of the same person. The approximate normal distance being known, the increased distance at which two points have to be placed for their discrimination is a measure of the degree of defect. The points should be a little dulled so as not to cause pain. Common compasses may be employed, but the most convenient æsthesiometer is Sieveking's in which the points are attached to a graduated bar. The principal points are:

Tip of tongue	1.5 min.
Finger-tips	2 to 3 min.
Lips	4 to 5 "
Tip of nose	6 min.
Cheeks and back of fingers	12 "
Forehead	22 "
Neck	34 "
Forearm, lower leg, and back of foot	40 "
Chest	45 "
Back	60 "
Upper arm and thigh	75 "

The distance is smallest at the tip of the tongue—that is, the sensitiveness is greatest. The most important aver-

age distance is the normal at which two points can be recognized.

Slight variations exist in different individuals, and a deviation from the normal that is uniform in degree throughout the body is probably physiological. The skin must be touched at the same time and with equal degree of pressure by both points. The examination requires time and patience. Increased power of discrimination on the part of the patient is acquired by practice. Conclusive results are rarely obtained unless there is a degree of defect that causes absolute loss; the test is therefore of much less practical value than was once thought.

Sensibility to pain is subserved by nerves of common sensibility; it is usually tested by a blunt pin-prick. A finer instrument such as a needle, should not be used; it may not only penetrate the skin, but in less sensitive parts where nerve endings are not close together a fine point may be unfelt although readily near by. The wire terminals of a faradic battery need to be applied with great care and the current carefully graduated if gain in exactness is to be obtained over that by the use of a pin-prick. Touch may produce pain if the common sensibility is greatly increased. Both tactile and painful impressions may produce subjective sensations distinctly abnormal, such as thrilling, tingling, etc.

When a prick is only perceived as touch, pain may not be lost but only delayed, as the patient only names the sensation first felt. The patient should be asked to name the sensations the instant they are perceived and whether any new sensation is perceived after when sense of touch is intact. The test to pain must be kept uniform, gauged in amount by exposing only a small part of the pin-point between the fingers. Sensibility to pain is often diminished but not lost. Patients often mistake

the degree of pain they should recognize and often vary this idea during the examination; to avoid these two errors, frequent comparisons to some approximate standard of sensibility in another part of the body should be made. To these errors must often be attributed the variation and unreliability of many examinations.

Temperature Sense.—While sensibility to pain may vary although touch is intact, temperature is usually affected with sensibility to pain, but often not in the same degree, and one may be impaired, as in syringomyelia, without the other. Slight losses of temperature sense may not be detected, or there may be an absolute loss to both heat and cold as such.

While considerable degrees of difference are recognized correctly, a quality of difference is perceived analogous to that noted in the perception of difference of pressure. Hot objects may feel cold, and *vice versa*. Ordinary temperature may produce pain, or heat may be less readily perceived than normal. In cases showing an increased lapse in time between application of pin-prick, a similar delay in temperature test is so shown. In health, temperature sense is less quickly produced than that of pain, because time is required to raise the temperature of the skin sufficiently to enable the heat to stimulate its nerves.

For rough examination hot and cold spoons may be employed, or a hinged tongue depressor, one blade of which may be heated; better still two similar test-tubes containing hot and cold water may be used. Both heat and cold must be sufficient in degree to be unquestionable, so that the patient's doubt may be definitely recorded as pathological and not due to the uncertainty of lukewarmness of the testing object. For ascertaining the power of differential discrimination, large test-tubes

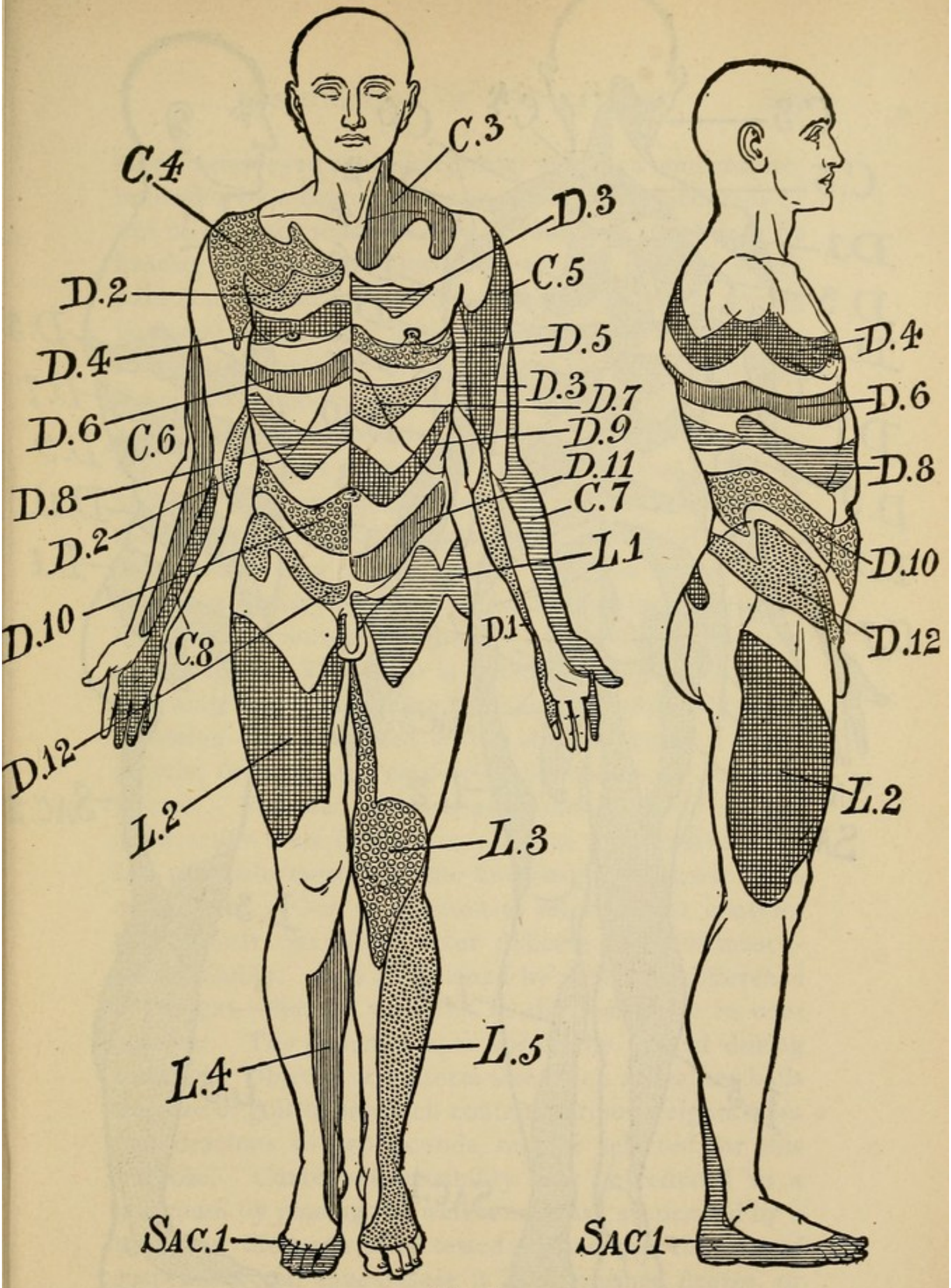

C.4
C.3
D.3
C.5
D.2
D.4
D.5
D.6
D.3
D.7
C.6
D.9
D.8
D.11
C.7
D.2
L.1
D.10
D1
C.8
D.12
L.2
L.3
L.4
L.5
SAC.1
D.4
D.6
D.8
D.10
D.12
L.2
SAC 1

FIG. 77.

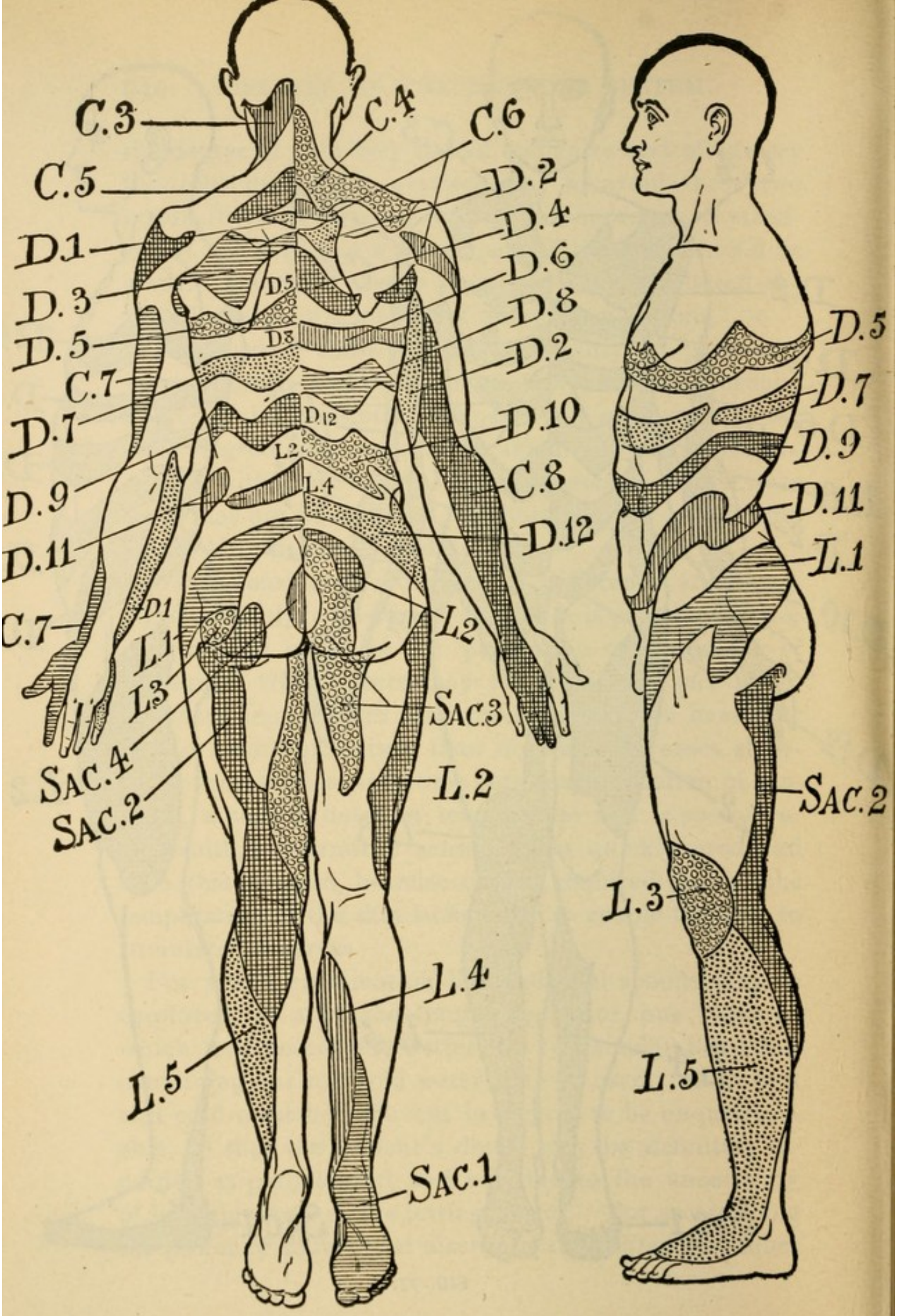

C.3
C.4
C.6
C.5
D.2
D.1
D.4
D.3
D.6
D.5
D.8
D.7
C.7
D.2
D.9
D.10
C.8
D.11
D.12
C.7
L.1
L.2
L.3
SAC.3
SAC.4
L.2
SAC.2
L.4
L.5
SAC.1
D.5
D.7
D.9
D.11
L.1
SAC.2
L.3
L.5

FIG. 78.

may be employed containing water of known temperature, indicated by a thermometer in each. The accuracy of test of the pain sense may be verified in a rough way by heating the hot-water test-tube to that point at which pain is produced. Sensibility to heat and cold should be tested separately at some time during the examination as the two sensations are subserved by different nerves. Some minute points appreciate only heat while others in a like manner appreciate cold only. Finally the two sensations may be affected in different degrees. Fig. 77 and Fig. 78 show approximately the sensory skin areas of the different spinal cord segments devised by Head.

Muscular sense includes more than one sensation; it embraces *muscle pain sense*. The afferent impulses originate from the muscle spindle which is excited by pressure and traction. The latter is tested by gripping the belly of a muscle and comparing the uncomfortable or painful sensation thus produced with a similar test in a sound muscle, or in general employing an imaginary standard of test. Postural sense is ascertained either at rest or in action from cutaneous, joint, or muscle structure, but probable most from the knowledge of muscle tone or tension. Our recognition of resistance to contraction affords one of the most delicate tests for muscular sensibility. It is ascertained by detecting difference of weight—that is, variation in the resistance to contraction. The patient's eyes should be closed during the trial. Objects of uniform size, such as leather balls the size of golf balls which contain various weights from two drachms to two pounds, may be selected for this purpose. Cutaneous sensibility may be reduced to a minimum by placing the weight in a bag suspended by a string on the parts to be tested; or increase the area of suspension and thus diffuse it in diminished degree, or

increase the weight and therefore the pressure in both area and degree so that the addition to be discovered shall bear only a small proportion to the total stimulation. The discrimination of weights by muscular sensibility is greater than by the skin; a difference of $\frac{1}{40}$ of the total weight may be appreciated by the former. The electrical test of the sensibility of the skin is not practical. The sensibility of muscle to the electric current can only be ascertained by eliminating that of the skin by cocaine anæsthesia.

A rough and practical but rather inaccurate test for muscular sensibility is the power of recognizing passive posture. If cutaneous sensibility is normal the parts handled must be grasped firmly and pressed on both sides so that the direction of pressure may not suggest the posture. The patient may be asked to place the limb of the other side in the same posture or indicate the direction of movement with the finger. Several examinations should be made to determine the chance of accidental error. Data of disorders of touch, pain, and temperature sensibility should be charted on outline figures made especially for this purpose. Written descriptions of such data are too confusing and obscure for rapid reference in a clinical study of the case. The stereognostic sense is best tested by requiring the patient to recognize with closed eyes the size, weight, and form of various familiar objects at hand such as keys, coins, etc.

Disorders of General Functions and Visceral Examination.

Aphasia.—If the speech defect is not an articulatory one it is called aphasia and it is necessary to apply certain tests for the determination of the exact difficulty. In routine examination one tests in turn the integrity of

the motor memory, visual memory, and auditory memory. To appreciate the condition of the motor speech centre and its association tracts, the examiner must note the patient's ability to speak voluntarily and his ability to repeat words spoken by the observer. To test visual speech one must test the patient's capacity to understand written or printed words; his ability to write spontaneously, his ability to write common things he sees, such as knife, button, etc., which he hears, or which he touches; his ability to copy writing; his ability to understand written directions as shown by his ability to execute them. To test the integrity of the auditory speech and its association the following are to be noted: the power of the patient to recall names of familiar objects seen, heard, touched, etc.; ability to understand ordinary speech, and his ability to recognize musical airs (whistling *Yankee Doodle*, for example).

Vaso-motor and Trophic Disorders should be noted as expressed in ulcers, nutrition of skin, hair, nails, and changes in color and heat of the skin in different parts of the body. Aside from a thorough visceral examination, such as is made in internal medicine, of the blood, urine, temperature, pulse, etc., the condition of the sphincters is to be especially noted in spinal affections. Incontinence of the bladder occurs under three different conditions, (*a*) in general lowered cerebral function, as in tumor cases where through neglect of the personal toilet a retention and final overflow is permitted, (*b*) paralytic incontinence through loss of sphincter control, as in spinal paralysis, and (*c*) incontinence due to mental defect, as seen in idiocy.

(The following composite case-record is given to illustrate the method of recording data obtained in examining the patient by the system detailed in Chapter VII.)

Ex. Sept. 13, 1904. John Doe, 34, m., German, U. S. 14 yrs.

Diagnosis: Ataxic Paraplegia.

Complains of: paralysis of the right side of body for 2 yrs.

F. H.—G. F., d. 72, senile dementia; G. M. l. and well, 82. F., d. at 43, alc. and tuber. M., 64, l. and well. First, boy, 36 (our pt.). Second, girl, 24, idiopathic, Gm., epilepsy from 8 yrs. Third, girl, 22, always anæmic, unmarried. Fourth, d. at 3 yrs., meningitis (?).

P. H.—Full term child; labor difficult and prolonged (14 hrs.); no instruments; breast fed; one or two convulsions at dentition. Measles at 3; scarlet fever at 6 with rt. otitis media. Puberty normal; school until 14; fairly competent journeyman tailor; always lived in New York; married at 22; 3 child., aged 12, 10, and 6, all l. and well; wife no misc.; pt. has used spirits and tobacco moderately since 20 yrs. old; gonorrhœa at 18; syphilis denied, but exposure admitted at 20–22; typhoid at 32; constipated 8 yrs.

(From Case of Cerebellar Tumor.)

P. I.—One year ago (Oct., 1904), dull paroxysmal headaches appeared suddenly without cause, most localized over right occiput; afternoons at first, then (3 mos. after) constant. Scalp tender to mild pressure; pain from headache radiated into right neck and shoulder muscles, marked stiffness and rigidity of the same; vomiting on rising in the morning almost daily during last 8 mos.; for 9 mos. a very unsteady gait; diplopia for 10 mos. at times corrected by prisms; some failure of eyesight past 4 mos.

P. C.—Well built, muscular, blond, Swede. Looks his age (36); appears rather depressed and dazed; walks with uncertain, reeling gait; tendency to walk to the right; eyes to floor; quite antæmic.

Cranial Nerves:

Motor and Sensory and Special Sense.

1.—Detects an odor in musk and peppermint, but does not differentiate.

Chronic catarrhal rhinitis for 5 yrs.

2.—*Rv.* $\frac{20}{20}$ *Lv.* ^{8} .

Vis. fs.—normal.

L. O. D. 2 d. swelling; few retinal hemorrhages.

3.—*3, 4, and 6 Rt. ext. rectus weak, sec. deviation, erroneous project. 2″; diplopia intermit., corrected by prisms.*

(From a Case of Trigeminal Neuralgia.)

5.—*Painful (sensitive to touch) in entire skin area (see chart); exquisite radiating pains on pressure at first and second foraminal openings; vaso-motor paresis marked; no motor changes; no taste changes.*

(From Case of Bell's Palsy.)

7.—*Rt. normal.*

Lt. entire paralysis.

Complete R. D. Bell's in type.

8.—*Rt. = fork at 3″. Lt. = bone conduction only (labyrinthian disease).*

(From Case of Bulbar Palsy.)

9.—*Liquids regurgitated through nose.*

(From Case of Tumor in Pons.)

10.—Slight difficulty in completing act of swallowing.
Resp. normal.

11.—Rt. sterno-cleido mastoid weak.

12.—Increased height of tongue in retraction, weak in left cheek; protruded, but deviates to right; articulation indistinct; first act of swallowing slightly defective.

(From Case of Brachial Birth Palsy.)

Upper Extrem.
(*Motor*). *Scapula and Shoulder Joint.*

	R.	L.
Trapezius	*o*	*o*
Rhomboids	*o*	*o*
Levator ang. scap	*weak*	*o*
Serratus mag.	*slightly weak*	*o*
Deltoid	*no action, atrophy marked*	*o*
Supra spinatus	*paralyzed*	*o*
Infraspinatus	*paralyzed and atrophied*	*o*

Teres minor	*o*	*o*
Subscapularis	*o*	*o*
Latissimus dorsi	*o*	*o*
Pectoralis major	*slight contract*	*o*
Pect. minor	*o*	*o*
Teres major	*slight contract*	*o*

Muscles of Forearm and Hand.

Triceps	*o*	*o*
Br. anticus	*weak*	*o*
Biceps	*marked atrophy, very weak*	*o*
Sup. longus	*o*	*o*
Sup. brevis	*no action*	*o*
Pronators	*o*	*o*
Fl. of wrist	*o*	*o*
Ext. of wrist	*weak*	*o*
Fl. of fingers	*contract*	*o*
Ext. of fingers	*o*	*o*

Summary.—Weakness or paralysis and atrophy in supra- and infra-spinatus, deltoid, biceps, br. anticus, supinator brevis (Erb's palsy distribution).

Scapula and whole arm dwarfed; internal rotation of humerus; contracture-deformity at shoulder joint, elbow, and wrist. Internal rotators of rt. humerus in negative contract; may be partly overcome. Flexors of wrist in same condition of contracture.

Comparative Measurements.

	R.	L.
Dynam	33″	60″
Length of arm	21″	23″
Length of humerus	11¾″	12½″
Cir. of arm	10″	10¾″
Cir. of forearm	10¼″	10½″
Cir. of wrist	7″	7¾″

Measurements show atrophy and shortening largely in rt. shoulder and arm.

Trunk and Lower Ext.—Normal musculature in all movements; no atrophies.

(From a Case of Right Hemiplegia of Two Weeks' Duration.)

REFLEXES

Superficial.

	R.	*L.*
Conjunc.	*absent*	*o*
Corneal	*diminished*	*o*
Cilio-spinal	*weak*	*o*
Scapular	*absent*	*o*
Abdom	"	*o*
Epigastric	"	*o*
Cremasteric	"	*o*
Plantar	*weak*	*o*

Deep.

Pupillary	*o*	*o*
Trigem.-facial	*o*	*o*
Jaw-jerk	*o*	*o*

Pectoral	*exag*	*o*
Triceps	"	*o*
Biceps	"	*o*
Supinator	"	*o*
Ulnar	"	*o*
Knee-jerks	"	*o*
Achilles jerk	"	*o*
Front tap	"	*o*
Babinski	*present*	*o*

(From Case of Ant.-Polio. M.)

Elect. Ex.—Rt. Ant. tibial = R. D. partial.
Rt. Ext. peroneal group = R. D. complete.
Sensory Ex.—Touch.
Pain.
Temp.
Muscle sense.
Muscle pain sense.

(From Case of Unilateral Crush of Cord D. *11′*. (See Chart, pg. 243, fig. *48*? for manner of charting sensory data.)

On side of lesion: *Skin hyperæsthesia D.* 11″.
Muscle sense impaired.
Temp. slightly raised D. 11″.

On side opposite to lesion: *Loss of sensibility D. 11″.*
Muscle sense normal.
Temp. normal.

(From a Case of Right Hemiplegia in a Woman.)

Disorder of General Function (Organ. Reflex); Visceral Ex.:
Partial motor aphasia.
Repeats words slowly and imperfectly.
Asthmatic; temp. 99° F., pulse 74, normal.
Rectal and vaginal ex. neg.
Mitral regurg.; severe arterial sclerosis.

Urine normal; blood not exam.
Weight, 93 lbs.; height, 5 ft. 6 in.
Vaso-motor and trophic disorders ;
Ulcer on rt. buttock (pt. bed-ridden).
Skin dry and scaly, hair and nails brittle on rt.
Blueness and coldness of exts. on rt. side.

The first page of the history blank should be reserved for a short synopsis of the chief symptoms upon which the diagnosis rests. It may be arranged as an epitome of the complete record detailed here at length.

CHAPTER VIII.

ILLUSTRATIONS OF DIAGNOSIS.

THE following illustrative cases are placed here with a view to showing how the methods of diagnosis are employed in actual practice. The cases are for the most part typical, and present few diagnostic difficulties but some of them deviate considerably from the common types of disease, and illustrate some of the difficulties in diagnosis that so frequently arise.

1. A man fell headlong from a scaffold, a distance of twelve feet, and at once became paraplegic. Examination showed the sixth cervical vertebra to be somewhat displaced. Below the arms there was complete motor paralysis ; in the arms there was complete paralysis of the intrinsic muscles of the hands, flexors and extensors of the wrists, and triceps, and partial paralysis of the biceps, supinators, and pronators. There was loss of sensation to pain or touch below a V-shaped line across the upper part of the chest, and, roughly speaking, in the inner half of the arms, forearms, and hands. It was inferred from the distribution both of the motor and the sensory loss, that there was a destructive lesion of the seventh and eighth cervical segments of the spinal cord (see pp. 20 and 249). Above the line of sensory loss was an area of hyperalgesia, which extended to the lower part of the neck. This indicated that the fifth and sixth segments of the cord were partially damaged. From the completeness of the motor and sensory loss in the parts below the level of the lesion, it was inferred that the cord was destroyed in a large part of its transverse extent at the level of damage. The examination of the reflexes confirmed this opinion, for the knee-jerks, cremasteric, abdominal, and plantar reflexes were abolished, the knee-jerks being thus lost in cervical injuries only in cases of severe damage to the cord. The

bladder and rectum were emptied automatically, which showed that the reflex centres of the cord for the bladder and rectum were no longer under cerebral control.

The diagnosis of the nature of the lesion in this case presented no difficulties. The damage to the cord was plainly such as could be produced only by the crushing effect of displacement of bone. The fact that there was only slight external evidence of displacement did not render this view any less probable, because it is common in cases of fracture-dislocation of the cervical spine for the displaced vertebra to spring back at least partially into place.

The patient died in less than two days after the accident, and the autopsy confirmed the diagnosis. There was a dislocation forward of the sixth upon the seventh cervical vertebra, the seventh and eighth cervical segments of the cord were severely crushed, and there were indications of slighter damage (chiefly hemorrhage into the central canal) to the sixth and fifth segments.

2. A young man, aged twenty-three, and of unusually strong build, was suffering from a considerable degree of paralysis in the legs and slighter paralysis in the arms when he first came under observation. Examination showed that the loss of power in the legs involved chiefly the anterior and posterior tibial groups of muscles and the extensors of the knee, the adductors and abductors of the thigh and the flexors of the knee being involved in less degree. In the upper extremity the loss of power was almost confined to the muscles of the forearm and hand, the extensors of the wrist being especially weak. The paralyzed muscles, both in the upper and lower extremities, were flabby and wasted, and showed the RD, the wasting and the changes in irritability being most pronounced in the muscles that were weakest. Sensibility to touch and pain was slightly diminished below the knees. Elsewhere in the legs there was a marked degree of hyperalgesia. The anterior crural nerves were exceedingly sensitive to pressure. The muscles of the legs were very tender to firm pressure. From time to time there was considerable spontaneous pain of a shooting character in the legs. In the upper extremities there were no disturbances of cutaneous sensibility, but the forearm muscles were tender to pressure, and there was some spontaneous pain in the arms. The knee-jerks were abolished. The bladder and rectum were under perfect control. The patient's mind was clear. The temperature was normal. The paraplegic distribution of the symptoms in this case immediately suggested disease of the spinal cord or peripheral nerves.

Spinal cord disease was, however, quickly excluded. Slow compression of the cervical cord might cause partial loss of power in the upper and lower extremities, and disturbances of sensibility, but could not be held responsible for the muscular atrophy, tenderness of the muscles, and loss of knee-jerk observed in this case. A lesion in the lumbar cord might account for the loss of power, sensory disturbances, wasting, and loss of knee-jerk, but such a lesion would probably involve the functions of the bladder and rectum, and would certainly not explain the symptoms in the upper extremities. In short, no single lesion of the spinal cord would be adequate to explain all the symptoms here present, and the supposition of multiple lesions of the cord was not to be seriously considered until more likely explanations had been sought. On the other hand, the assumption that the symptoms were due to a multiple affection of the peripheral nerves, afforded an explanation of the phenomena present that was thoroughly satisfactory. The paralysis, atrophy, RD, sensory disturbances, loss of knee-jerk, and preservation of the functions of the bladder and rectum could be thus explained. That the process in the nerves was a neuritis, was shown by the tenderness of nerve-trunks, the pain, the hyperalgesia, and the tenderness of the muscles. The diagnosis, then, as based on the objective features of the case, was multiple neuritis involving mixed nerves of both upper and both lower extremities. Certain features of the case, namely the slight degree of anæsthesia, the prominence of irritative sensory symptoms, as pain and hyperalgesia, and the tenderness of the muscles, suggested the dependence of the multiple neuritis on alcohol. This inference was borne out by the history. It was learned that for several years the patient had been in the habit of using alcoholic drinks very freely, though never to the extent of producing intoxication. He drank chiefly beer, the quantity averaging from fifteen to twenty glasses a day. Until eight weeks before he came under our observation he was in good health. Then he began to suffer from numbness and pains in the legs, to which was soon added a gradual but rather rapidly progressive paralysis, first of the lower then of the upper extremities. Thus the history confirmed the diagnosis of alcoholic multiple neuritis. The ultimate recovery of the patient was another evidence of the correctness of the diagnosis.

3. A boy, aged eighteen, complained of weakness in both lower extremities. It was immediately observed that his gait was peculiar. Each leg was brought forward in turn with considerable effort, and

was observed to be rigid and extended. Examination showed that the loss of power in the legs was nowhere considerable ; the weakness was most marked in the flexors of the thighs, knees, and ankles. The extensors of the knees showed no appreciable loss of power. In the upper extremities power was normal. The muscles of the legs were the seat of extensor spasm, which was especially marked during movement. The muscles showed no atrophy or loss of tone but were, on the contrary, well developed and firm. The electrical reactions were normal. The knee-jerks were greatly exaggerated on either side and an ankle clonus was readily obtainable. The functions of the bladder and rectum were normally discharged.

It was inferred from this combination of weakness and spasm in the legs that the case was one of involvement of the pyramidal tracts of the cord. That the lesion was confined to these tracts was made probable by the entire absence of sensory and trophic symptoms. It was learned from the patient that the spasm and weakness had come on gradually during the two preceding years, the symptoms having been at first more marked in the right than in the left leg. The apparent limitation of the process to the pyramidal tracts and the gradual and slow development of the symptoms left little doubt that the morbid process was degenerative in nature. The case was, therefore, to be regarded as one of spastic paraplegia from sclerosis of the lateral pyramidal tracts. But there remained the question, is this lateral sclerosis primary or secondary? Usually cases of lateral sclerosis are secondary to disease of the brain or spinal cord. There were no evidences whatever of a cord lesion to which a lateral sclerosis might be secondary—no symptoms suggesting myelitis, or tumor or compression of the cord from any cause. There were no signs of vertebral disease. An endeavor was next made to discover signs of brain disease, old or recent. It was observed that the patient was mentally dull, and had some hesitation in speech, but there were no other cerebral symptoms. The skull was well developed and symmetrical. There was no strabismus or nystagmus. The patient was free from headache. Inquiry into the history of the case likewise failed to give evidence of cerebral disease. There was no history of traumatism during birth or of convulsive seizures during infancy or childhood, and, as already stated, the symptoms of spinal cord disease did not begin until two years before the patient came under observation. As no evidences could be obtained of brain or cord disease to which degeneration of the lateral tracts might be secondary, the case was regarded as one of primary

lateral sclerosis. It must be admitted, however, that this diagnosis was not perfectly satisfactory. The mental retardation and speech disturbance remained unaccounted for ; and as cases of unquestionable primary lateral sclerosis are of very rare occurrence as compared with cases of secondary lateral sclerosis, the possibility that the process was in this case secondary to an old and obscure cerebral lesion could not be entirely excluded.

4. A boy, aged ten, slipped upon the ice and fell to the ground in the sitting posture. He walked to his home, which was some distance away, and after several hours began to complain of weakness, numbness and pain in his legs. Examination about twenty-four hours after the fall showed complete loss of power below the knees and considerable weakness of the thigh and hip muscles. Sensation was unimpaired but for numbness and tingling in the legs. The knee-jerks were abolished, and there was incontinence of urine and fæces. There was a slight elevation in temperature. In the course of a few days the muscles of the thighs and hips lost all power, and the tactile and pain senses were abolished below a transverse line a short distance above the pubes. The plantar and cremasteric reflexes were lost at this time, but the abdominal reflex was preserved. There was still a slight rise in temperature.

The diagnosis of the position of the lesion in this case presented no difficulties. The initial loss of power in the legs, the abolition of the knee-jerks, and the paralysis of the sphincters indicated clearly that the structures in the lower part of the lumbar enlargement which subserve motion and the visceral and tendon reflexes were the seat of disease. The initial preservation of sensibility in the legs made it probable that the damage was limited chiefly, if not entirely, to the motor structures of the cord. The subsequent loss of sensibility in the legs and the ascent of the motor paralysis to the flexors and extensors of the thigh led to the inference that there had been an increase both in the transverse and vertical extent of the lesion. The completeness of the motor and sensory loss made it probable that the lesion involved the greater part of the transverse extent of the cord. The vertical extent of the lesion was inferred from the distribution of the paralysis and the state of the various reflexes. The paralysis of the rectal sphincter, the loss of sensibility over the perineum, and the loss of plantar reflex showed that the cord was involved as low as its sacral segments. The loss of cremasteric reflex showed that the cord was involved as high at least as the first or second lumbar segments.

That the lesion did not extend much higher than this was inferred from the preservation of the abdominal reflex, which depends on the integrity of the dorsal nerves from the seventh to the twelfth, and also from the transition to normal sensibility a short distance above the pubes.

The diagnosis of the nature of the lesion was less simple. The extension and progress of the motor and sensory symptoms and the presence of fever made it certain that the lesion soon became inflammatory in character, whatever may have been its original character. It was believed by some that the initial lesion was hemorrhage into the gray matter of the cord, but the facts that the symptoms did not come on for several hours after the fall, and that they developed rapidly but not *suddenly*, made it more probable that the lesion was from the first an inflammation. The diagnosis of lumbar myelitis was confirmed, if such confirmation was needed, by the subsequent course of the case. The paralyzed muscles atrophied greatly, and showed the RD, bed-sores formed, and the patient at length died of exhaustion.

5. A man, aged thirty-two, presented himself with inco-ordination and weakness in the lower extremities. Both these symptoms were apparent in his gait. As the patient progressed unsteadily across the floor, his feet were lifted high and brought to the ground with an irregular flap. A slight degree of dropping of the feet from weakness of the anterior tibial groups of muscles was also observable. Examination showed slight inco-ordination of the upper extremities, but no weakness. In the legs there was a high degree of inco-ordination, and the patient was unable to stand with his eyes closed. The loss of power in the legs appeared to be confined to the anterior tibial and peroneal groups of muscles, but the patient said he felt weak also in the knees. The anterior tibial and peroneal muscles showed a slight degree of atrophy and a partial form of RD. The posterior tibial group showed partial RD, but no appreciable weakness or wasting. Sensibility was normal except on the soles and backs of the feet, where there was slight anæsthesia and analgesia. There were paræsthesiæ in other parts of the legs, and from time to time considerable sharp pain. The knee-jerks were abolished on both sides.

By far the most obtrusive symptom in this case was the inco-ordination. A high degree of inco-ordination may be of cerebral, spinal, or peripheral origin. In this case there was nothing to suggest

brain disease. The associated loss of power in the legs, the atrophy, the RD, the disturbances of sensibility, and the loss of knee-jerks, all indicated that the lesion was one of the cord or peripheral nerves. A lesion of the posterior columns of the cord and posterior root-zones would explain the presence of inco-ordination, pains in the legs, slight anæsthesia, and loss of knee-jerks. A lesion of these structures almost invariably depends on a degenerative process, and then gives rise to the clinical picture of locomotor ataxia. But the diagnosis of locomotor ataxia did not afford a full explanation of the symptoms, in this case; it did not explain the presence of weakness, wasting, and altered electrical reactions. These symptoms might depend either on a limited lesion of the ganglion cells of the anterior horns in the middle of the lumbar enlargement, or on disease of the motor fibres originating in these structures and forming part of the peripheral nerves. The former condition is not an associated lesion of locomotor ataxia; the latter frequently is. All the symptoms, then, might be explained on the supposition that we had to deal with a case of locomotor ataxia with associated changes (neuritis) in certain peripheral nerves. But this is not the only condition that would account for the symptoms. As intimated above, the inco-ordination present might depend on a lesion limited exclusively to the peripheral nerves. Such a lesion, involving both the motor and sensory fibres of mixed nerves, would explain satisfactorily not only the inco-ordination but also the weakness, atrophy, RD, loss of knee-jerks, and sensory symptoms. In short, the symptoms of the case might be satisfactorily explained either by a locomotor ataxia with associated neuritis or by a multiple neuritis alone. The distinction between these conditions could not be made from the facts gathered by the examination of the patient. It therefore became necessary to see what bearing the history of the case might have upon the diagnosis. It was learned that six months before the date of examination the patient was in fair general health. He then began to lose flesh and suffered from numbness, tingling, and sharp pains (never very severe) in the legs. In a few months he grew unsteady in walking and developed some weakness in the legs. Both these symptoms increased rather rapidly. Ten years ago the patient had a hard chancre and during the two years previous to examination had used alcohol in great excess. The history of excessive use of alcohol before the onset of the symptoms rendered an alcoholic neuritis a probable condition; the clear history of a hard chancre would explain the presence of loco-

motor ataxia. The facts of etiology, therefore, did not aid in solving the diagnostic problem. But the following facts all pointed to the presence of alcoholic neuritis rather than locomotor ataxia: the relative youth of the patient (thirty-two years), the development of all the symptoms within so short a time as six months, the absence of very severe fulgurating pains, and the absence of all ocular symptoms —especially the absence of the Argyll-Robertson pupil.

This diagnosis was made with some caution, but was considered highly probable by several skilled physicians. It has not been borne out by time. A year has elapsed since the patient first came under notice, but there has been little or no improvement in the symptoms and the pains have been more severe than before. The diagnosis,[1] therefore, still remains uncertain.

6. A man, aged thirty-three, complained of pain in his right arm. Inspection showed that the extensors of the wrist were paralyzed and gave rise to pronounced wrist-drop. The flexors of the wrist showed some apparent loss of power owing to the dropping of the wrist, but there was no actual paralysis. The muscles on the back of the forearm were considerably atrophied and showed the RD. Sensibility was unimpaired. The limitation of paralysis and degenerative atrophy to the extensors of the wrist on one side immediately suggested the dependence of these symptoms on a lesion of the musculo-spiral nerve. The power of the supinator longus muscle was then tested, and it was found to be somewhat diminished. It also showed a diminution in faradic irritability, and equal and somewhat sluggish polar reactions to the galvanic current. These observations tended to confirm the diagnosis of musculo-spiral paralysis. The history of the case was next considered with a view to its bearing on this diagnosis. Careful inquiry was made for some cause of pressure on the musculo-spiral nerve, but none could be found. It was learned that the paralysis had come on in the course of a few days, and that it had now lasted several weeks. It was also learned that for some weeks before the paralysis appeared there had been pains in *both* forearms. The patient was a painter and had recently been much exposed to lead in "flatting." These facts strongly suggested the dependence of the paralysis on lead, but the apparently unilateral distribution of the palsy, together with the absence of a lead line

[1] This case illustrates well the difficulties of diagnosis. The correct diagnosis was of much importance in this case, as the prognosis is very different in the two forms of disease.

or a history of colic, rendered lead palsy improbable. A more careful examination was now made of the left arm. Slight weakness was soon detected in the extensors of the wrist and an electrical examination showed a decided diminution of faradic irritability in these muscles. The discovery of involvement of the extensors of the left arm, slight though it was, entirely altered the aspect of the case. The bilateral extensor paralysis, preceded by slight sensory symptoms and occurring in a painter, made it evident that the case was one of lead neuritis. The unequal involvement of the muscles of the two sides is an unusual feature of lead paralysis. The slight loss of power in the supinator longus of the right side did not militate against the diagnosis, for such partial involvement of the supinator is not rare in lead palsy.

7. A boy, aged ten years, came under my observation on account of the frequently repeated epileptiform seizures to which he was subject. Inspection showed the patient to be much below the average size of boys of his own age—his twin brother was about four inches taller and correspondingly larger. The walk of the patient was exceedingly unsteady, and showed considerable weakness in the lower extremities; there was a high grade of mental weakness (imbecility), and only monosyllabic speech. Examination showed all four extremities to be small and imperfectly developed. The weakness in the legs was not confined to a particular group of muscles, but involved the leg as a whole. The extensors, however, were very slightly affected. The muscles, though small, showed no indication of atrophy. Their tone was normal or even greater than normal, there being slight extensor spasm at times. The electrical reactions were normal. Both knee-jerks were much exaggerated. The skin reflexes were all active. Ankle clonus could not be obtained. In the upper extremity there was a well marked intention tremor. Sensibility was everywhere normal. The head was abnormally small and the palate was high-arched. The prominent features of this case were the mental weakness, the evidences of arrest of development in the extremities and in the cranium, the occurrence of epileptiform attacks, and the weakness and spasm in the legs. All of these conditions indicated the presence of a cerebral lesion. Certain of the symptoms, as weakness and spasm in the legs, occurring by themselves would suggest spinal cord disease rather than a cerebral process, but with the associations above mentioned these symptoms indicate a cerebral and not a spinal cord lesion. The bilateral distribution of the symptoms and their involve-

ment of the arms as well as the legs, indicated that the motor path was implicated in a considerable part of its extent in both hemispheres of the brain. The signs of retarded development, both mental and physical, made it plain that the morbid process was one of long duration, in fact that it must have commenced in infancy or early childhood. Cerebral palsies at this time of life are either congenital or acquired, the congenital form being generally dependent upon injury during birth, the acquired form depending on a variety of different conditions in different cases (see p. 367). It was impossible to say with certainty in which of these catagories the case belonged, without taking into consideration the history of the case. But the presence of a high grade of mental defect, the physical evidences of considerable retardation of growth, and the bilateral and symmetrical distribution of the symptoms suggested that the condition was a birth palsy. This view was corroborated by the history of the case. It was learned that the patient was the first child born in a twin labor, that the mother was at that time a primipara, that the second stage of labor lasted nearly twenty hours, and that the child had several general convulsions during the hour following delivery. These convulsions were frequently repeated during the first months of life and had continued, with decreasing frequency, ever since. These facts were a perfect confirmation of the diagnosis, and removed any doubts as to the nature of the lesion, which could have been nothing else than a meningeal hemorrhage, extending over both convexities, resulting eventually in a sclerosis and atrophy of the parts of the cortex and subcortical substance involved and secondary degeneration of the pyramidal tracts.

8. A child, three years of age, came under observation suffering from paralysis in both lower extremities and partial loss of power in the right arm. There were typical athetoid movements in the fingers of the right hand, with temporary flexor spasm, moderate atrophy of the forearm muscles, and slight loss of power. The faradic irritability of the forearm muscles was slightly diminished, but there was no RD in any muscles of the upper extremity. Sensibility was nowhere affected. These conditions in the upper extremity at once suggested a cerebral palsy, and this idea was strengthened by observation that the patient was backward in intelligence and speech. The lower extremities were next examined to see whether their condition corroborated the diagnosis of a cerebral palsy. There was a high grade of paralysis of all the muscles of the legs except the flexors of

the hips, the paralyzed muscles were greatly atrophied and flabby, and showed the RD. Below the knees the paralysis was absolute and the feet were in equinus. The knee-jerks were lost. The sphincters were unaffected. Sensibility was everywhere normal in the lower extremities. The extremities were cold and the color of the skin showed defective circulation.

The presence of a high grade of paralysis and atrophy in the legs, the reaction of degeneration in the paralyzed muscles, and the loss of knee-jerks all pointed to the existence of a lesion of the lumbar enlargement, involving the ganglion-cells of the anterior horns. That the lesion was confined to these motor structures was shown by the fact that only the trophic, motor, and reflex functions of the cord were impaired, the various forms of sensibility being intact. The same case therefore presented unmistakable evidences of two quite distinct lesions, one involving the motor path in the brain, probably in the cortex or subcortical region, the other involving motor ganglion-cells in the lumbar enlargement of the cord. The cord symptoms were of the kind observed in an advanced stage of acute poliomyelitis. Further indications as to the nature of the lesions in the brain and cord were derived from a consideration of the onset of the symptoms. It was learned from the mother of the patient that, one year before the child came under observation, it became restless and feverish for several days, and at the end of this became delirious, had high fever, and vomited repeatedly. During this latter period of acute illness there were repeated clonic spasms in the right arm and leg, strabismus, and ptosis. The one-sided convulsions lasted about two days and then the rigid arm was noticed to be without power. The mother was uncertain as to the state of the right leg at this time, but believes it was little or not at all weak. Five days after the onset of the convulsions it was observed that there was complete loss of power in both legs. There was gradual recovery of power in the right arm, but little or no improvement in power in the legs, which soon wasted. With this history there could be little doubt that both the cerebral and the spinal cord lesion were dependent on similar morbid processes, infectious in origin, and giving rise in the one case to an acute cortical lesion in the left motor region (polio-encephalitis, thrombosis of cerebral veins?), in the other to an acute anterior poliomyelitis. Cases similar to this one are very rare, but are instructive as illustrating the relationship in etiology between some cases of acquired infantile cerebral palsy and anterior poliomyelitis.

9. A girl, aged nine, was suffering from partial paralysis of the right arm and right leg when she first came under observation. In the leg the paralysis was of slight degree and involved about equally all the muscles of the limb. In the arm the degree of weakness was greater, and there was a well marked contracture of the flexors of the forearm. The face was uninvolved. There was no atrophy of the affected muscles, and sensibility was everywhere normal. The hemiplegic distribution of the weakness in this case suggested the dependence of the paralysis on a cerebral lesion. Some support was lent this view by the fact that there was internal strabismus of the left eye, such as often remains after an infantile cerebral palsy. Moreover, the knee-jerk was greatly exaggerated on the paralytic side and unchanged on the normal side, and this indicated a secondary degeneration of the pyramidal tracts on the paralytic side, such as usually follows a cerebral palsy. But, on the other hand, certain facts about the case failed to confirm the diagnosis of a cerebral palsy, and even tended to disprove it. First there was no loss whatever in mental power. The patient was, on the contrary, highly intelligent and well developed mentally. Such entire immunity from mental defect is unusual in infantile cerebral palsies which give rise to a considerable degree of permanent paralysis. Secondly, there were no evidences of arrest of development in the affected limbs, and such evidences are almost always present in cerebral palsies of children if the paralysis has endured a considerable length of time.

An inquiry into the history of the case soon did away with any doubts as to the position and nature of the lesion. It was learned that the child had been perfectly well until she was seven years of age (or two years before), and that she then developed weakness in both legs. This weakness slowly increased in the legs and extended to the arms, so that at the end of six months there was complete paralysis of motion below the neck. There was also entire loss of sensation below the neck. The muscles of the neck had meanwhile grown weak, and the patient was unable to keep the chin from resting on the neck. She was kept quiet in the recumbent position for many months, and gradually the anæsthesia wore away. Then there was a gradual return of power in the legs and left arm, but the right arm continued weak. In course of time the muscles of the neck regained full power. The paraplegia could have been due only to disease of the upper cervical region of the cord, and the gradual onset of the

symptoms and recovery after a period of recumbency left no doubt that the lesion was a compression of the cord from caries of the upper cervical vertebræ. At the time when the patient came under observation there were no evidences whatever of disease of the cervical vertebræ, and the conditions bore no little resemblance to those observed in cerebral palsies. The strabismus, it was learned, was present many years before the development of nervous symptoms and therefore had nothing to do with these symptoms. It is doubtful whether a correct diagnosis could have been made in this case without some knowledge of the previous history.

10. A man, aged fifty-four, complained for several days of headache and slight numbness in the left extremities, and soon after arising one morning became rapidly hemiplegic. The loss of power involved the left leg, left arm, and left side of the face in its lower part. There was no loss of consciousness at the time of the onset, but the patient felt giddy and had considerable headache. When examined, about twenty-four hours after the onset of the hemiplegia, the patient was in a state of stupor from which he could be partially aroused. Temperature, 100° Fah.; pulse, 80; radial arteries, atheromatous; respiration, 20, regular. There was complete left hemiplegia, and the arm and leg muscles were quite relaxed. Sensibility was diminished in the extremities in their distal portions. The knee-jerk was greatly diminished on the left side, normal on the right. The pupils were equal and moderately large. There was no spasm or other irritative symptoms.

The diagnosis of the position of the lesion was not difficult. Complete left-sided hemiplegia might result from a lesion in the right motor path anywhere between the motor cortex and the upper half of the pons. The absence of spasm made it exceedingly improbable that the lesion was cortical or subcortical, and there was nothing to indicate a lesion of the crus. It was therefore highly probable that the lesion was in the right internal capsule—the seat of damage in most cases of sudden hemiplegia. The diagnosis of the nature of the lesion was less easy. A sudden hemiplegia usually depends on hemorrhage or acute softening. Acute softening is generally due to thrombosis from atheroma or from syphilitic endarteritis, or to embolism. Embolism was excluded because there were no evidences of heart disease, and because there were distinct premonitory symptoms before the onset of the hemiplegia. Thrombosis from atheroma was considered improbable on account of the

age of the patient. Either cerebral hemorrhage or thrombosis from syphilitic endarteritis would explain the symptoms, and between these two conditions it was difficult to decide. The facts which favored the supposition of hemorrhage were the age of the patient and the presence of atheroma of the accessible arteries. The facts favoring syphilitic endarteritis were the presence of distinct premonitory symptoms (headache, numbness, slight weakness) for several days before the onset, and the absence of loss of consciousness. These facts also weighed against the diagnosis of hemorrhage, for in hemorrhage true premonitory symptoms are rare, and a capsular hemorrhage sufficiently large to produce complete hemiplegia would almost certainly have given rise to loss of consciousness. Although no history of syphilis could be obtained, several tubercles were observed on the upper part of the chest, which had the appearance of gummata. On the whole, therefore, the facts of the case pointed to acute softening from syphilitic endarteritis rather than to hemorrhage. The patient was put on large doses of iodide of potassium, and in time made a good recovery. In about two weeks from the time of the onset he had largely recovered power in the hemiplegic parts and the headache had grown much less severe. Eventually gross power was entirely regained, but there remained some awkwardness in executing the finer movements of the fingers. Slight attacks of giddiness occurred from time to time after recovery of power, and the knee-jerk became greatly exaggerated on the left side. There also developed an ankle clonus on this side. It was learned many months after the attack that the patient had had a chancre several years before. About eighteen months after the hemiplegia the wife of the patient came under observation for an abundant syphilitic eruption over both legs. These facts all confirmed the diagnosis of the nature of the lesion underlying the hemiplegia.

11. A woman, aged thirty-four, was admitted to the hospital in a condition of stupor and with slight elevation of temperature. About two inches above the root of the nose and just a little to the left of the median line was a suppurating incised wound. Examination showed that the frontal bone was fractured just beneath the wound. The patient could be roused for a moment from her stupor, and at times uttered partially intelligible monosyllables when so aroused. The eyes had a fixed, staring expression. At times the left hand was placed on the head while the patient groaned with pain. The

temperature was 101°. There was nothing noteworthy about the pulse or respiration. The pupils were equal, of moderate size, and reacted to light. There was incomplete ptosis of the left lid. The right arm was almost completely paralyzed; the right leg was less completely paralyzed. The right arm was constantly rigid; no spasm elsewhere. There was general hyperæsthesia. On the right side there was great exaggeration of the knee-jerk and violent and prolonged ankle clonus. On the left side the knee-jerk was increased, but ankle clonus could be obtained only with difficulty. There was entire loss of control over the sphincters.

The presence of loss of power in the right arm and partial loss of power in the right leg, with exception of the face, made it certain that there was a lesion in the left motor path. A lesion involving the cortex might give rise to such a partial hemiplegia but would produce more decided irritative symptoms than were here present. A lesion in the internal capsule or in the motor path below the internal capsule would not be apt to exempt the face unless the lesion were very small. But a lesion between the cortex and the internal capsule might be of considerable size and nevertheless involve chiefly the path to the arm of the opposite side, and such a lesion would not be apt to cause clonic spasm. It was upon the whole probable, therefore, that the lesion was one of the subcortical white substance of the left motor path. The diagnosis of the nature of the lesion could be determined with less certainty. The limitation of the paralysis to the parts above-mentioned and the completeness of the motor loss made it probable that the lesion was destructive in character. Such a lesion might be a vascular lesion, or an inflammatory lesion, or a new growth. But it was known that the symptoms were not of sudden onset, and hence a vascular lesion was out of the question. The presence of fever made it likely that the lesion was inflammatory in nature. It could scarcely be meningitis, for mere meningitis would not give rise to such complete and definitely limited paralysis, and would cause more decided irritative symptoms than were here present.

An abscess, however, would be competent to cause a local paralysis, would not necessarily be accompanied with irritative symptoms and would almost certainly be attended with fever. The presence of a neglected compound fracture of the frontal bone offered a sufficient explanation for the occurrence of abscess.

The patient died in a few days without developing any new symptoms of importance. The autopsy showed the presence of an abscess

of the left frontal lobe, which contained from two to three ounces of pus. The abscess extended back to and somewhat into the white substance beneath the motor area.

12. A boy, aged ten, much emaciated and anæmic, was in a state of deepening coma when examined for the first time. He lay upon his back picking alternately at the bedclothes and at his lips. The pulse was 130, small, feeble, and irregular. Respiration was irregular (20–30 per minute). Temperature, 103½° Fah. There was conjugate deviation of the eyes to the left. The pupils were of moderate size, equal, and reacted to light. There was some rigidity of the muscles of the back and sides of the neck. The right arm and leg and the left arm were the seat of rigidity, which varied somewhat in degree from hour to hour. No distinct loss of power could be made out. The knee-jerk of the right side was exaggerated. The left knee-jerk could not be tested as the leg and hip-joint were immobilized on account of an exacerbation of the hip-joint disease from which the patient had suffered several years. Examination revealed the presence of a considerable grade of optic neuritis.

The most important localizing symptoms in this case were symptoms of an irritative nature—rigidity in the extremities, conjugate deviation of the eyes and rigidity of the muscles at the back and sides of the neck. The presence of these conditions indicated a diffuse meningeal process. The rigidity of the right limbs and the conjugate deviation of the eyes to the left were referred to irritation of different portions of the left and right motor regions respectively. The rigidity of the neck muscles was referred to irritation of structures at the base of the brain. It was inferred from the presence of the irritative symptoms just mentioned, from the presence of double optic neuritis and the considerable elevation in temperature, that the morbid process was inflammatory; in other words, a meningitis affecting both the base and convexity of the brain. The presence of hip-disease made it exceedingly probable that the process was tubercular in nature. No physical signs of lung disease could be detected. The presence of optic neuritis and of irregular respiration suggested that the process was tubercular, although both these conditions may be and are met with in other forms of meningitis.

The diagnosis of meningitis was confirmed by the history of the case. The patient had had hip-disease for several years. About ten days before the examination above recorded he had an exacerbation of pain in the hip, with increase of rigidity of the muscles about the

joint. During this the temperature rose to 105° and he complained of general headache. The temperature fell to normal and the headache stopped. About this time he vomited once without known gastric cause. Four days before the examination the temperature rose to 103° and the patient complained of intense headache. The temperature continued, ranging between 101° and 104°; there was repeated vomiting, and delirium developed. Two days before, there was widespread rigidity and temporary paralysis of the left arm and leg.

On the ninth day after the examination the patient died. At the autopsy was found tubercular meningitis of the base and convexity of the brain. Fresh miliary tubercles were present in both lungs.

13. A boy, aged twenty, complained for several months (probably five or six) of headache and of numbness and occasional "twitching" in the right leg. In time the right leg became weak and occasional twitchings occurred in the right arm, the fingers of which sometimes became numb. When examined for the first time the patient was in fair general health but suffered from severe and almost continuous headache, which was sometimes general, sometimes limited to the left side of the head. The right leg showed some loss of power, which was most marked in the muscles below the knee. There was no wasting of the muscles, and the electrical reactions were normal. The right knee-jerk was exaggerated, and an ankle clonus could be obtained on this side. Tactile sensibility was diminished on the foot and lower part of the leg. In the arm there was slight loss of power in the flexors of the wrist and fingers, as shown by diminution of the grasping power. From time to time, usually several times a day, there occurred convulsive twitchings in the right leg. At times these spasms were confined to the anterior and posterior tibial groups of muscles, at other times they extended to the muscles of the right hand and forearm and, more rarely, to the right side of the face. In some of these seizures in which the spasm was not limited to the leg the patient became unconscious for a few minutes. There was a temporary increase in weakness in the extremities after they had been the seat of spasm.

Intellect was unimpaired, but speech was somewhat disordered. There was usually a decided slowness in speech and an undue separation of syllables. At times the patient was unable to think of the names of familiar objects as paper, ink, knife, etc. Spoken and written words were perfectly understood. The use of objects was perfectly understood, and the patient was distressed at his inability

to recall and utter familiar words. The motor speech defect was regularly worse after spasm, whether this was or was not accompanied by loss of consciousness. At times the motor speech defect was very slight. Examination of the fundus of either side showed a high grade of optic neuritis. The temperature, pulse, and respiration of the patient were normal.

It was inferred from the hemiplegic distribution of the paralysis and spasm, from the disturbance of speech, and from the severity of the headache, that the patient was suffering from an intracranial process. The initial involvement of the right leg, and the gradual extension of the symptoms (weakness and spasm) to the right arm, made it exceedingly probable that the lesion involved the left motor tract where the paths for the leg and arm are farther separated than in the internal capsule or the parts below; in other words, that the lesion involved the motor tract somewhere above the internal capsule—in the motor cortex or the corona radiata. From the frequently repeated clonic spasm it was inferred that the lesion invaded the motor cortex or the white substance immediately beneath it. The extension of the paralysis and spasm made it probable that the morbid process began in the motor structures related to the leg and had subsequently extended to the arm. It was learned that the involvement of the face in spasm was a recent development, and this indicated that the morbid process was gradually invading the path for the face.

As to the nature of the lesion there could be little doubt. The gradual development of the symptoms during a period of several months made it certain that the morbid process was either a new growth or an inflammation. The inflammations which it was necessary to consider were chronic meningitis and abscess. Only a chronic syphilitic meningitis could give rise to well marked focal symptoms, and it was doubtful whether such decided focal symptoms as existed in this case could be explained by a purely meningitic process. No history of syphilis could, however, be obtained, and while syphilitic infection could not be positively excluded, there was reason to believe such infection very unlikely. An abscess occupying the subcortical motor region would account for all the focal symptoms present, and perhaps for the headache. But there were no causal indications of abscess—no ear or bone disease, no evidences of suppuration elsewhere, not even a history of injury to the head. Moreover, the absence of fever made abscess improbable.

On the other hand, everything learned from the examination and history of the case pointed to the presence of tumor. The gradual but distinct progress of well marked focal symptoms, especially paralysis and spasm, the severe and constant headache, the presence of a high grade of optic neuritis, the absence of fever—all these conditions favored the diagnosis of tumor.

The diagnosis of the nature of the tumor was difficult; could not, in fact, be made. The absence of a history of syphilis made a syphiloma unlikely. The good general condition of the patient, the absence of fever during several weeks' observation, and the absence of physical signs of tubercular lung disease made a solitary tubercle unlikely, but did not justify its positive exclusion. Of the remaining forms of tumors, glioma and sarcoma were the ones to be most seriously considered; but between these it was impossible to decide. The diagnosis of cerebral tumor was confirmed by autopsy. During the remaining six weeks of the patient's life the paralysis extended to the face and tongue, and a high grade of motor aphasia was developed. The number, intensity, and extent of the unilateral spasms increased, and the patient one day had an apoplectic seizure, with repeated general convulsions, in which he died. The autopsy revealed a large glioma involving the white matter, and, to a less extent, the cortex of the left hemisphere, in the motor region, and the adjacent portion of the frontal lobe. The tumor was highly vascular. In the middle of the mass was a recent hemorrhage of considerable size, which was undoubtedly the direct cause of the apoplectic seizure immediately preceding death.

14. A woman, aged forty-five, presented herself with weakness and loss of sensation iu her right arm. Examination showed that there was some loss of power in all the muscles of the arm, but no movements were entirely lost. There was no wasting of the limb, and all the muscles reacted normally to faradic and galvanic stimulation. There was loss of sensibility to pain and diminution in tactile sensibility over the entire arm as high as a sharply defined line over the acromion to the axilla. There were no trophic or vasomotor changes in the limb. The knee-jerks were active, but not exaggerated. The patient was well nourished and in good general health, barring irregular menstruation incidental to the climacteric.

The presence of weakness in the right arm without muscular atrophy and changes in the electrical irritability of the muscles made it at once evident that the monoplegia was of cerebral origin. The seg-

mental distribution of the motor and sensory loss, and the sharp limitation of the latter, made it exceedingly probable that these symptoms were dependent on disturbance of the functions of the cortex, and were due to functional and not to organic disease. The functional nature of the disturbance was furthermore indicated by the entire absence of all indications of organic cerebral disease. Interrogation of the patient elicited the fact that the arm trouble had developed a few weeks before, within a few minutes after the announcement of the sudden death of her brother. The dependence of the monoplegia upon emotional disturbance left no doubt that it was of purely functional (hysterical) nature. Applications of the faradic current to the weak arm caused an immediate improvement in grasping power and in tactile sensibility. It gave rise also to partial return of sensibility to pain in irregular areas over the arm. These facts were additional confirmation of the hysterical nature of the case.

15. A boy, aged ten, was picked up in the street in an unconscious condition and brought to the hospital. When seen a few minutes after he was having irregular movements of nystagmus and clonic spasms in the lower extremities. The seizures began regularly in the left leg and extended to the right leg, recurred every few minutes, and lasted from ten to sixty seconds. The spasm involved chiefly the anterior and posterior tibial groups of muscles. The limbs were quite relaxed after each paroxysm. A convulsive seizure could readily be excited by dorsal flexion of the right foot, or by an attempt to test the knee-jerk. On the right side no knee-jerk could be obtained ; on the left side the knee-jerk was exaggerated. No true ankle clonus could be obtained, but dorsal flexion of the left foot gave rise to irregular semi-voluntary contractions of the calf-muscles, which in a few seconds passed into spasm of the same distribution as the spontaneous seizures. The patient could with difficulty be partially roused for a few seconds. The rectal temperature was $100\frac{2}{5}°$, the pulse 100, weak, and irregular.

In the absence of any knowledge of the history of this case it was impossible to come to a final conclusion as to the diagnosis. The most conspicuous symptom, clonic spasm in the lower extremities, indicated the presence of an irritative process in some part of the motor path. That this irritation was of cerebral origin was rendered practically certain by the presence of stupor and nystagmus. The combination of clonic spasm, stupor, and elevation of temperature might have suggested meningitis, but the slight degree of the fever, the

rapid recurrence of the clonic spasms, the absence of rigidity, and presence of nystagmus would not have lent much support to this view. The presence of nystagmus suggested the dependence of most of the symptoms upon a general disturbance of cerebral functions. No diagnosis was made, and it was decided to watch the further development of the symptoms before attempting to come to a definite conclusion. The next morning all the symptoms had passed away. It was learned that the patient had slipped upon the sidewalk and had fallen, striking his head against the stone, just before being brought into the hospital. The symptoms that have been described were therefore due, in all probability, to a severe concussion of the brain.

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