Handbook of the diseases of the nervous system / by James Ross.

DISEASES OF THE NERVOUS SYSTEM.

HANDBOOK

OF THE

DISEASES OF THE NERVOUS SYSTEM.

BY

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PREFACE.

THIS Handbook is intended for the use of students, and such of my medical brethren as are so fully occupied in practice that little time is left to them for reading lengthy treatises, and monographs on special subjects. The work is divided into two parts—a general and special neurology. In the former I have endeavored to give a brief outline of the evolution and dissolution of nervous structures and functions, adding a chapter on the general principles of treatment. In approaching a new subject our capacity to master it may be measured by the ease and thoroughness with which the mind assimilates with its previous stock of knowledge the new facts and relations presented to it. The student may, indeed, acquire much information concerning diseases or any other new subject of study without assimilating the new facts, which come under his notice, with well-established principles, but the knowledge thus obtained—if it deserves the name of knowledge—is not properly organized, and will be found to be both fleeting and ill-adapted for guidance in emergencies. Keeping these considerations in view, it has been my endeavor in this part to arrange the anatomical and physiological facts, with which the student is already more or less familiar, in such a way that his mind may be prepared readily to comprehend the multiform phenomena of disease, and to associate them with the fundamental laws of development. In the special part my great aim has been to make the work thoroughly practical. With this view I have adopted, as far as possible, a clinical classification, so that the diseases which are most apt to be mistaken for each other will be found described in close proximity, and the reader can thus note the various features which differentiate nervous diseases clinically allied. My rule of

selection has been to give the clinical descriptions with tolerable fulness, and to leave out the details of morbid anatomy and physiology, and almost all reference to the opinions and theories of different authorities. But although want of space has prevented me from quoting authorities, I am none the less grateful to men like Hughlings-Jackson, Wilks, Ferrier, Charcot, Westphal, Leyden, and the other great masters who have placed our knowledge of nervous diseases upon a secure and unshakable foundation.

JAMES ROSS.

MANCHESTER, November, 1885.

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ERRATA AND IMPORTANT OMISSIONS.

Page 24, line 3rd from the bottom, *for* "Fig. 2" *read* "Fig. 3."

,, 45, line 19th from the top, *for* "these" *read* "the."

,, 47, line 8th from the top, *for* "casserian" *read* "Gasserian."

,, 69, line 13th from the top, *for* "first" *read* "just."

,, 69, line 12th from the bottom, *for* "spirits" *read* "speech."

,, 108, line 13th from the bottom, *for* "Dissolution of the nervous system (continued)" *read* "Dissolution of the tissues when combined into a nervous system."

,, 116, between the 2nd and 3rd lines from the top *interpose* the whole of pages 118 and 119, and the first three lines of page 120.

,, 120, line 3rd from the top, *after* "disorders" *insert* "Féré found that this bundle is degenerated more frequently than has hitherto been suspected. It is found degenerated in lesions of the anterior segment of the internal capsule, and in cortical lesions of the fronto-parietal region. It is degenerated in many long-standing cases of aphasia."

,, 120, line 16th from the top, *after* "Fig. 63" *insert* "In a case reported by Féré, in which a small spot of softening was found post-mortem in the anterior segment of the pons, a bundle of degenerated fibres was traced transversely on each side as far as the middle peduncle of the cerebellum.

,, 120, line 17th from the top, *for* "Degeneration of nerve centres and conducting paths from disease" *read* "Degenerations secondary to disease of efferent conducting paths and sensory centres."

,, 122, line 1st and heading of pages 122, 123, and 124, *omit* "Anatomy and ".

,, 189, line 19th from the bottom, *after* "in front" *insert* "The internal rectus also acts in directing the eye to lateral objects in monocular vision when the other eye is closed."

,, 252, line 4th from the bottom, *for* "sub-clavicular" *read* "supra-clavicular."

,, 255, line 13th from the top, *for* "vertebra" *read* "vertebræ." And line 6th from the bottom, *for* "of the second" *read* "from the second."

,, 276, line 11th from the top, *for* "and softening" *read* "or softening."

,, 291, line 10th from the top, *for* "afforded the exercise" *read* "afforded for the exercise."

,, 360, line 11th from the bottom, *for* "Bristow" *read* "Bristowe."

,, 423, line 2nd from the top, *for* "said now" *read* "said never."

,, 424, line 9th from the bottom, *after* "paralyzed on either side" *insert* "The patellar-tendon reaction was exaggerated on the right and normal on the left side."

,, 520, line 19th from the top, *after* "liable to occur" *add* "Erb has reported cases of rupture of the tendo-Achillis in tabes, and it is therefore probable that some of the tendons undergo nutritive changes in this disease."

,, 595, line 2nd from the bottom, *for* "caases" *read* "causes."

,, 613, line 3rd from the top, *for* "ingested" *read* "injected."

,, 631, line 5th from the top, *for* "neuroma" *read* "neuromata."

,, 642, line 18th from the bottom, *for* "paralesia" *read* "paralexia."

,, 644, line 10th from the top, *for* "Riel" *read* "Reil."

,, 653, line 5th from the top, *omit* "should."

,, 667, line 16th from the bottom, *for* "become" *read* "becomes." And line 15th from the bottom, *for* "them" *read* "the vessels."

,, 680, line 6th from the top, *for* "Dr. Monry" *read* "Dr. Money."

,, 693, line 17th from the top, *for* "Pitre's" *read* "Pitres'."

,, 694, line 19th from the bottom, *for* "Riel" *read* "Reil."

,, 696, line 19th from the top, *for* "Bristow" *read* "Bristowe."

,, 697, line 6th from the top, *omit* one "the."

,, 702, line 12th from the foot, *after* "three" *add* "decades."

DISEASES OF THE NERVOUS SYSTEM.

CHAPTER I.

ANATOMICAL INTRODUCTION.

Before entering upon the study of the nervous system it is desirable to lay down a few general principles, to which all nervous structures, with their corresponding functions, must conform. The key to the interpretation of the form assumed by the constituent parts of the nervous system, and of the actions performed by each part, is best obtained by a close study of the order of development of nervous structures and functions as manifested in passing from the lower to the higher animals, and from the initial to the adult stages of animals. The knowledge obtained by these studies constitutes the comparative anatomy and embryology of the nervous system. The great law to which all developing organisms and organs, as well as all developing functions, must conform is the law of evolution. This law may be defined as a progressive integration of structure and function, during which there is a passage from the uniform to the multiform, from the simple to the complex, and from the general to the special.

In the lowest organisms, which consist of individual cells, or of an aggregation of cells without definite parts, each part performs all the vital functions. Each part possesses the fundamental property of irritability, and is capable of initiating movement, or, in other words, is automatic and contractile, and each is likewise metabolic, excretory, and reproductive. But on ascending in the scale of organization it is found that certain parts of the organism acquire the power of performing more perfectly a few functions, and ultimately one special function, while losing to a greater or less extent the power of performing the general functions. One part or tissue, for example, becomes adapted to the performance of the functions of contraction, while at the same time it gives up the functions of initiating movement and reproduction,

and only performs the metabolic and excretory functions in a very subordinate degree. This process has been named a "physiological division of labor," or a "specialization of function," when regarded from the point of view of the actions of the organism, and its anatomical counterpart in development is to be found in what is known as the "differentiation of structure."

FORMATION OF NERVOUS CONSTITUENTS.

The first beginning of a differentiation of the nervous from the other elements of organisms is well illustrated in *Hydra*. In this organism the internal end of an ectodermic cell is prolonged into a process, which, being shielded from external influences, tends only to contract when it receives a stimulus through the external end. In other words, the internal end performs the work, and the external determines when the work shall be done: the one is *operative*, the other *regulative*. This differentiation of structure is carried still further in *Beræ*, where the internal and external ends of the ectodermic cells are represented by two different cells connected by a thin fibre. The changes set up in the external or sensitive cell are conducted through the fibre to the internal cell, which it excites to contract. This new arrangement of fibre introduces us to a new specialization of function. The regulative or *automatic* cell and the work cell are separated from each other by a considerable distance, but the molar contraction of the one is coördinate with the molecular motion of the other by the *internuncial* function of the fibre. The property the fibre possesses of transmitting the state of activity from one end to the other is called its *conductivity*.

The next step in development consists in the differentiation of the external or sensitive cell into two: one of which becomes adapted to responding to external stimulation alone, and the other to modifying the impulses which are sent to it from the external cell and transmitting them to the work cell. The nervous mechanism now consists of an external sensory cell, an *afferent* fibre connecting it with a central regulating cell, and an *efferent* fibre or fibres connecting this last with work cells. This mechanism constitutes a *reflex* loop, and its function is named *reflex action*. One other complication of this simple mechanism may be mentioned: When an automatic or reflex cell is already engaged in action, a new stimulus brought to it by means of a second afferent fibre may check, instead of still further exciting that action, so that this function introduces us to *inhibitory* as well as *excito-motor* fibres. The inhibitory fibres are either afferent, or connect one central cell with another central cell, being then *intercentral*, and probably never connect a central cell directly with a work cell.

FORMATION OF NERVOUS TISSUES.

On looking at a simple nervous system, the greatest contrast in structure is manifested between certain knots termed *ganglia* and certain cords termed *nerves*, and, since these parts exhibit the greatest structural contrast existing in the nervous system, they may also be expected to exhibit the widest functional contrast. The ganglia are composed of nerve cells, with their connecting processes, held together by a fine connective tissue; and the nerves are composed of nerve fibres arranged side by side in a bundle—these being also held together by a firm connective tissue and by a fibrous sheath.

Functionally regarded, the ganglia are originators of motion, and, to some extent, conductors also; while the nerves are mainly conductors, although it is probable that they are also in some small degree originators of motion. A still further examination shows that the afferent fibres are provided with peripheral end-organs, which are adapted for receiving impressions from environing agents and objects; while the efferent fibres are provided with arrangements by means of which the molecular motion of the nervous system is transmitted into the molar motion of the work organs.

FORMATION OF A NERVOUS SYSTEM.

It has just been seen how the cells and fibres which constitute the nerve elements integrate so as to form the simple tissues, and now we must follow this progressive integration to still higher stages. In the higher animals, the ganglia, instead of appearing as small knots, have come, by approximation and fusion, to form a continuous mass, which, from its color, is termed the *gray substance;* and the fibres, instead of always appearing as cords connecting separate ganglia, also form a continuous mass called the *white substance.* Now, this fusion of nerve centres and nerve strands takes place when the functions are integrated in corresponding degree, and fails to take place when the functions remain in large measure independent of one another.

The internal organs of the body which are derived from the hypoblast of the embryo are much more independent of one another in their actions than are the external organs derived from the epiblast; and the functions of the former are regulated chiefly by means of the ganglia of the *sympathetic system*, which are only connected by small cords, while those of the latter are regulated by means of large masses of gray and white substance, which constitute the *encephalo-spino-neural system.*

The parts of the body which are derived from the mesoblast form a system of organs which serve to connect the external and internal organs; and in so far as the intermediate tissues subserve the functions of the external organs their actions are regulated from the encephalo-spinal and the spino-neural centres; and when they subserve the functions of the internal organs their actions are regulated by the sympathetic system; while in so far as their function is intermediate between the internal and external organs, but partially independent of them, their actions are regulated by an intermediate and partially independent system, termed the *vaso-motor*.

A plane passing longitudinally and from behind forwards through a human being bisects the body into two bilaterally symmetrical divisions, and this statement is equally true of the nervous system as of the body as a whole. The sympathetic system of each half of the body is represented by a gangliated cord, which is situated along the side of the vertebral column; while the encephalic and spinal systems of each side are represented by the cerebral and cerebellar hemispheres, the crus cerebri, and the lateral halves of the pons, medulla oblongata, and spinal cord. Although the two lateral halves of the body are more or less symmetrical, the organs of the one side are to a considerable extent dependent upon those of the other in their functions, and consequently the actions of the organs of the two sides must be duly coördinated. The structural counterpart of this interdependence is to be found, in the case of the sympathetic system, in the plexus of fibres and small ganglia which pass in front of the vertebral column and connect the gangliated cords of the two sides with one another; and in the encephalic and spinal systems by the commissures of the spinal cord, the median raphé of the medulla oblongata and pons, the middle peduncles of the cerebellum, the commissures of the third ventricle, and the corpus callosum.

The body is composed of segments placed end on end, and there is a similar distribution of the nervous system. That this is the case with the greater part of the sympathetic is readily recognized. Each segment of the body is represented by a vertebra and its appendages, and most of the vertebræ have a corresponding ganglion for each lateral half, although fusion of two or more ganglia in the successive segments of the cervical region and in those of the coccyx prevents the correspondence from being altogether perfect.

In the spinal cord the analogous ganglia of the same segment and the homologous ganglia of successive segments have become so completely integrated that they form a closed tube of gray matter, which extends from the conus medullaris up through the whole length of the

cord, the floor of the fourth ventricle, the aqueduct of Sylvius, and the gray matter of the third ventricle, to terminate at the tuber cinereum. One consequence of the fusion of homologous ganglia is that the parts of the cord which correspond to the different segments of the body have undergone considerable displacement. The cord usually ends at the lower border of the body of the first lumbar vertebra; but the nerves which descend to pass out through the remaining lumbar intervertebral foramina and through the sacral and sacro-coccygeal foramina, show that the lower part of the cord presides over the functions of the lower segments of the body, although it has by the fusion and approximation of the homologous centres suffered considerable displacement.

A still further integration has taken place in the cephalic centres, and the highest ganglia—the cerebrum and cerebellum—consist of large aggregations of gray and white matter, in which it is difficult to find any trace of the separate ganglia and the conducting paths of which they are composed. The cephalic ganglia, spinal cord, and peripheral nerves form one functionally indivisible system which may be named the *encephalo-spino-neural system.*

Encephalo-spino-neural System.

We shall now make a few general remarks on (1) the arterial supply of this system; (2) its topography; (3) its relations to the skeleton; (4) the topography of the internal parts of the cerebrum, as being the part of the system in which this knowledge is most generally required for practical purposes; and (5) the internal structure of the system.

1. *The Arterial Supply.*

a. The Spinal Arteries.

(1) The *spinal branches* enter the intervertebral foramina along with the roots of the spinal nerves, and are distributed to the vertebræ, spinal membranes, and spinal cord. These arteries are derived from the lateral sacral, ilio-lumbar, lumbar, aortic intercostal, the ascending cervical branch of the inferior thyroid, and vertebral arteries.

(2) The *anterior spinal artery* is a small vessel derived from the vertebral artery near its termination in the basilar. This vessel joins with its fellow of the opposite side to form a single vessel which descends along the anterior aspect of the spinal cord, and is continued to the lower end of the cord as the *anterior median artery.*

(3) The *posterior spinal artery* is derived from the inferior cerebellar

artery; it winds round the medulla oblongata to reach the posterior aspect of the cord, when it is continued onwards to the cauda equina.

(4) The *nutritive arteries* of the spinal cord are sufficiently indicated in the annexed diagram (Fig. 1).

FIG. 1.

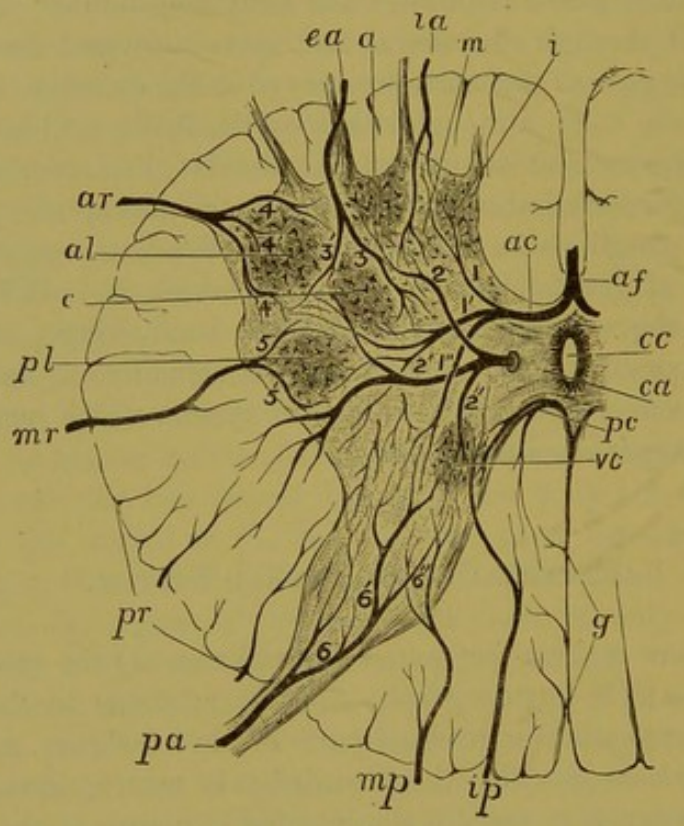


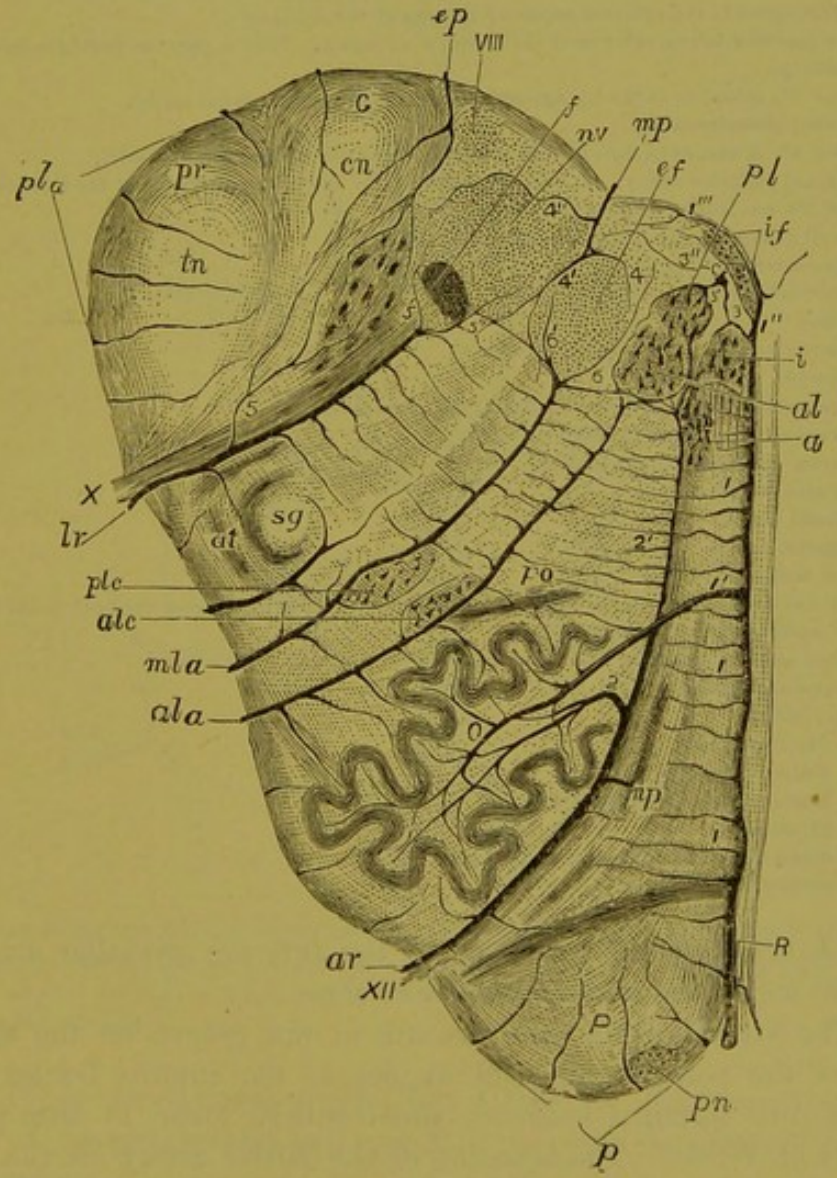
DIAGRAM OF THE DISTRIBUTION OF THE BLOODVESSELS, AND GROUPING OF GANGLION CELLS IN THE SPINAL CORD. (YOUNG.)

Anterior median artery.
af, Arteries of the anterior median fissure.
ac, Artery of the anterior commissure.
1, Anterior branch.
1′, Median branch.
1″, Posterior branch.
ca, Central artery.
2, Anterior branch.
2′, Median branch.
2″, Posterior branch.
pa, Posterior root arteries.
6 6′ 6″, Arteries of posterior horns.
ia, Internal anterior root artery.
ea, External anterior root artery.
3 3′, Internal and external branch.
ar, Antero-lateral branch.
4, Anterior branch.
4′, Median branch.
4″, Posterior branch.
mr, Median lateral artery.
5 5′, Anterior and posterior branches.
pr, Posterior lateral arteries.
ip, Internal posterior artery.
mp, External posterior artery.
g, Arteries of the column of Goll.
pc, Artery of the posterior commissure.
vc, Vesicular column of Clarke.
i, Internal group of cells.
a, Anterior group.
al, Antero-lateral group.
pl, Postero-lateral group.
c, Central group.
m, Median area.

b. The Arteries of the Medulla Oblongata, Pons, Crura Cerebri, and Cerebellum.

(1) The *vertebral artery* winds backwards round the articulating process of the atlas, and, after piercing the dura mater, enters the skull through the foramen magnum, and terminates at the lower border of the pons Varolii by uniting with the corresponding vessel of the opposite side to form the *basilar artery*. Besides the spinal branches already

Fig. 2



Section of the Medulla Oblongata, showing the Distribution of the Vessels.

R, Artery of the Median Raphé.

1 1 1, Branches to the *formatio reticularis*.

1', Branch to the olivary body.

1'', Branches to the hypoglossal nucleus.

1''', Branches to the floor of the fourth ventricle, and to the internal inferior nuclei of the facial (*if*).

p, Pyramidal arteries.
ar, Anterior root artery (hypoglossal).
2′, Branch to the olivary body.
2″, Branches to the *formatio reticularis*. It terminates in branches to the hypoglossal nucleus.
lr, Lateral root artery (vagus).
5, Branch to the restiform body and the inner division of the inferior cerebellar peduncle.
5′, Branches to the nucleus of the vagus. Also gives branches to the ascending root of the fifth and the *formatio reticularis*.
ala, The anterior lateral artery of the medulla oblongata. It supplies branches to the *formatio reticularis*, olivary body, anterior nucleus of the lateral column (*alc*), and terminates in branches to the hypoglossal nucleus.
mla, The middle lateral artery of the medulla oblongata. It supplies branches to the *formatio reticularis*, the posterior nucleus of the lateral column (*plc*), and terminates in branches which are distributed to the external accessory nucleus of the facial (*ef*).
pla, The posterior lateral arteries of the medulla oblongata. They supply the restiform bodies.
C, Central artery.
3 3′ 3″, Branches to the hypoglossal and external accessory facial nuclei.
mp, Median posterior artery.
4 4′ 4″, Branches to the external accessory facial and pneumogastric nuclei.
ep, External posterior artery. It supplies branches to the internal division of the inferior peduncle of the cerebellum and restiform body.
i, Internal group of cells of the hypoglossal nucleus.
al, Antero-lateral " "
pl, Postero-lateral " "
a, Anterior " "
alc, Anterior nucleus of the lateral column.
plc, Posterior " "
VIII, Inferior portion of the posterior median acoustic nucleus.
if, Internal accessory facial nuclei.
ef, External accessory facial nucleus.
f, Fasciculus rotundus.
XII, Hypoglossal nerve.
X, Pneumogastric nerve.
G, Column of Goll.
pr, Posterior root-zone. The direct cerebellar tract forms a thin band lying external to the column of Goll and posterior root-zone.
cn, Clavate nucleus.
tn, Triangular nucleus.
o, Olivary body.
po, Parolivary body.
np, Nucleus of the pyramid.
pn, Nucleus of the arciform fibres.
P, Anterior pyramid.
at, Ascending root of the trigeminus.
sg, The substantia gelatinosa.

described, the vertebral gives rise to the *inferior cerebellar artery*, as well as to meningeal and muscular branches.

(2) The *basilar artery* runs forwards in the groove on the anterior surface of the pons Varolii, and divides at the anterior border of the pons into two terminal branches, which will be found to form part of the circle of Willis. The branches of the basilar artery are the transverse arteries of the pons, the middle and superior cerebellar arteries, and the posterior cerebral artery. These branches are sufficiently indicated in the annexed diagram (Fig. 2).

(3) The nutritive arteries of the medulla oblongata and pons are shown in the annexed diagram (Fig. 2).

c. The Arteries of the Cerebrum.

(1) The *posterior cerebral arteries* are the terminal branches of the basilar trunk, and each artery winds round the crus cerebri to reach the occipital lobe.

(*a*) The *posterior median group* (Fig. 3, 2), which pierce the posterior perforated space and supply the internal surface of the optic thalamus and the walls of the third ventricle.

Fig. 3.

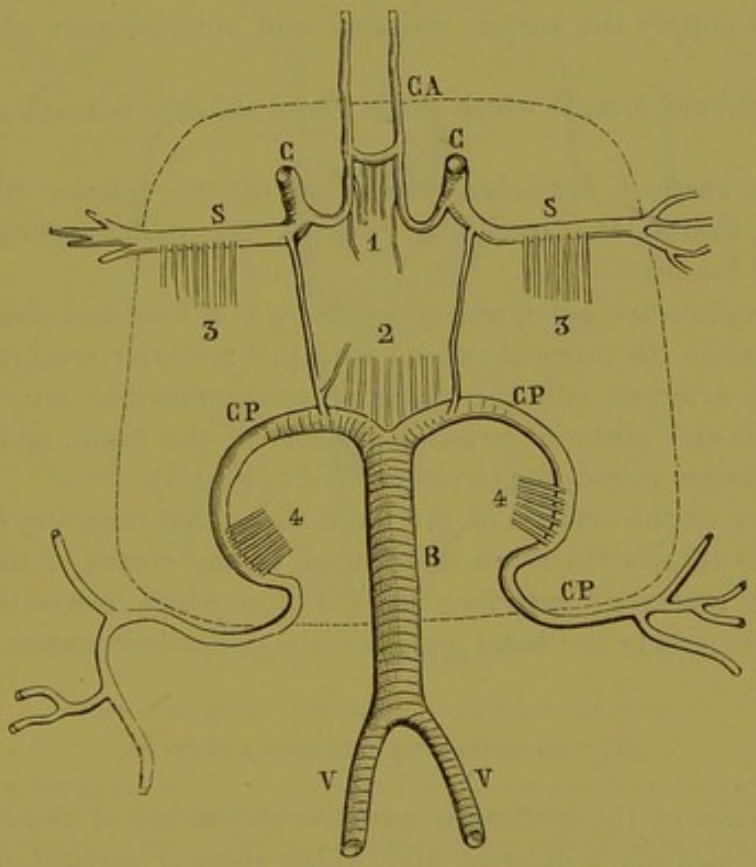


Diagram of the Distribution of the Vessels at the base of the Cerebrum. (After Charcot.)

CA, Anterior cerebral artery; S, S, Sylvian arteries; V, V, Vertebral arteries; B, Basilar; CP, CP, Posterior cerebral arteries; 1, 2, 3, 3, 4, 4, Groups of nutritive arteries. The line ------ limits the ganglionic vascular area.

(*b*) The *postero-lateral group* (Fig. 3, 4) enter the brain behind the border of the crus cerebri and pass into the optic thalamus and corpora quadrigemina.

(*c*) The *cortical branches*, consisting of the anterior temporal, the posterior temporal, and the occipital arteries.

(2) The *anterior cerebral artery* (Fig. 3, CA), derived from the internal carotid artery, runs forwards in the longitudinal fissure, and,

turning round the corpus callosum, is distributed to the anterior part of the cerebrum. The arteries of the two sides are united at their commencement by a short transverse branch, the *anterior communicating artery*.

(*a*) The *anterior median group* (Fig. 3, 1) supply the anterior part of the head of the caudate nucleus.

(*b*) The cortical branches.

(3) The *middle cerebral or Sylvian artery* (Fig. 3, S) is the most important branch, and the direct continuation of the internal carotid.

(*a*) The *antero-lateral group* (Fig. 3, 3) pierce the anterior perforated space, and supply the corpus striatum and anterior part of the optic thalamus.

(*b*) A *choroid branch* which winds round the crus to reach the choroid plexus.

(*c*) The *cortical branches*, consisting of the inferior frontal, the ascending frontal, the ascending parietal, the parieto-sphenoidal and sphenoidal branches.

(4) The *posterior communicating artery* is a long and slender vessel which connects the internal carotid with the posterior cerebral arteries. These two arteries, with the posterior communicating artery, connect the anterior and posterior arterial systems of the brain, and complete the anastomosis round the circle of Willis (Fig. 3).

A survey of the arteries of the brain shows that they may be divided into (1) the vessels which are distributed to the cortex of the brain—the *cortical system of arteries;* and (2) those which pierce the base of the organ to supply the basal ganglia—the *ganglionic system.*

(1) THE CORTICAL SYSTEM OF ARTERIES.

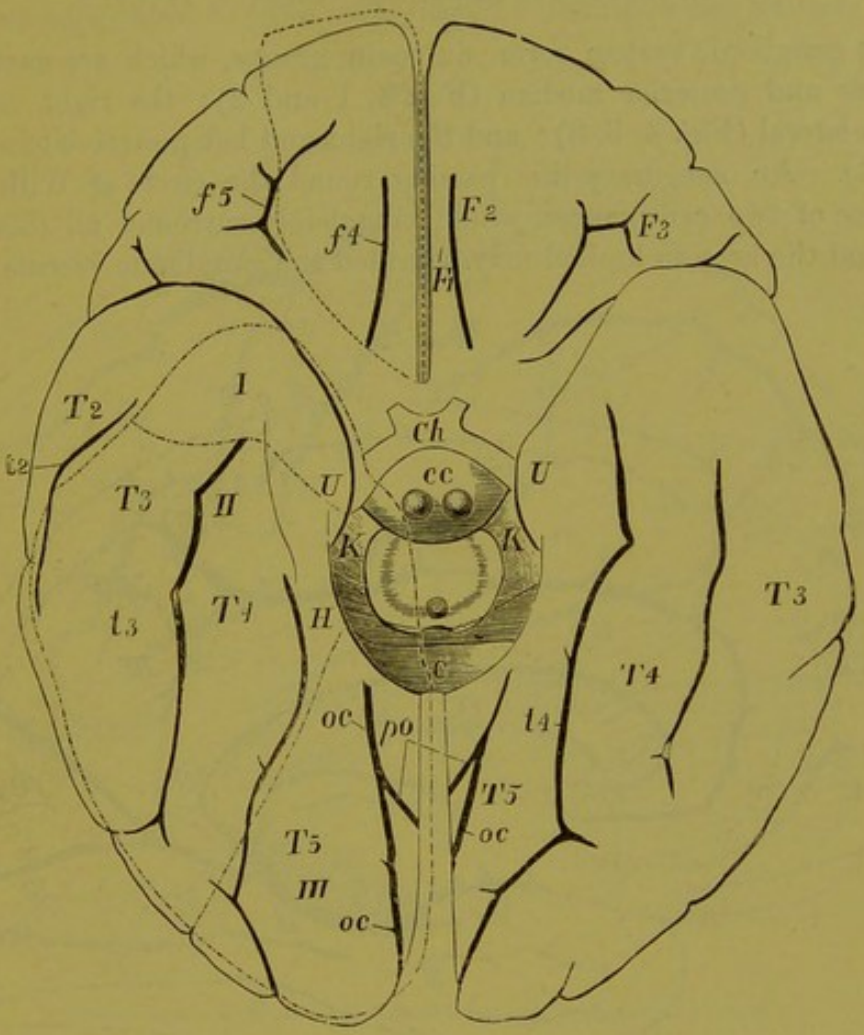
The arteries of the cortical or nutritive system are of two kinds: (*a*) the *long or medullary arteries*, and (*b*) the *short or cortical arteries.*

(*a*) The *long or medullary arteries* pass into the substance of the centrum ovale without communicating with one another, except by fine capillaries, and consequently each constitutes a small, independent vascular territory. These vessels extend as far as the ganglionic system, but do not appear to anastomose with it.

(*b*) The *cortical or nutritive arteries* arise from the vascular network of the pia mater, and most of them terminate in the gray matter; but a few of them send capillaries to the centrum ovale.

The distribution of the cortical arteries is shown in the annexed diagrams (Figs. 4, 5, and 6).

FIG. 4.



VIEW OF THE BRAIN FROM BELOW. (After ECKER and DURET.)

Distribution of Vessels.

The region bounded by the line (- - - - - -) represents the territory over which the *Internal and Inferior Frontal Branches* of the ANTERIOR CEREBRAL ARTERY are distributed.

The regions bounded by the line (— — — — — — —) represent the territories over which the branches of the POSTERIOR CEREBRAL ARTERY are distributed

I. Is the region of the *Anterior Temporal Artery.*
II. " " *Posterior Temporal Artery.*
III. " " *Occipital Artery.*

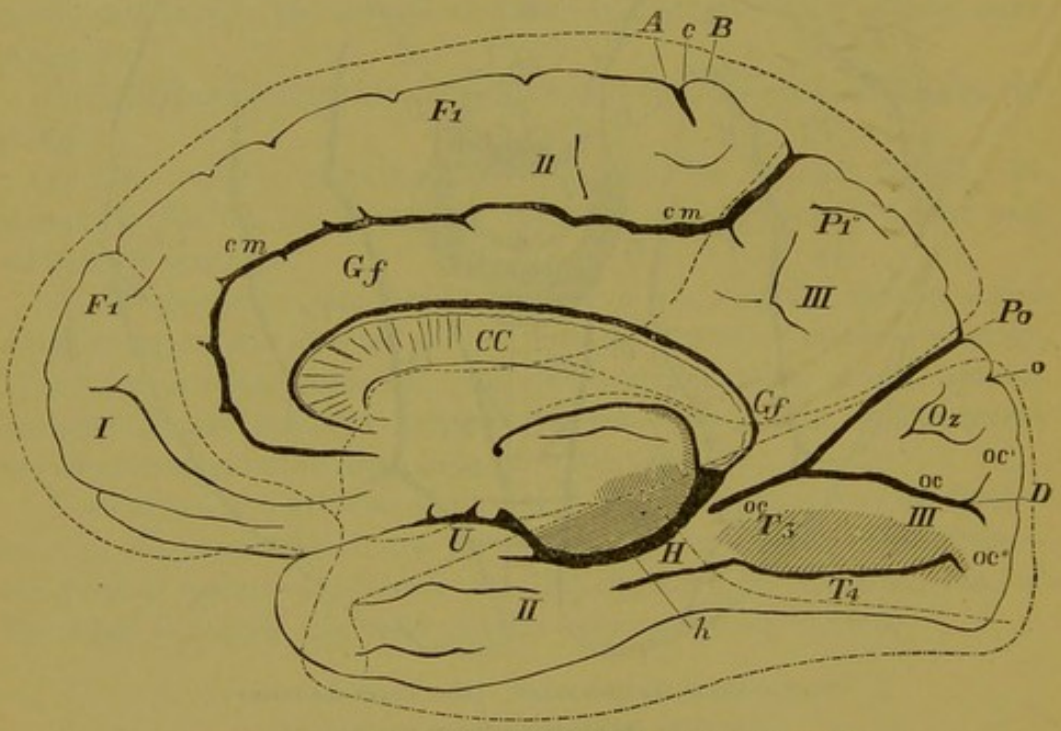
Fissures and Convolutions.

F1, Gyrus Rectus.
F2, Middle frontal convolution.
F3, Inferior frontal convolution.
f4, Sulcus olfactorius.
f5, Sulcus orbitalis.
T2, Second or middle temporo-sphenoidal convolution.
T3, Third or inferior temporo-sphenoidal convolution
T4, Gyrus occipito-temporalis lateralis (lobulus fusiformis).
T5, Gyrus occipito-temporalis medialis (lobulus lingualis).
t4, Sulcus occipito-temporalis inferior.
t3, Sulcus temporo-sphenoidalis inferior.
t2, Sulcus temporo-sphenoidalis medialis.
po, Parieto-occipital fissure.
oc, Calcarine fissure.
H, Gyrus hippocampi.
U, Gyrus uncinatus.
Ch, Optic chiasma.
cc, Corpora albicantia.
KK, Crura cerebri.
C, Corpus callosum.

(2) THE CENTRAL OR GANGLIONIC SYSTEM OF ARTERIES.

The ganglionic system form six main groups, which are named the anterior and posterior median (Fig. 3, 1 and 2); the right and left antero-lateral (Fig. 3, 3, 3); and the right and left postero-lateral (Fig. 3, 4, 4). An imaginary line passing round the circle of Willis, at a distance of two centimetres, would completely surround all these vessels, and the area so limited may be called the ganglionic vascular area.

FIG. 5.



INNER SURFACE OF RIGHT HEMISPHERE. (After ECKER and DURET.)

Distribution of Vessels.

The regions bounded by the line (- - - - - -) represent the territories over which the branches of the ANTERIOR CEREBRAL ARTERY are distributed.

I. Is the territory of the *Interior and Anterior Frontal Artery.*
II. " " *Internal and Middle* " "
III. " " *Internal and Posterior* " "

The regions bounded by the line (— — — — — — — —) represent the territories over which the branches of the POSTERIOR CEREBRAL ARTERY are distributed.

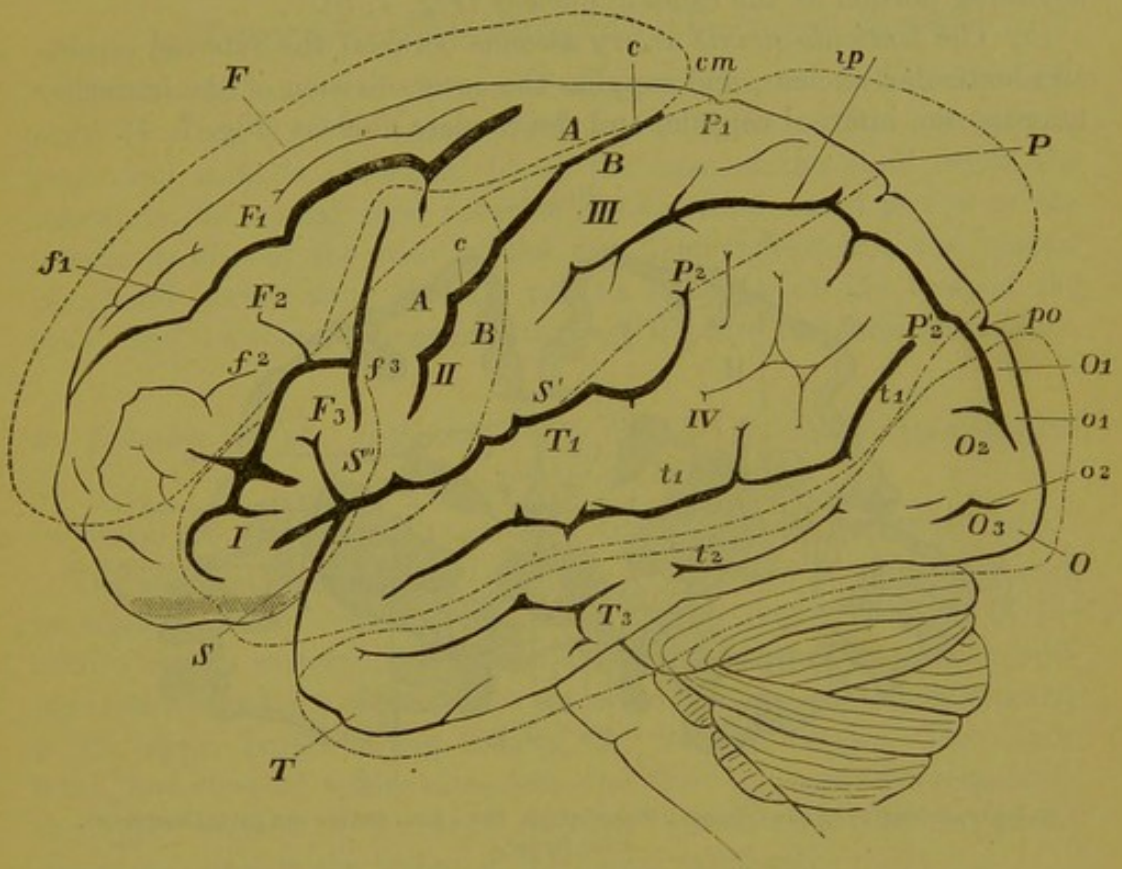
II. Is the territory of the *Posterior Temporal Artery.*
III. " " *Occipital Artery.*

Fissures and Convolutions.

CC, Corpus callosum, longitudinally divided; Gf, Gyrus fornicatus; H, Gyrus hippocampi; h, Sulcus hippocampi; U, Uncinate gyrus; cm, Sulcus calloso-marginalis; F_1, Median aspect of the first frontal convolution; c, Terminal portion of the sulcus centralis, or fissure of Rolando; A, Anterior; B, Posterior central convolution; P_1'', Præcuneus; Oz, Cuneus; Po, Parieto-occipital fissure; o, Sulcus occipitalis transversus; oc, Calcarine fissure; oc', Superior, oc'', Inferior ramus of the same; D, Gyrus descendens; T_4, Gyrus occipito-temporalis lateralis (lobulus fusiformis); T_5, Gyrus occipito-temporalis medialis (lobulus lingualis).

All these vessels are *terminal arteries*. Some of these branches are of sufficient importance to have been specially described and named.

FIG. 6.



OUTER SURFACE OF THE LEFT HEMISPHERE. (After ECKER and DURET.)

Distribution of Vessels.

The region bounded by the line (- - - - - -) represents the territory over which branches of the ANTERIOR CEREBRAL ARTERY are distributed.

The anterior regions bounded by the line (— — — — — — —) represent the territories over which branches of the MIDDLE CEREBRAL ARTERY are distributed.

I. Is the region of the *External and Inferior Frontal Artery.*
II. " " *Anterior Parietal Artery.*
III. " " *Posterior Parietal Artery.*
IV. " " *Parieto-sphenoidal Artery*

The posterior and inferior region bounded by the line (— - — - — - — - — - —) represents the territory over which branches of the POSTERIOR CEREBRAL ARTERY are distributed.

Fissures and Convolutions.

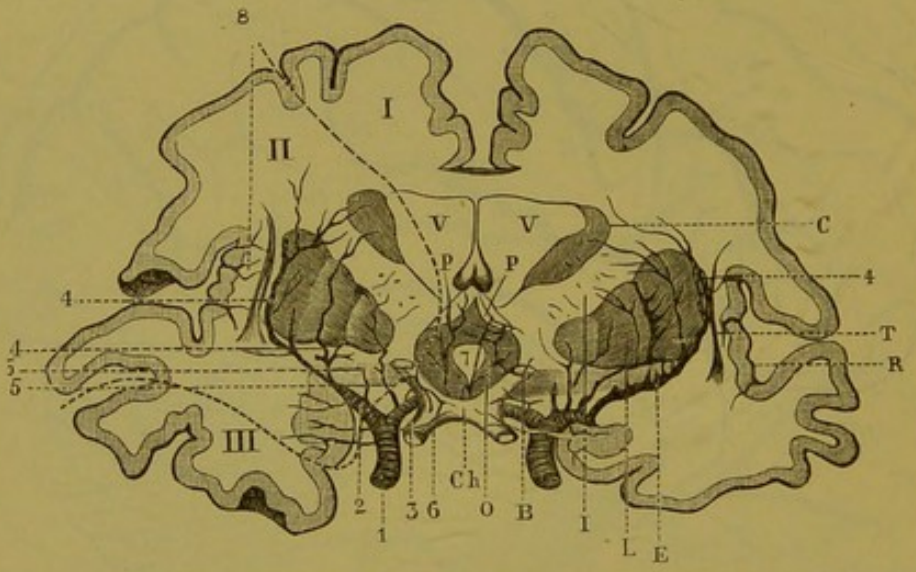
F, Frontal lobe. P, Parietal lobe. O, Occipital lobe. T, Temporo-sphenoidal lobe. S, Fissure of Sylvius, S′ Horizontal, S″ Ascending ramus of the same. *c*, Sulcus centralis or fissure of Rolando. A, Anterior central or ascending frontal convolution. B, Posterior central or ascending parietal convolution. F_1 Superior, F_2 Middle, and F_3 Inferior frontal convolutions. f_1 Superior and f_2 Inferior frontal sulcus; f_3 Sulcus præcentralis. P_1 Superior parietal or postero-parietal lobule; P_2 Inferior parietal lobule, viz.: P_2 Gyrus supra-marginalis, P_2' Gyrus angularis. *ip*, Sulcus intra-parietalis *cm*, Termination of the calloso-marginal fissure. O_1 First, O_2 Second, O_3 Third occipital convolutions. *po*, Parieto-occipital fissure. *o*, Sulcus occipitalis transversus; o_2 Sulcus occipitalis longitudinalis inferior. T_1 First, T_2 Second, T_3 Third temporo-sphenoidal convolutions. t_1 First, t_2 Second temporo-sphenoidal fissures.

Branches of the Middle Cerebral Artery.

(*a*) The *lenticular branches* are two or three small twigs which are distributed to the two inner divisions of the lenticular nucleus and the adjoining portion of the caudate nucleus (Fig. 7, 5).

(*b*) The *lenticulo-striate artery* ascends between the external capsule and lenticular nucleus, and supplies the outer division of the lenticular nucleus, the internal capsule, and the caudate nucleus (Fig. 7, 4).

FIG. 7.



TRANSVERSE SECTION OF THE CEREBRAL HEMISPHERES, ABOUT 1 CM. BEHIND THE OPTIC COMMISSURE. (From DURET.)

ARTERIES OF THE CORPUS STRIATUM.—*Ch*, Chiasma; *B*, Section of the optic tract; *L*, Lenticular nucleus; *I*, Internal capsule; *C*, Caudate nucleus; *E*, External capsule; *T*, Claustrum; *R*, Island of Reil; *V*, *V*, Section of the lateral ventricle; *P*, *P*, Anterior pillars of the fornix; *O*, Gray substance of the third ventricle.

VASCULAR AREAS.—I, Anterior cerebral artery; II, Middle cerebral artery; III, Posterior cerebral artery.—1, Internal carotid artery; 2, Sylvian artery; 3, Anterior cerebral artery; 4, 4, External arteries of the corpus striatum (lenticulo-striate artery); 5, 5, Internal arteries of the corpus striatum (lenticular arteries). The opto-striate artery is not represented in the figure.

(*c*) The *lenticulo-optic artery* also ascends between the external capsule and the lenticular nucleus, and supplies branches to the posterior part of the nucleus and of the internal capsule, and terminates in the anterior and external part of the optic thalamus.

Branches of the Anterior Cerebral Artery.

The *anterior median group* supplies the anterior part of the caudate nucleus.

Branches of the Posterior Cerebral Artery.

(*a*) The *posterior internal artery* of the optic thalamus is distributed to the internal surface of that ganglion.

(*b*) The *posterior external artery* of the optic thalamus is distributed to the posterior and external portion of that ganglion, and also supplies the external geniculate bodies.

2. *Topography of the Encephalo-spino-neural System.*

The fissures and convolutions of the cerebrum are sufficiently indicated in Figs. 4 to 6 without further description. The cerebellum, pons, and medulla oblongata lie at the base of the skull below the tentorium, the spinal cord occupies the vertebral canal as low as the inferior border of the body of the first lumbar vertebra, the cranial peripheral nerves issue through various foramina at the base of the brain, and the spinal nerves pass through the intervertebral foramina.

3. *Relation of the Different Parts of the Encephalo-spino-neural System to the Skeleton.*

a. Relations of the Spinal Cord and Nerves to the Vertebral Canal.

The annexed diagram (Fig. 8) shows the position of the spinal cord in relation to the vertebral canal in which it lies. The tips of the cervical spines correspond nearly to the lower borders of the corresponding vertebræ. Each of the upper three dorsal spines corresponds nearly to the upper border of the body of the vertebra below. The ninth, tenth, and eleventh spines slope less, and their tips again correspond to the upper borders of the next vertebræ below, while the rest of the spines are opposite the bodies of their own vertebræ.

b. Relations of the Cerebral Convolutions to the Skull.

The convolutions and fissures of the surface of the brain have just been described, and we must now give a brief account (1) of the topography of the surface of the skull; and (2) of the relations existing between the different areas of the skull and the respective convolutions and fissures of the brain.

(1) topography of the external surface of the skull.

The topography of the external surface of the skull has been studied by Broca, Féré, Turner, and others, with the view of connecting definite points and areas on the surface with the respective convolutions and fissures of the brain. We shall adopt the divisions and descriptions of Turner in this place:

Definite Landmarks on the Surface of the Skull.—The following structures and markings are easily recognized on the skull. The external occipital protuberance (Fig. 9, *o*), the parietal (P) and frontal (F)

eminences, and the external angular process of the frontal bone (A), the coronal (*c*) and lambdoidal (*l*), squamous (*s*), squamoso-sphenoid

FIG. 8.

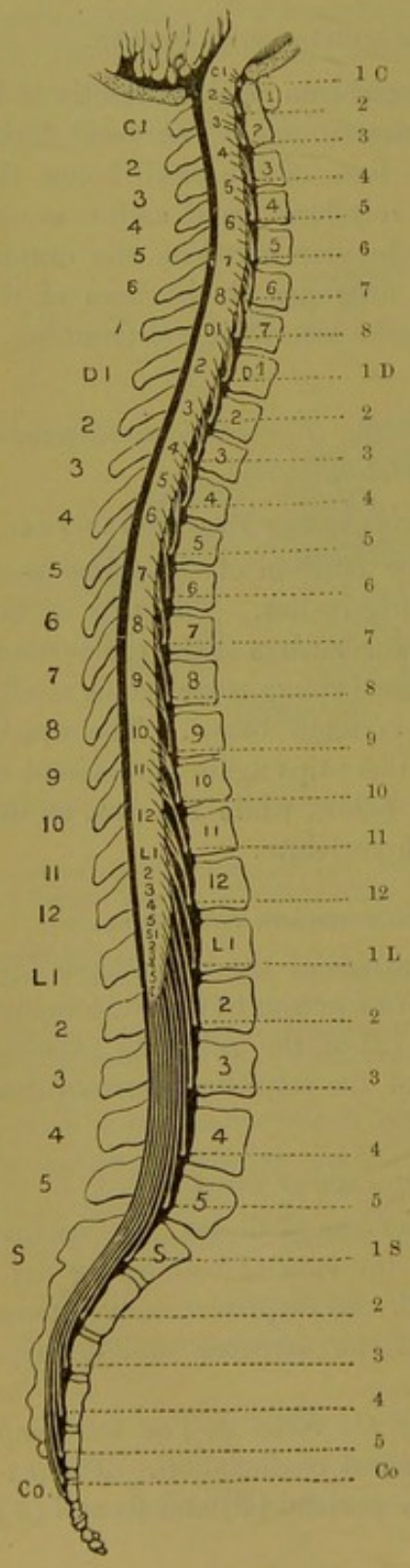


DIAGRAM SHOWING THE RELATION OF THE SPINES OF THE VERTEBRÆ TO THE ORIGIN OF THE NERVES. (After GOWERS.)

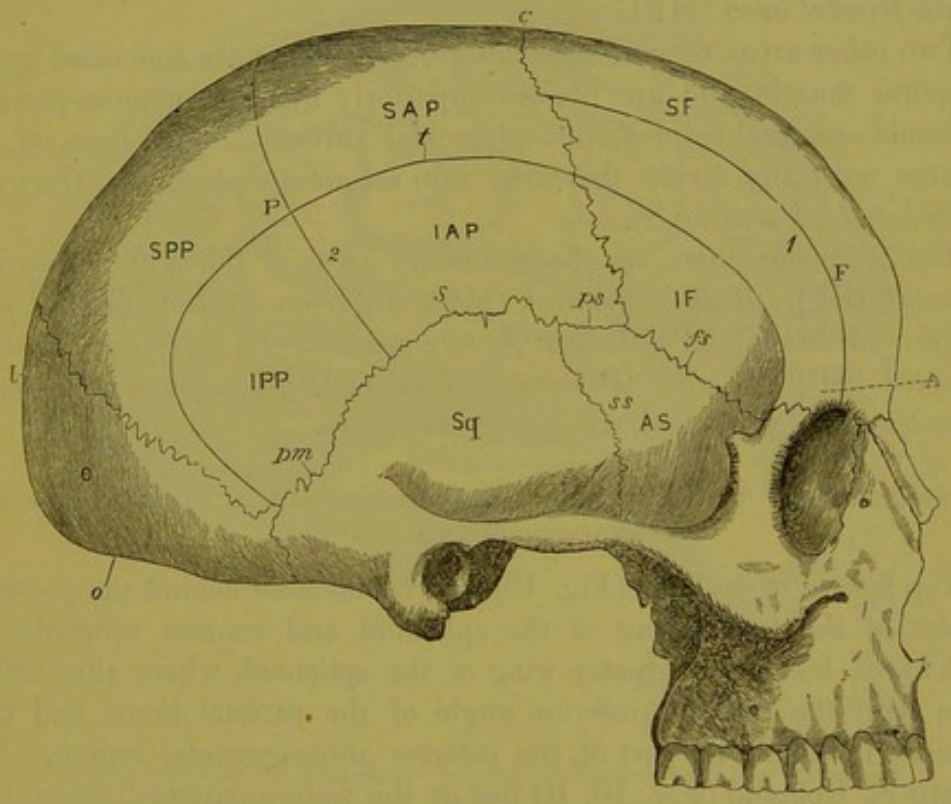
The first three cervical spines are opposite the origins of the 3d, 4th, and 5th cervical nerves. The 6th and 7th pairs arise opposite the intervals between the 4th and 5th and the 5th and 6th cervical spines respectively The 6th cervical spine corresponds to the origin of the 8th cervical nerve, and the 7th cervical spine to the first dorsal nerve. The first four dorsal spines vary. The 1st spine corresponds to the interval between the 2d and 3d pairs, or to the origin of the 3d pair. The 2d spine is between the 3d and 4th pairs, or opposite the 4th pair The 3d spine is opposite the 5th, or the interval between the 5th and 6th pairs. The 4th spine is opposite the lower part of the origin of the 6th pair, or even below it The 5th spine always corresponds to the origin of the 7th pair; the 6th spine to the 8th pair; the 7th to the 9th pair; the 8th to the upper part of the 10th pair; the 9th to the 11th pair; and the 10th to the 12th pair. The 1st lumbar arise opposite the 11th dorsal spine; the second lumbar opposite the interval between the 11th and 12th spines; the 3d and 4th opposite the 12th spine; the 5th lumbar and the 1st sacral opposite the interval between the 12th dorsal and 1st lumbar spines, while the remaining sacral nerves arise nearly opposite the 1st lumbar spine.

The cervical enlargement of the cord corresponds nearly to the bodies and spines of the cervical vertebræ, while the lumbar enlargement corresponds to the bodies of the 11th and 12th dorsal and 1st lumbar vertebræ, and to the lower three dorsal and 1st lumbar spines.

(*ss*), and parieto-sphenoid sutures (*ps*), and the curved line of the temporal ridge (*t*).

Primary Areas of the Skull.—The coronal suture (*c*) forms the posterior boundary of the *frontal area.* A vertical line (Fig. 9, 2) drawn from the squamous suture (*s*) upwards through the parietal eminence (P) to the sagittal suture lies almost parallel to the coronal suture,

FIG. 9.



LATERAL VIEW OF THE HUMAN SKULL. (FERRIER.)

A, The external angular process of the frontal bone. F, The frontal eminence. P, The parietal eminence. *o*, The occipital protuberance. *c*, The coronal suture. *l*, The lambdoidal suture *s*, The squamous suture. *t*, The temporal ridge. *fs*, The fronto-sphenoid suture. *ps*, The parieto-sphenoid suture. *ss*, The squamoso-sphenoid suture. *pm*, The parieto-mastoid suture 1, Frontal line. 2, Parietal line. SF, MF, IF, The supero-, mid-, and infero-frontal subdivisions of the frontal area. SAP, The supero-antero-parietal area. IAP, The infero-antero-parietal area. SPP, The supero-postero-parietal area. IPP, The infero-postero-parietal area. O, The occipital area. Sq, The squamoso-temporal area. AS, The ali-sphenoid area.

and subdivides the parietal region into an *antero-parietal* (Fig. 9, SAP+IAP) and a *postero-parietal area* (Fig. 9, SPP+IPP). The *occipital region* lies between the lambdoidal suture (*l*) and the occipital protuberance (*o*), with the superior curved line extending from it (Fig. 9, *o*).

Secondary Areas of the Skull.—These four primary divisions of the skull may be subdivided into secondary areas. The temporal ridge

(Fig. 9, *t*), starting from the external frontal process, curves backwards across the frontal (A), antero-parietal, and post-parietal areas to the internal angle of the occipital bone, and subdivides each of these regions into an upper and a lower area. The upper frontal area, which includes all the frontal regions above the temporal ridge, is again divided by a line drawn vertically upwards and backwards from above the orbit through the frontal eminence to the coronal suture (Fig. 9, *c*). This line divides the upper frontal area into a *supero-frontal* (SF) and a *mid-frontal area* (MF).

Two other areas remain to be described. These are concealed by the temporal muscle, and are limited superiorly by the squamoso-parietal, sphenoido-parietal, and fronto-sphenoidal sutures. The lines of the sutures naturally divide this area into a *squamoso-temporal* (Sq) and *ali-sphenoidal* area (AS).

The following, then, are the secondary areas of the skull: *Superior Frontal* (SF), *Middle Frontal* (MF), *Inferior Frontal* (IF), *Upper Antero-parietal* (SAP), *Lower Antero-parietal* (IAP), *Upper Postero-parietal* (SPP), *Lower Postero-parietal* (IPP), *Occipital* (O), *Squamoso-temporal* (Sq), and *Ali-sphenoidal* (AS).

(2) RELATIONS OF THE DIFFERENT AREAS OF THE SKULL TO THE RESPECTIVE CONVOLUTIONS AND FISSURES.

The fissure of Sylvius (Fig. 10, SS) commences behind the posterior border of the lesser wing of the sphenoid, and courses upwards and backwards below the greater wing of the sphenoid, where this articulates with the anterior inferior angle of the parietal bone, and then appears in the lower part of the inferior antero-parietal region. The fissure of Rolando (Fig. 10, R) lies in the antero-parietal region, both in its superior and inferior divisions, its upper extremity being as much as two inches and its lower an inch and a half behind the respective ends of the coronal suture. The coronal suture does not, therefore, correspond to the boundary between the frontal and parietal lobes of the brain.

The parieto-occipital fissure (Fig. 10, PO) is situated on an average about 0.7 to 0.8 inch in front of the apex of the lambdoidal suture.

Contents of the Respective Areas.

The *frontal area* is occupied by the frontal lobe, but does not cover the whole of it, the posterior extremities of the three frontal convolutions lying behind the coronal suture. The frontal area, therefore, corresponds to the part of the frontal lobe supplied by the anterior cerebral artery, and which is not excitable to stimulation. The supe-

rior, middle, and inferior frontal areas of the skull correspond respectively to the superior, middle, and inferior frontal convolutions, with the exception of their posterior extremities.

The *upper antero-parietal area* (Fig. 10, SAP) contains the upper two-thirds of the ascending frontal (AP) and ascending parietal (S)

Fig. 10.

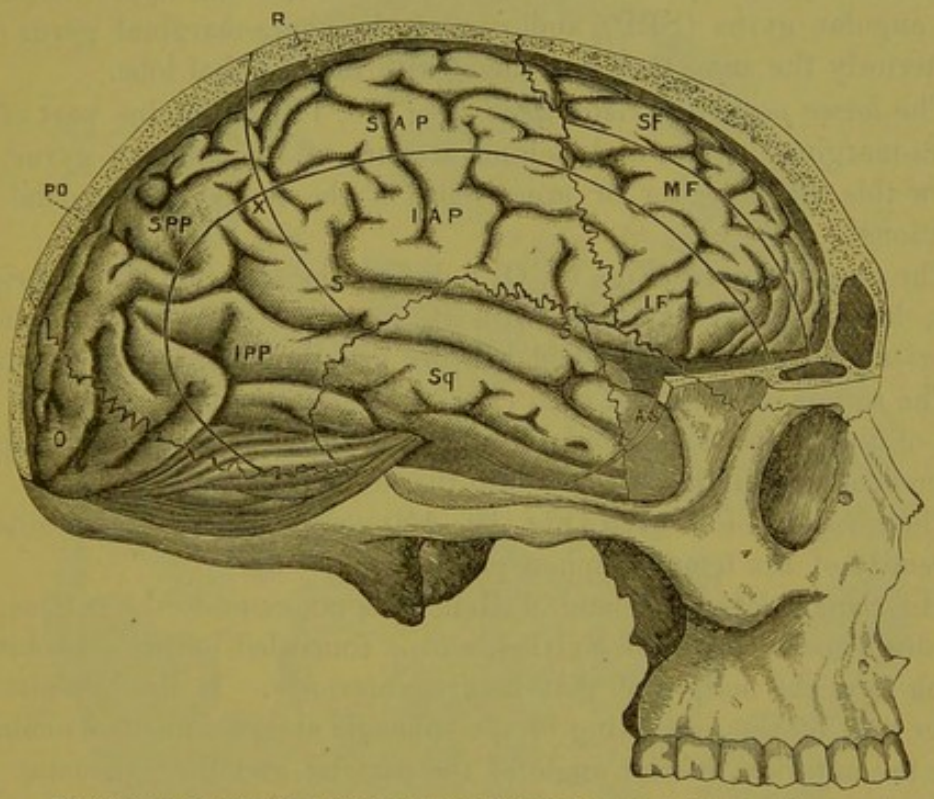


Diagram showing the Relations of the Convolutions to the Skull. (Turner.)

R, The fissure of Rolando, which separates the frontal from the parietal lobe. PO, The parieto-occipital fissure between the parietal and occipital lobes. SS, The fissure of Sylvius, which separates the temporo-sphenoidal from the frontal and parietal lobes. SF, MF, IF, The supero-, mid-, and infero-frontal subdivisions of the frontal area of the skull: the letters are placed on the superior, middle, and inferior frontal convolutions. SAP, The supero-antero parietal area of the skull: S is placed on the ascending parietal convolution, AP on the ascending frontal convolution. IAP, The infero-antero-parietal area of the skull: I is placed on the ascending parietal, AP on the ascending frontal convolution. SPP, The supero-postero-parietal area of the skull: the letters are placed on the angular convolution. IPP, The infero-postero-parietal area of the skull: the letters are placed on the mid-temporo-sphenoidal convolution. ×, The convolution of the parietal eminence, or supra-marginal gyrus. O, The occipital area of the skull: the letter is placed on the mid-occipital convolution. Sq, The squamoso-temporal region of the skull: the letters are placed on the mid-temporo-sphenoidal convolution. AS, The ali-sphenoid region of the skull: the letters are placed on the tip of the supero-temporo-sphenoidal convolution.

convolution, and the posterior extremities of the superior (1.2 in.) and middle frontal (1.3 in.) convolutions. At the upper posterior angle of this area part of the postero-parietal lobule is visible, and below this part of the supra-marginal lobule may appear.

The *lower antero-parietal area* (Fig. 10, IAP) contains the lower

third of the ascending parietal (1 in.) and ascending frontal (AP) convolutions, and the posterior extremities (1 in.) of the inferior frontal convolution. A small portion of the supra-marginal gyrus is visible at the upper posterior angle of this area, and below it a small portion of the superior temporo-sphenoidal convolution.

The *upper postero-parietal area* (Fig. 10, SPP) contains the greater part of the postero-parietal lobule. Below it lies the upper portion of the angular gyrus (SPP), and part of the supra-marginal gyrus (×) Posteriorly the annectant gyri blend with the occipital lobe.

The *lower postero-parietal area* (Fig. 10, IPP) contains part of the supra-marginal gyrus, and behind it part of the angular gyrus, and below this the posterior or upper ends of the temporo-sphenoidal convolutions.

The *occipital area* (Fig. 10, O) indicates the situation of the occipital lobe, but is not coextensive with it, inasmuch as a portion extends anteriorly beyond the lambdoidal suture into the postero-parietal area.

The *squamoso-temporal area* (Fig. 10, Sq) contains the greater portion of the temporo-sphenoidal convolutions, but the superior temporo-sphenoidal convolution ascends into the lower parietal areas.

The *ali-sphenoidal area* (Fig. 10, AS) contains the lower or anterior extremity of the temporo-sphenoidal lobe.

The *central lobe*, or Island of Reil, does not come to the surface, but lies deep in the fissure of Sylvius, and is concealed by the convolutions which form the margin of that fissure anteriorly. It lies opposite the upper part of the great wing of the sphenoid and its line of articulation with the anterior inferior angle of the parietal and the squamous part of the temporal.

The convolutions situated on the internal aspect of the hemisphere are altogether out of relation to the surface of the skull.

The deep-seated position and direction of the hippocampal region are superficially indicated by the convolutions of the temporo-sphenoidal lobes, contained chiefly in the inferior postero-parietal, squamoso-temporal, and ali-sphenoidal areas.

4. *Topography of the Internal Parts of the Cerebrum.*

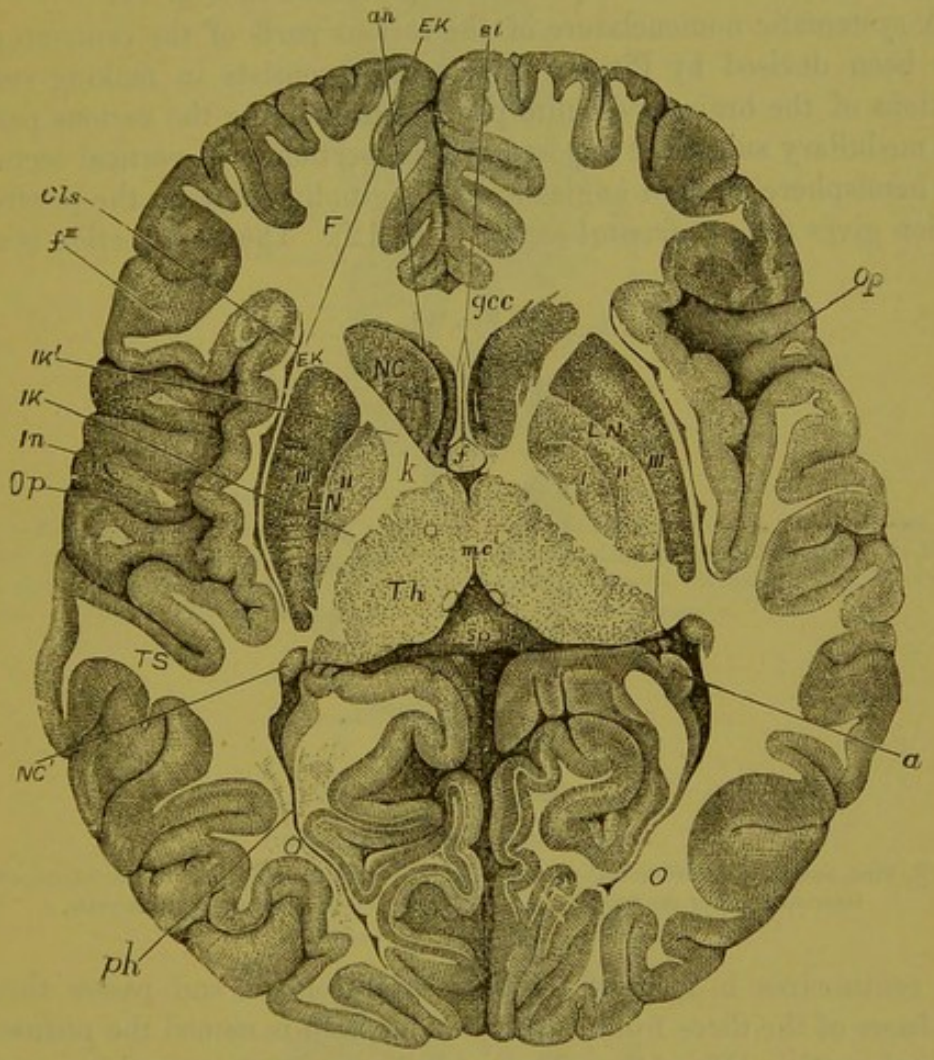
The anatomy of the cerebrum is most conveniently studied by successive horizontal and vertical sections.

Centrum Ovale.

A horizontal section made half an inch above the corpus callosum displays the white matter of each hemisphere surrounded on all sides

by the gray matter of the convolutions. The white central mass in each hemisphere was named by Vicq. d'Azyr the *centrum ovale minus*. A section made at the level of the corpus callosum shows that the white substance of that part is continuous with the central white substance of

FIG. 11.



HORIZONTAL SECTION OF THE BRAIN OF A CHILD NINE MONTHS OF AGE, THE RIGHT SIDE BEING AT A SOMEWHAT LOWER LEVEL THAN THE LEFT HALF. (After FLECHSIG.)

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

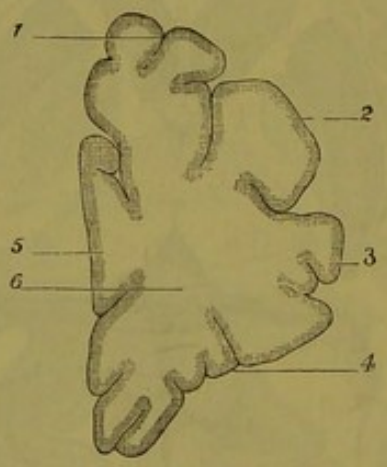
each hemisphere. The large, white medullary mass thus displayed is named the *centrum ovale majus*. The corpus callosum connects the convolutions of one cerebral hemisphere with corresponding points in

the convolutions of the other. It consists of bundles of nerve fibres, almost the whole of which pass transversely between the two hemispheres, although a few fibres run longitudinally on its surface, named the *striæ longitudinales or nerves of Lancisi.*

A horizontal section nearer to the base of the brain reveals the basal ganglia and the internal capsule, as represented in Fig. 11.

A systematic nomenclature of the various parts of the centrum ovale has been devised by Pitres. His system consists in making vertical sections of the brain at definite points, and naming the various parts of the medullary substance exposed in each section. A vertical section of the hemisphere at right angles to its longitudinal axis in the præfrontal region gives the *præfrontal section* (Fig. 12). The next section is made

FIG. 12.



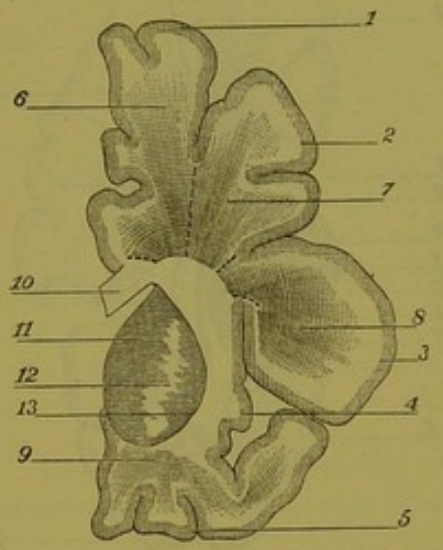
PRÆFRONTAL SECTION. (After PITRES.)

1, 2, 3, First, second, and third frontal convolutions. 4, Orbital convolutions. 5, Convolutions on the internal aspect of the frontal lobe. 6, Præfrontal fasciculi of the centrum ovale.

two centimetres in front of the fissure of Rolando and passes through the bases of the three frontal convolutions, and is named the *pedunculo-frontal section* (Fig. 13). The medullary substance in this section is subdivided into a *superior*, *middle*, and an *inferior pedunculo-frontal fasciculus*, corresponding with the respective frontal convolutions. The next section is made through the ascending frontal convolution, parallel with the fissure of Rolando, and is named the *frontal section.* It also passes through a small portion of the sphenoidal lobe. The medullary substance of this section is also subdivided into *superior*, *middle*, and *inferior frontal fasciculi* (Fig. 14). The fourth section is carried through the ascending parietal convolution, and is named the *parietal section.* It is subdivided into *superior*, *middle*, and *inferior*

parietal fasciculi (Fig. 15). The next is the *pedunculo-parietal section*, made by dividing the hemisphere three centimetres behind the

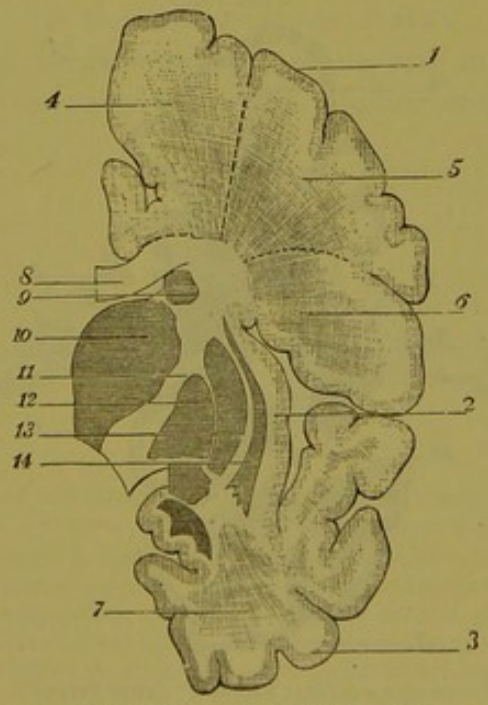
FIG. 13.



PEDUNCULO-FRONTAL SECTION. (After PITRES.)

1, 2, 3, First, second, and third frontal convolutions. 4, Anterior extremity of the insular lobe. 5, Posterior extremity of the orbital convolutions. 6, Superior pedunculo-frontal fasciculus. 7, Middle pedunculo-frontal fasciculus. 8, Inferior pedunculo-frontal fasciculus. 9, Orbital fasciculus. 10, Corpus callosum. 11, Caudate nucleus. 12, Internal capsule. 13, Lenticular nucleus.

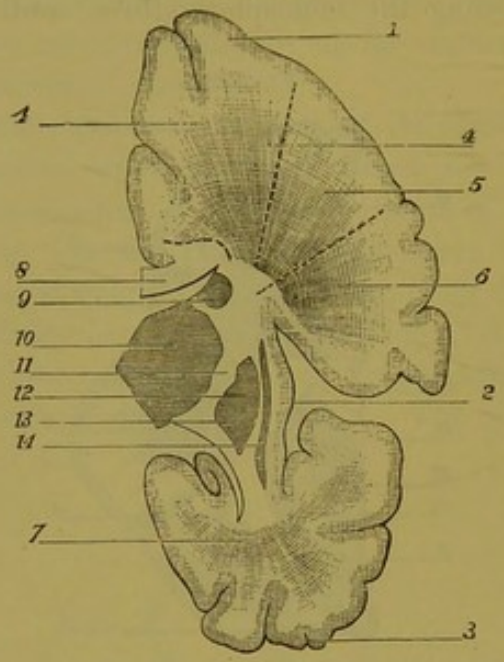
FIG. 14.



FRONTAL SECTION (After PITRES.)

1, Ascending frontal convolution. 2, Insular lobule. 3, Sphenoidal lobe. 4, 5, 6, Superior, middle, and inferior frontal fasciculus. 7, Sphenoidal fasciculus. 8, Corpus callosum. 9, Caudate nucleus. 10, Optic thalamus. 11, Internal capsule. 12, Lenticular nucleus. 13, External capsule. 14, Claustrum.

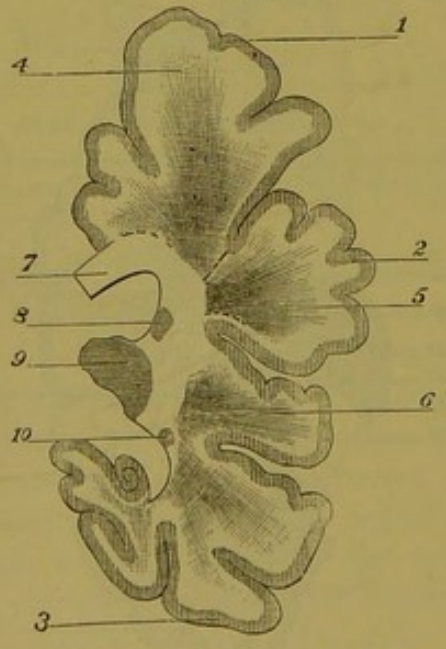
FIG. 15.



PARIETAL SECTION. (After PITRES.)

1, Ascending parietal convolution. 2, Insular lobe. 3, Sphenoidal lobe. 4, Superior parietal fasciculus. 5, Middle parietal fasciculus. 6, Inferior parietal fasciculus. 7, Sphenoidal fasciculus. 8, 9, 10, 11, 12, 13, 14, as in the preceding figure.

FIG. 16.



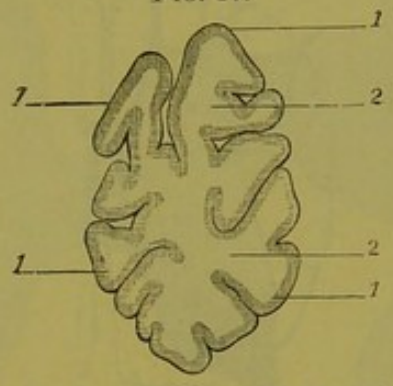
PEDUNCULO-PARIETAL SECTION. (After PITRES.)

1, Superior parietal lobule. 2, Inferior parietal lobule. 3, Sphenoidal lobe. 4, Superior pedunculo-parietal fasciculus. 5, Inferior pedunculo-parietal fasciculus 6, Sphenoidal fasciculus. 7, Corpus callosum. 8 and 10, Caudate nucleus. 9, Optic thalamus.

fissure of Rolando, and cutting the superior and inferior parietal lobules. It is subdivided into *superior* and *inferior* pedunculo-parietal and *sphenoidal fasciculi* (Fig. 16).

The last is the *occipital section* (Fig. 17), in which no separate fasciculi are distinguished.

FIG. 17.



OCCIPITAL SECTION. (After PITRES)

1, Occipital convolutions. 2, Occipital fasciculi of the centrum ovale.

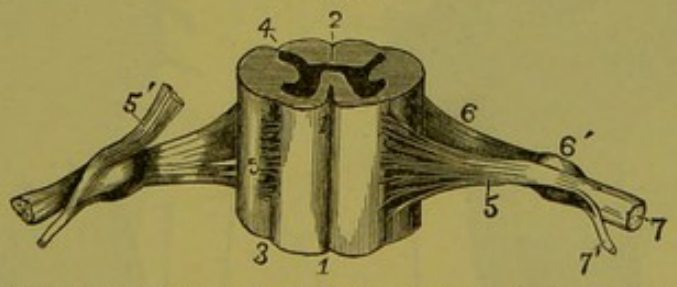
5. *The Internal Structure of the Encephalo-spino-neural System.*

The internal structure of the encephalo-spino-neural system must now be briefly described, and for this purpose we shall divide the system into (1) the spino-neural, and (2) the encephalo-spinal systems.

(1) THE SPINO-NEURAL SYSTEM.

A spinal segment consists of a disk of nervous matter to each lateral half of which a nerve is attached by an anterior and posterior root, the

FIG. 18.



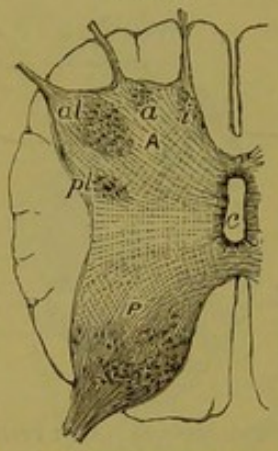
THE ANTERIOR SURFACE OF THE SPINAL SEGMENT, THE ANTERIOR ROOT OF THE RIGHT SIDE BEING DIVIDED. (After ALLEN THOMSON, from QUAIN.)

1, The anterior median fissure; 2, Posterior median fissure; 3, Anterior lateral depression, over which the anterior nerve roots are seen to spread; 4, Posterior lateral groove into which the posterior roots are seen to sink; 5, Anterior roots passing the ganglion; 5', The anterior root divided; 6, The posterior roots, the fibres of which pass into the ganglion, 6'; 7, The united or compound nerve; 7', The posterior primary branch seems to be derived in part from the anterior and in part from the posterior root.

latter being furnished with a ganglion (Fig. 18). The gray substance which represents the gangliated structure occupies the central parts of

the cord in the well-known shape of the letter H. The median part of the gray substance contains the central canal and the central gray nucleus of Kölliker, the anterior gray and white commissure lying in

FIG. 19.



SECTION FROM THE MIDDLE OF THE CERVICAL ENLARGEMENT OF THE SPINAL CORD AT THE THIRD MONTH OF EMBRYONIC LIFE.

C, Central canal. The other letters indicate the same as the corresponding letters in Fig 1.

FIG. 20

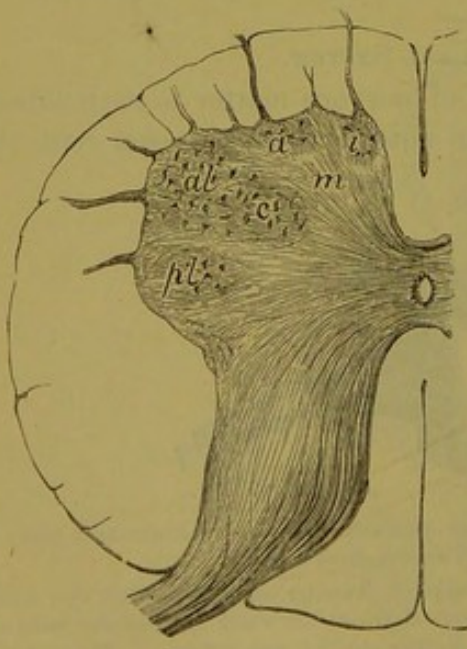
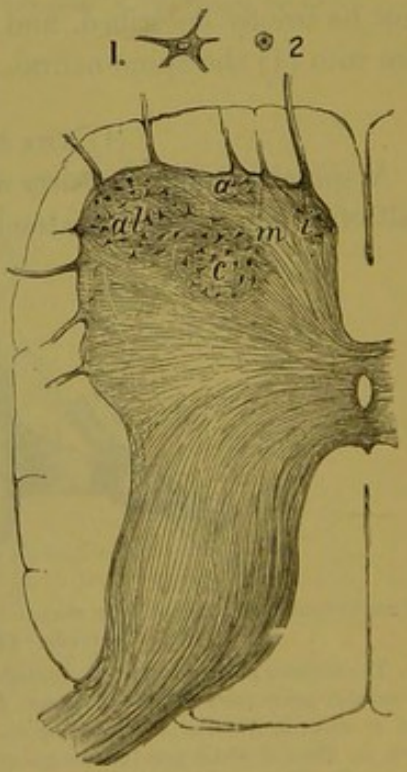


FIG. 21.



SECTIONS OF SPINAL CORD OF A FIVE-MONTHS HUMAN EMBRYO, FROM THE MIDDLE OF THE CERVICAL AND LUMBAR ENLARGEMENTS RESPECTIVELY. (YOUNG.)

i, internal; *a*, anterior; *al*, antero-lateral; *pl*, postero-lateral, *c*, central, and *m*, median groups of ganglion cells: 1, ganglion cell of the centre of the antero-lateral group; 2, ganglion cell of the median group.

FIG. 22.

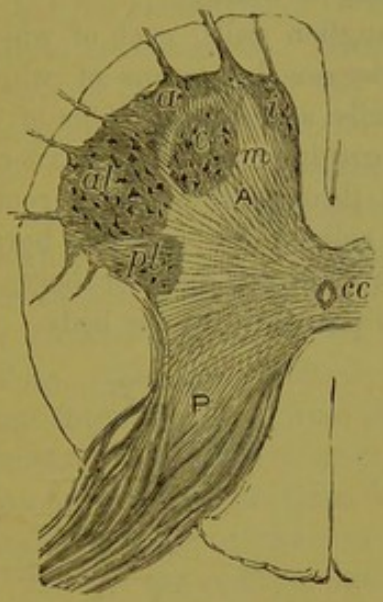
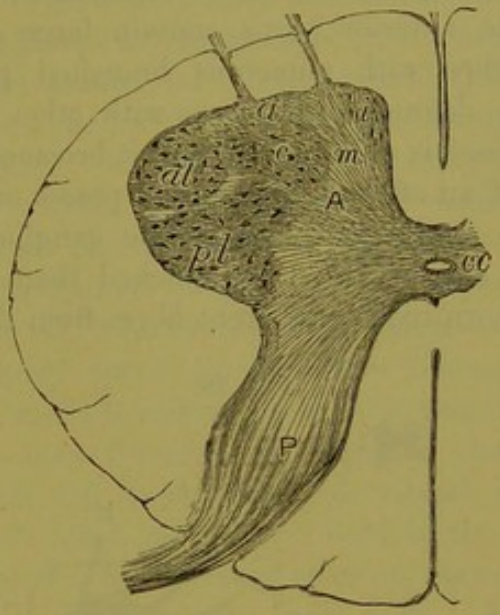


FIG. 23.



SECTIONS OF SPINAL CORD OF A NINE-MONTHS HUMAN EMBRYO, FROM THE MIDDLE OF THE LUMBAR AND CERVICAL ENLARGEMENTS RESPECTIVELY. (YOUNG.)

A, anterior, and P, posterior horns. The small letters indicate the same as in Figs. 20 and 21. The normal size of the section from which the drawing was made is shown above each figure.

FIG. 24.

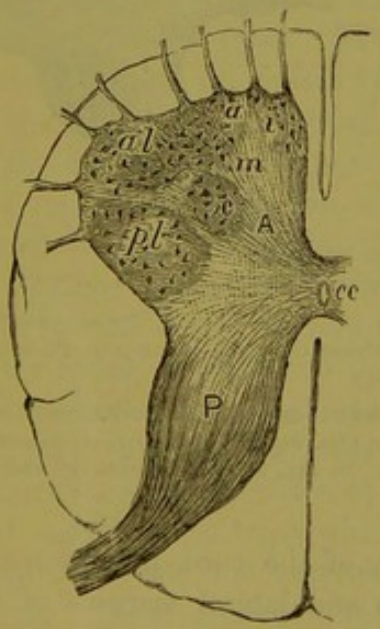
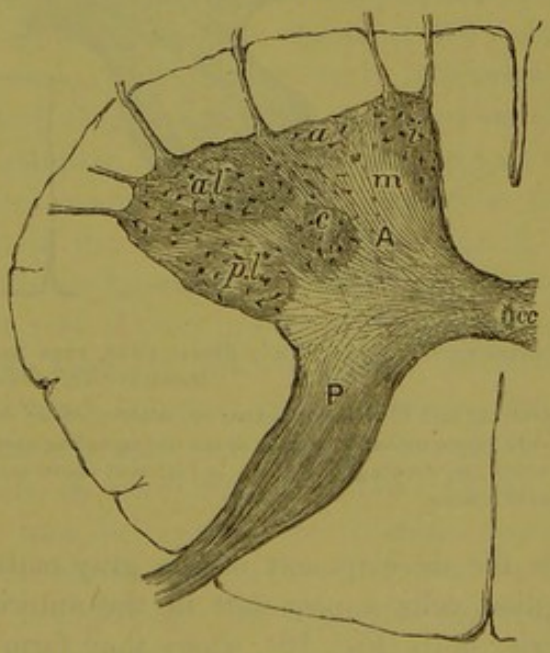


FIG. 25.



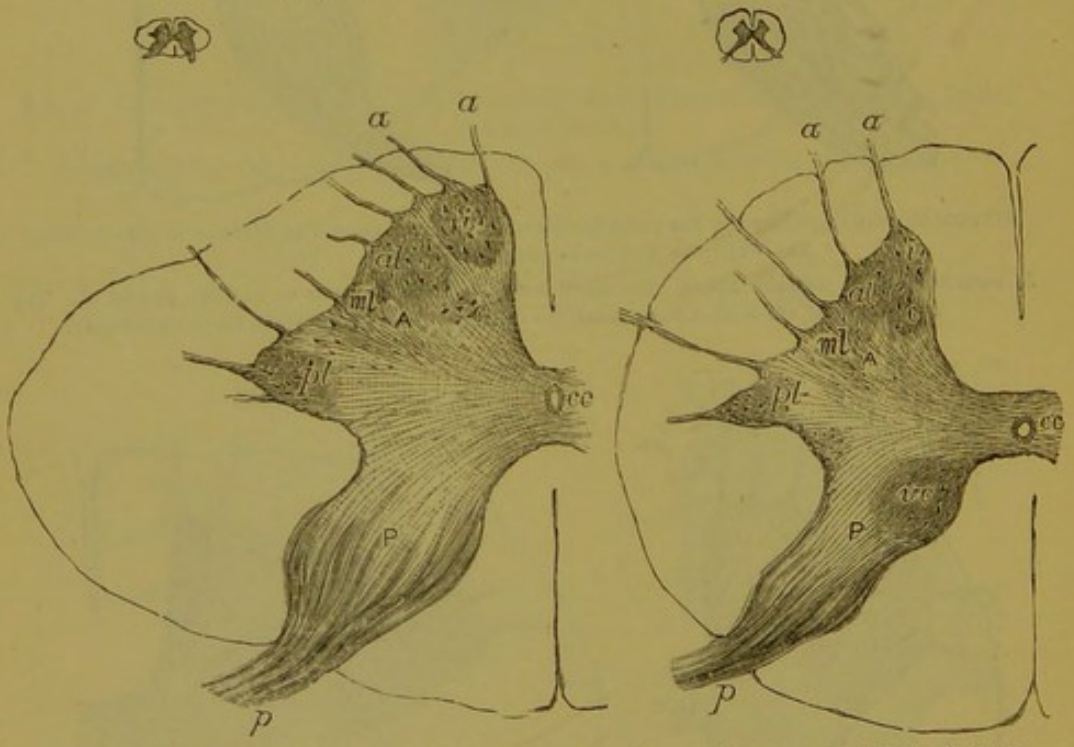
SECTIONS OF THE ADULT SPINAL CORD FROM THE MIDDLE OF THE LUMBAR AND CERVICAL ENLARGEMENTS RESPECTIVELY. (YOUNG.)

The letters indicate the same as those in Figs. 20 and 21.

front, and the posterior commissure behind it. The lateral parts or columns consist of an anterior, middle, and posterior part, the first of them representing the *anterior*, and the last the *posterior* gray horn, and the middle chiefly the vesicular column of Clarke.

The anterior horns contain large ganglion cells, each of which is furnished with numerous branched processes by means of which it forms definite connections with other cells and fibres. One of these processes is unbranched, and it becomes continuous with the axis-cylinder of an efferent fibre which passes out through the anterior root and nerve to the periphery. The ganglion cells of the posterior horn are much smaller and less branched than those of the anterior horns, and this horn receives afferent fibres from the periphery of the body.

FIG. 26. FIG. 27.



SECTIONS OF THE ADULT HUMAN SPINAL CORD, FROM THE UPPER CERVICAL AND DORSAL REGIONS RESPECTIVELY. (YOUNG.)

A, anterior, and P, posterior horns; *aa*, anterior roots; *cc*, central canal; *ml*, the medio-lateral area. The other letters indicate the same as the corresponding ones in Figs. 20 and 21 The size of the sections from which the drawing was taken is indicated above each. In Fig. 27, *vc* represents the vesicular column of Clarke.

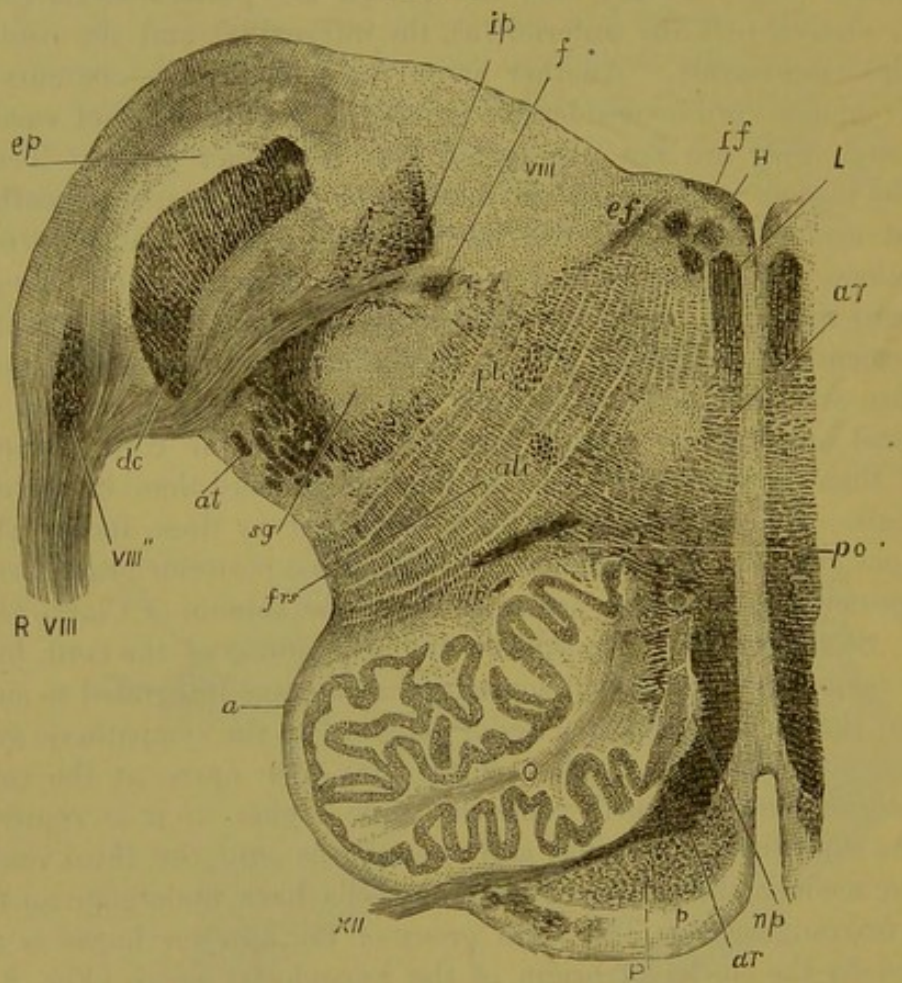
In the development of the gray matter of the cord, the groups of ganglion cells appear first in the anterior and lateral margins of the anterior horn (Fig. 19), where they form several distinct groups, which are as far as possible removed from the central canal. The gray matter surrounding the central canal represents the growing or embryonic por-

tion of the gray matter, and every new ganglion cell which develops becomes superadded at the margins of the first forward groups. The groups of ganglion cells are pretty constant for the same portions of the cord, but the arrangement varies considerably when sections at different elevations are compared. These groups are represented in the annexed diagrams (Figs. 20–27), and are named the postero-lateral (*pl*), the antero-lateral (*al*), the anterior (*a*), the internal (*i*), and the central (*c*) groups respectively. Another area—the median (*m*)—contains in the adult human cord a considerable number of caudate cells of small size, although they are not aggregated into a distinct group. The cells of the median area, and of the margins of the groups, representing the latest evolved structures, will be the counterpart of the latest evolved functions. The last evolved purely spinal function (not taking into account those regulated from the medulla oblongata) are the complicated movements of the hand, and it may therefore be expected that the median area and the marginal cells will be more fully developed in the cervical enlargement than in any other part of the cord, in the adult cord than in the embryo, and in the human cord than in the cord of animals. This expectation is fully borne out by these facts. A very distinct grouping of ganglionic cells lies in the posterior gray horn near the posterior commissure, named the vesicular column of Clarke (Fig. 1, *vc*). So far we have only spoken of one segment of the cord, but the gray matter of the different segments has become integrated to such an extent that it forms, not a series of ganglia like the sympathetic system, but a continuous tube. The central gray tube opens at the calamus scriptorius into the fourth ventricle, while higher up it is represented by the aqueduct of Sylvius, and then opens into the third ventricle. In the medulla the groups of ganglion cells have undergone an extensive rearrangement. The main group of the anterior horns is represented by the nuclei of origin of the hypoglossal nerve (Fig. 2, *i*, *a*, *al*, *pl*), but a portion of the antero-lateral and postero-lateral groups have become detached from the rest of their groups, and are aggregated in the formatio reticularis in two tolerably distinct groups, the anterior and posterior nuclei of the lateral column of the medulla (Fig. 2, *alc*, *plc*). The median area and the marginal cells are represented by a much larger aggregation of cells than anything to be found in the cord, and constitute groups which may be named the internal accessory (Fig. 2, *if*) and the external accessory nuclei (Fig. 1, *ef*) of the facial nerve. These groups of cells are probably the structural counterparts of mimetic facial and articulatory movements.

The *vesicular column* of Clarke is only represented in the dorsal region of the spinal cord. It contains bipolar ganglion cells, and cells

of similar character are to be found in the nuclei of the vagus (Fig. 2, *nv*) and glosso-pharyngeal nerves, and it is therefore probable that the nuclei of these nerves are, to some extent at least, the representatives of the vesicular column of Clarke in the medulla.

FIG. 28.



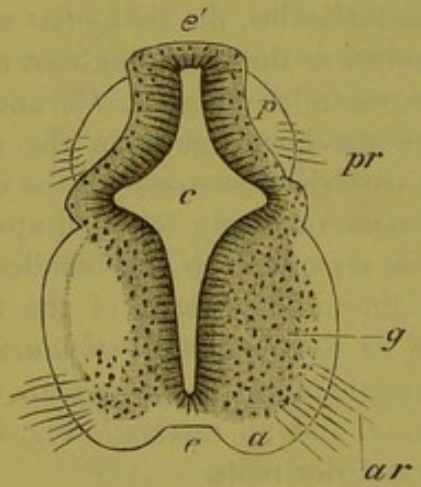
SECTION OF THE MEDULLA OBLONGATA ON A LEVEL WITH THE SUPERFICIAL ORIGIN OF THE ACOUSTIC NERVE. (Modified from FLECHSIG.)

R VIII, Root of the acoustic nerve.
VIII, Posterior median acoustic nucleus
VIII'', Posterior lateral acoustic nucleus.
H, Nucleus of the hypoglossal nerve.
ip, Internal division of the inferior peduncle of the cerebellum.
ep, External division of the inferior peduncle of the cerebellum.
frs, Formatio reticularis.
a, Arciform fibres.

The posterior gray horn is represented in the medulla by the substantia gelatinosa (Fig. 2, *sg*). In addition to the sensory nuclei of common sensation represented by the substantia gelatinosa, the medulla oblongata contains special sensory nuclei. It is probable that part of

the nuclei of the glosso-pharyngeal nerve, and probably also of the fifth nerve, represent the special nerves of taste; four nuclei, namely, the posterior median (Fig. 28, VIII), the posterior lateral the anterior median, and the anterior lateral acoustic nuclei, represent the auditory nerve; while the corpora quadrigemina and the internal geniculate body represent the nuclei of origin of the optic nerve. The *spinal ganglia*, situated in the posterior roots of each pair of nerves, belong to the spinal system. The casserian ganglion is the representative of these ganglia in the cranial end of the spinal system. The *middle sensory nucleus of the trigeminus* is also in structure very similar to the ganglia of the posterior roots, and it also is probably the homologue of the spinal ganglia. The *external geniculate body* likewise contains cells which are very like those of the ganglia of the posterior roots, and it must possibly be regarded as a ganglion for the optic nerve homologous with the spinal ganglia.

FIG. 29.



TRANSVERSE SECTION OF THE CERVICAL PART OF THE SPINAL CORD OF A HUMAN EMBRYO OF SIX WEEKS. (From KÖLLIKER.)

c, Central canal; *e*, *e'*, Its epithelial lining; *g*, Gray substance; *ar*, Anterior roots; *pr*, Posterior roots; *a*, Anterior root-zones; *p*, Posterior root-zones.

The *white* substance of a spinal segment is divided anatomically by the anterior and posterior median fissure, and the nerve roots into an anterior, lateral, and posterior column for each lateral half. A small portion only of this white substance really belongs to the spinal system, the remainder representing conducting paths between the portions of the cord below, or posterior to the segment examined, and the encephalic ganglia. This purely spinal portion of the white substance consists of the portions named the *anterior* and *posterior root-zones*. As might be expected, these areas are the first portions of the white substance to

appear in the development of the cord, and they are consequently least distinguished at an early period of embryonic life. During the first weeks of embryonic life the cord consists entirely of gray matter in the form of a tube, which, by the appearance of lateral slits, is imperfectly divided into anterior and posterior portions. Toward the end of the first month a zone of white substance appears on the surface of the anterior and posterior portions of the gray tube, and there constitute respectively the anterior and posterior root-zones (Fig. 29, *a*, *p*). These zones are supposed to consist of short looped fibres, the posterior one coördinating the functions of the gray substance on the sensory or ingoing side, and the anterior on the motor or outgoing side.

The most important fact with regard to the distribution of these zones is that, at any level of the cord, they correspond in size to that of the gray substance, these zones being, like the gray substance, of comparatively large size in the lumbar and cervical enlargements.

The *posterior root-zone* (Fig. 2, *pr*), on being followed upwards into the medulla oblongata, will be found to terminate in the gray nucleus of the cuneate fasciculus, the triangular nucleus; but the tracts of white substance known as the ascending root of the fifth nerve (Fig. 2, *at*), the fasciculus rotundus (Fig. 2, *f*), and the descending root of the fifth nerve are the homologues, in the cranial portion of the spinal system, of the posterior root-zones of the cord.

The *anterior root-zone*, on being followed upwards into the medulla oblongata, is seen to be separated up into bundles by the arcuate fibres of the medulla, and the interlacement of the two systems of fibres forms what is known as the *formatio reticularis* (Fig. 28, *frs*). The anterior root-zone consists of two portions: an *internal* (Fig. 37, *ar*), which lies to the inner, and an *external* (Fig. 37, *ar'*), which lies to the outer side of the anterior roots.

The *internal* portion of the anterior root-zone is pushed aside in the lower part of the medulla by the decussating fibres of the pyramidal tracts; but above the level of the decussation it is thrust backwards behind the anterior pyramid of the medulla, and is situated between the median raphé and the fibres of the hypoglossal nerve (Fig. 2, *ar*) In the spinal cord the internal portion of the anterior root-zone maintains a close relationship with the internal group of ganglion cells, and this portion of the root-zone is most probably represented in the medulla oblongata, pons, and crura cerebri by the bundle of fibres named the *posterior longitudinal fasciculus* (Fig. 2, *ar*). This fasciculus is said by Wernicke to be connected with the first division of the lenticular nucleus by the fibres which form the anterior portion of the collar of the crus cerebri.

of the anterior root-zone of the spinal cord is
the *formatio reticularis* of the medulla oblon-
his portion lies behind the olivary body, and
f the medulla in its lateral column; while it is
he root-fibres of the anterior motor nerves, ex-
res of the nerves of the lateral mixed system,
y matter. The interlacing fibres of the pons
front of this portion (*ar'*), and in the crus it
wards, so that it is only separated from the crusta
. 46, *ar'*).

ortion of the anterior root-zone which lies next
ior surfaces of the olivary body in the medulla,
fibres of the pons, is named the fillet. It is
l and an external portion corresponding to the
ivisions of the anterior root-zone. The internal
er side of the olivary body, a part of it even
t body, and between it and the anterior pyramid
the line of the olivary body this last portion is
s to join the external portion, and both divisions
ansverse fibres of the pons. The fibres of the
fillet seem to pass upwards to become connected
s, while those of the external division (Fig. 36,
an outer and inner bundle, the former of which
erior, and the latter in the anterior tubercle of
na.

HE ENCEPHALO-SPINAL SYSTEM.

l system consists of *a*, the gray substance of the
ay substance of the cerebrum; and *c*, the con-
nnect these gray masses with one another and
ube.

a. THE GRAY SUBSTANCE OF THE CEREBRUM.

(1) *The Cortex Cerebri.*—By far the greater portion of the gray substance of the cerebrum is disposed up the surface of the cerebral hemispheres. The cortical substance consists of cells and fibres embedded in a matrix similar to the neuroglia of the spinal cord.

The *cells* are of various forms, the most usual forms being spherical, stellate, pyramidal, and fusiform. The *fibres* radiate into the gray

Fig. 30.

Transparent Section of a Furrow of the Third Cerebral Convolution of Man. Magnified 100 decimetres. (After Meynert.)

1, Layer of the scattered small cortical corpuscles; 2, Layer of close-set, small pyramidal corpuscles; 3, Layer of large pyramidal cortical corpuscles (formation of the cornu Ammonis); 4, Layer of small, close-set, irregular-shaped cortical corpuscles (granule-like formation); 5, Layer of fusiform cortical corpuscles (claustral formation); *m*, the medullary lamina.

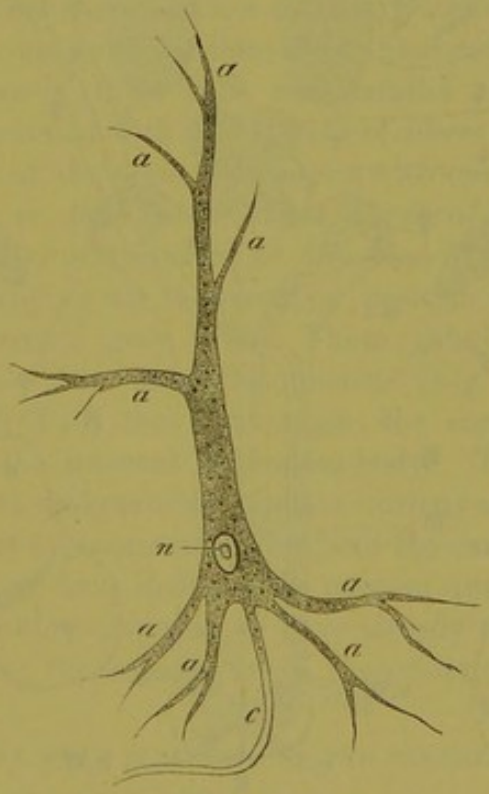
cortex from the white centre of each convolution, their course being vertical to the free surface of the convolution. They are arranged in bundles as they pass through the gray substance, and this gives to the nerve-cells a columnar arrangement. The radiating fibres are wanting in the sulci between the convolutions, but the internal layer of the gray substance of the cortex generally contains fibres which pursue an arciform course and connect adjacent convolutions. Fibres pass in all directions through the gray substance connecting its several layers, and forming a dense network, like that described by Gerlach in the spinal cord.

Layers of the Cortex.—The cortex of the cerebrum is divided into several layers, each of which possesses a definite histological character. The most commonly distributed form of structure is what Meynert has called the "five laminated type." The external layer consists of neuroglia and a layer of delicate nerve-tubes, along with a few scattered small nerve-cells which are destitute of processes. The next layer is composed of small angular or pyramidal nerve-cells with branching processes. The third layer contains large and small pyramidal cells with branching processes, arranged with their pointed extremities towards the surface of the convolutions, and separated into groups by bundles of the radiating fibres. In the innermost portion of this layer the pyramidal cells are larger than in the remaining portions, and it has therefore been described as a separate layer by Dr. Lockhart Clarke. In the cortex of the occipital lobe the

deeper cells of the third layer are pyramidal in form, with their bases turned inwards towards the medullary substance, but their basal processes are directed laterally so as to connect adjacent cells, and none of them appear to be directed inwards to connect the cells with the fibres of the medullary substance. In the anterior portion of the frontal convolutions the disposition of these cells is somewhat similar, but a distinct basal process has occasionally been observed, which is directed towards the medullary substance of the convolution, and which afterwards becomes continuous with one of the fibres of the centrum ovale.

In the central convolutions of the brain, Betz and Mierzejewski have discovered cells which are two or three times the size of the pyra-

Fig. 31.



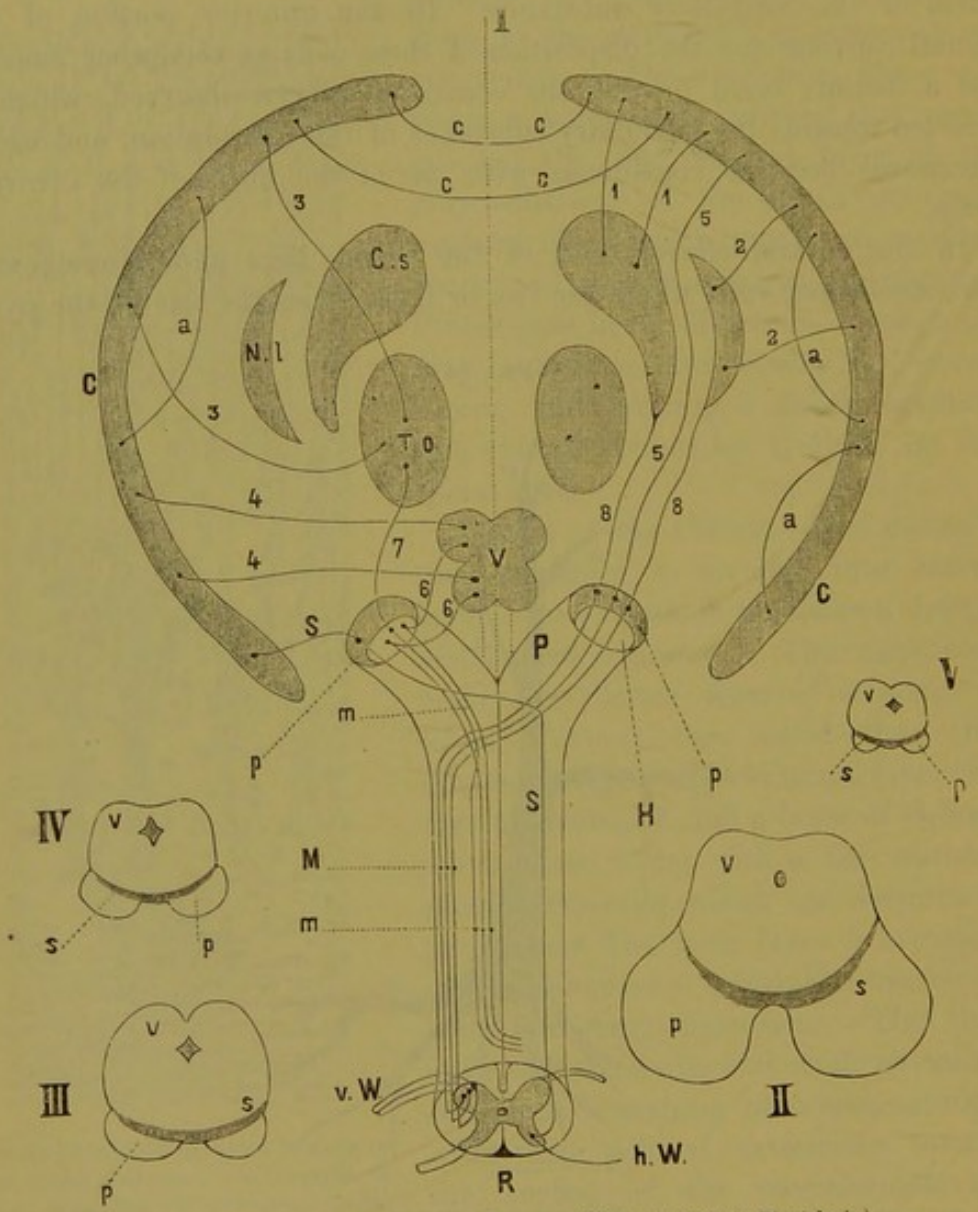
Pyramidal Giant-cell.

n, Nucleus; *a*, *a*, *a*, Branched processes; *c*, Unbranched basal process.

midal cells of the other regions of the cortex, and they have consequently named them *giant cells*. In addition to the branched protoplasmic processes (Fig. 31, *a*, *a*) which connect neighboring cells with one another, these cells possess a distinct axis-cylinder process (Fig. 31, *c*). The latter is always unbranched, and after becoming surrounded by a medullary sheath it forms the axis-cylinder of a nerve-fibre of the

centrum ovale. Giant cells have been observed in the paracentral lobule and in a portion of the postero-parietal, as well as in the ascend-

FIG. 32.



SCHEMA OF THE CEREBRO-SPINAL SYSTEM. (From LANDOIS'S *Physiologie.*)

C, C, Cortex of the Brain; C *s*, Corpus Striatum; N *l*, Lenticular Nucleus; T o, Optic Thalamus; V, Corpora Quadrigemina; P, Crura Cerebri; H, Tegmentum; *p*, Crusta; 1 1, Radiate Fibres of the Corpus Striatum; 2 2, those of the Lenticular Nucleus; 3 3, those of the Optic Thalamus; 4 4, those of the Corpora Quadrigemina and Tegmentum; 5 5, the Pyramidal Tract; 6 6, Fibres connecting the Corpora Quadrigemina and Tegmentum; *m*, their further course; 8 8, Fibres connecting the Corpus Striatum and Lenticular Nucleus with the Crusta; M, their further course; S S, course of the Sensory Fibres, R, Transverse Section of the Spinal Cord; *v* W, Anterior, and *h* W, Posterior Roots of the Nerves. *a a*, associating Fibres; *c c*, Commissural Fibres. II, Transverse Section through the Crura Cerebri of Man on a level with the posterior pair of the Corpora Quadrigemina (after MEYNERT); *p*, Crusta; S, Locus Niger; *v*, the posterior pair of the Corpora Quadrigemina with the Aqueduct of Sylvius. Similar sections from the Crura of—III, Dog; IV, Monkey; V, Guinea-pig.

ing frontal and parietal convolutions, and posterior extremities of the three frontal gyri. These cells are disposed in groups, and correspond in position to the motor centres of physiologists. The giant cells vary greatly in size, the largest being found, as we have already seen, in the paracentral lobule, which may be regarded as the upper extremity of the ascending frontal and parietal convolutions. Large pyramidal cells are also met with in the upper part of the ascending frontal convolutions, and Dr. Bevan Lewis has found that they diminish in size from the upper extremity until at the lower extremity they are but half the size. The pyramidal cells of the posterior extremities of the frontal convolutions are on the whole smaller than those of the ascending frontal, and the cells also diminish from above downwards, those in Broca's convolution being the smallest.

The fourth layer consists of closely set angular corpuscles with fine processes, placed irregularly and not distinctly separated into groups.

The fifth layer consists of medium sized, fusiform, and bipolar cells. The long diameters of these cells run parallel to the layers of the cortex, and are associated with the system of fibres which connects different convolutions of the same hemisphere with one another.

(2) *The Basal or the Intermediate Cerebral Ganglia.*—Several masses of gray matter are situated at the base of the cerebral hemispheres, there occupying an intermediate position between the cortex cerebri and the central gray tube. These ganglia are the caudate nucleus (Fig. 32, C *s*), the lenticular nucleus (Fig. 32, N *l*), the optic thalamus (Fig. 32, T o), the locus niger, the corpora quadrigemina (Fig. 32, V), and the internal geniculate body. The corpus striatum is sometimes used to designate the caudate nucleus alone, while at other times it is used as a generic term for both the caudate and lenticular nuclei. Although we have included the corpora quadrigemina amongst the intermediate cerebral ganglia, we have already seen some reason to regard them as being the homologues of the posterior gray horns of the spinal cord.

b. THE GRAY SUBSTANCE OF THE CEREBELLUM.

(1) *The Cortex Cerebelli.*—The greater portion of the gray substance of the cerebellum is also disposed on the surface, where it forms numerous *laminæ* or *folia*, which are separated by *fissures* or *sulci* of different depths.

(2) *The Intermediate Cerebellar Ganglia.*—Occupying the central part of the lateral lobe of the cerebellum is a folded mass of gray matter named the *corpus dentatum;* and Stilling has described two small masses of gray matter which are situated in the anterior end of the inferior vermiform process, and which he named *roof nuclei.*

These, then, are the only masses of gray matter which are to be found in the cerebellum itself; but the inferior, middle, and superior peduncles are connected with other aggregations of gray substance in the medulla oblongata, pons, and crura cerebri, and these must also be regarded as belonging most probably to the cerebellar system. The chief of these masses of gray matter are the *olivary bodies*, which are connected with the inferior peduncle of the cerebellum of the opposite side (the late researches of Flechsig appear to cast some doubt upon this statement); the *red nucleus* of the tegmentum, which is connected with the superior peduncle of the cerebellum of the opposite side; and the gray matter interposed between the transverse fibres of the pons, and which is connected with the middle peduncle of the cerebellum also of the opposite side. The *parolivary body*, the *superior olivary body*, and the *nucleus of the pyramid* are other small aggregations of gray matter having a structure somewhat similar to that of the corpus dentatum of the cerebellum, the olivary body, and the red nucleus of the tegmentum, but their connections are not yet made out. In addition to these are, in the lateral and posterior part of the medulla oblongata, the *triangular nucleus*, in which the posterior root-zones, and the *clavate nucleus*, in which the column of Goll, to be immediately described, terminate.

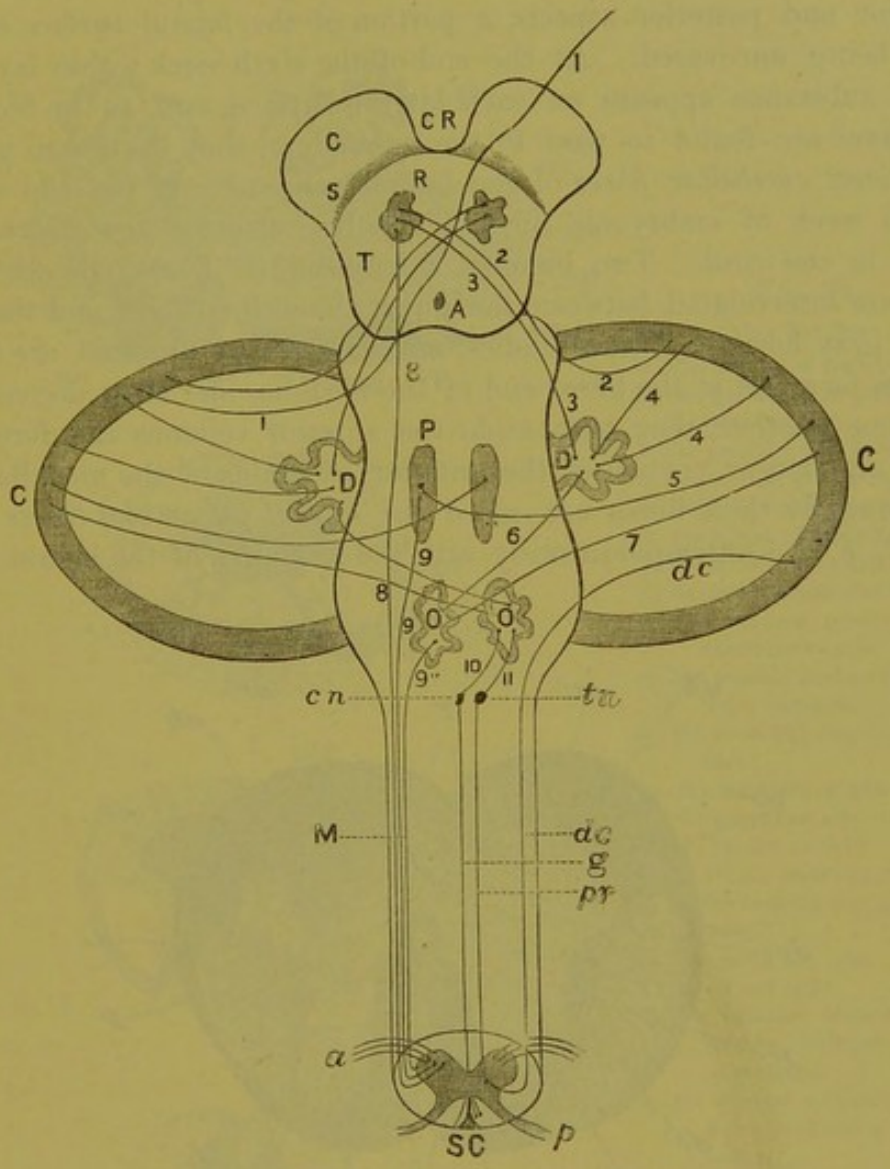
c. THE ENCEPHALO-SPINAL CONDUCTING PATHS.

The purely spinal portion of the spinal cord consists, as we have seen, of the central gray tube, with its commissures and attached nerves, and the anterior and posterior root-zones. Now these portions of the cord form more or less direct connections with the encephalic ganglia. The central gray tube is directly connected with the optic thalamus by means of the gray matter which lines the third ventricle. The anterior root-zone is connected with the lenticular nucleus by means of the posterior longitudinal fasciculus, with the optic thalamus by means of the internal portion of the fillet, and with the corpora quadrigemina through the external portion of the fillet. The greater portion of the posterior root-zone terminates in the triangular nucleus (Fig. 33, *tn*) of the medulla oblongata, and this nucleus is connected by means of arcuate fibres (Fig. 33, 11) with the olivary body (Fig. 33, O) of the same side, which in its turn is connected with the inferior peduncle of the cerebellum of the opposite side.

We must now pass on to describe the special conducting paths which connect the central gray tube with the higher ganglia, and a knowledge of these is best obtained by a study of the development of the cord.

At the end of the first month of embryonic life the spinal cord con-

FIG. 33.

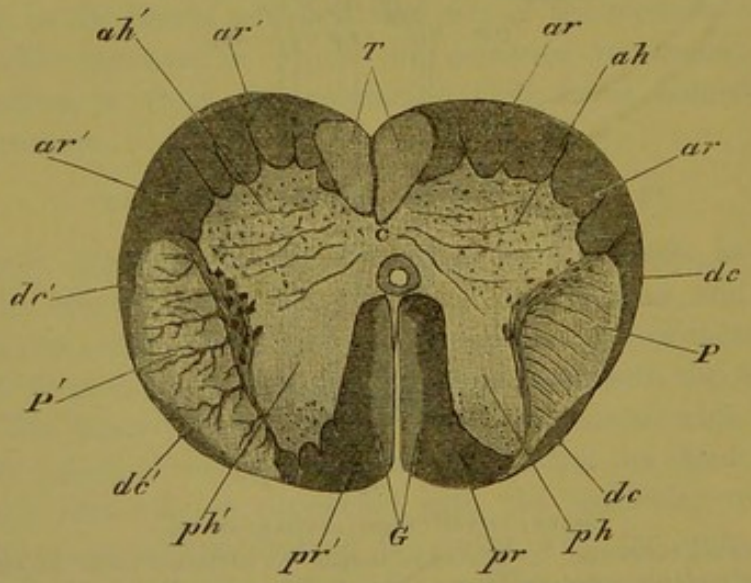


SCHEMA OF THE CEREBELLO-SPINAL SYSTEM.

C, C, Cortex of the Cerebellum. D, D, Corpora Dentata. O, O, Olivary Bodies. R, Red Nucleus of Tegmentum P, Gray matter interposed between transverse Fibres of the Pons. CR, Crura Cerebri. C, Crusta. S, Substantia Nigra. T, Tegmentum. A, Aqueduct of Sylvius. 1, Fibres which connect the cortex of the Cerebrum and that of the Cerebellum on the opposite side. 2, 2, Fibres connecting the Cortex of Cerebellum and Red Nucleus of the opposite side. 3, 3, Fibres connecting the Corpus Dentatum of the Cerebellum with the Red Nucleus of the opposite side. 4, 4, Fibres connecting the Cortex of the Cerebellum with the Corpus Dentatum. 5, Fibres connecting the Cortex of the Cerebellum with Gray substance interposed between the transverse Fibres of the Pons on the opposite side. 6, Fibres connecting the Corpus Dentatum with the Olivary body of the opposite side. 7, Fibres connecting the Cortex of the Cerebellum with the Olivary body of the opposite side. 8, 8, Fibres connecting the Red Nucleus. 9, 9, those connecting the interposed Gray substance of the Pons, and 9'', those connecting the Olivary body respectively with the anterior Gray Horn of the Spinal Cord. M, The Anterior Column of the cord through which the fibres pass. *g*, Column of Goll terminating in *cn* the Clavate Nucleus 10, Arcuate Fibres connecting the Clavate Nucleus with the Olivary body of the same side. *p r*, The Posterior Root-zone terminating in *tn*, the Triangular Nucleus 11, Arcuate Fibres connecting Triangular Nucleus and Olivary body of same side. *dc*, *dc*, Direct Cerebellar Fibres ascending in the lateral column of the cord and connecting the vesicular column of Clarke with the Cortex of the Cerebellum.

sists of a tube of gray matter with a layer of white substance on its anterior and posterior aspects, a portion of the lateral surface of the tube being uncovered. At the end of the sixth week a thin layer of white substance appears on each lateral surface, and, as the fibres of this layer are found to pass to the cerebellum, they have been named the *direct cerebellar fibres* of the lateral column. At the end of the eighth week of embryonic life still further changes are observed to occur in the cord. Two bundles of longitudinal fibres, one for each side, are intercalated between the direct cerebellar fibres and the posterior gray horns. These bundles, on being traced upwards, are found to pass forwards at the lower end of the medulla, and after decussating with one another, they push aside the anterior columns and form the inner and larger portion of the anterior pyramids of the medulla, and consequently these fibres are called the *lateral pyramidal tracts* (Fig. 34, *P*, *P'*). Contemporaneously with the formation of the lateral pyra-

Fig. 34.



Cord of Human Embryo at Five Months.

ah, *ah'*, Anterior Horns of gray substance; *ph*, *ph'*, Posterior Horns of gray substance; *ar*, *ar'*, Anterior Root-zones; *pr*, *pr'*, Posterior Root-zones; *P*, *P'*, Pyramidal Fibres of lateral columns; *T*, Columns of Türck; *G*, Columns of Goll; *dc*, *dc'*, Direct cerebellar fibres; *c*, Anterior Commissure.

midal tracts an analogous formation appears in the anterior columns, one of these being formed on each side of the anterior median fissure. These tracts have been called the columns of Türck, or of Lockhart Clarke, or the *anterior pyramidal tracts* (Fig. 34, *T*). At the same time that these changes are occurring in the lateral and anterior columns, two bundles of longitudinal fibres make their appearance in the posterior columns, one being situated on each side of the posterior

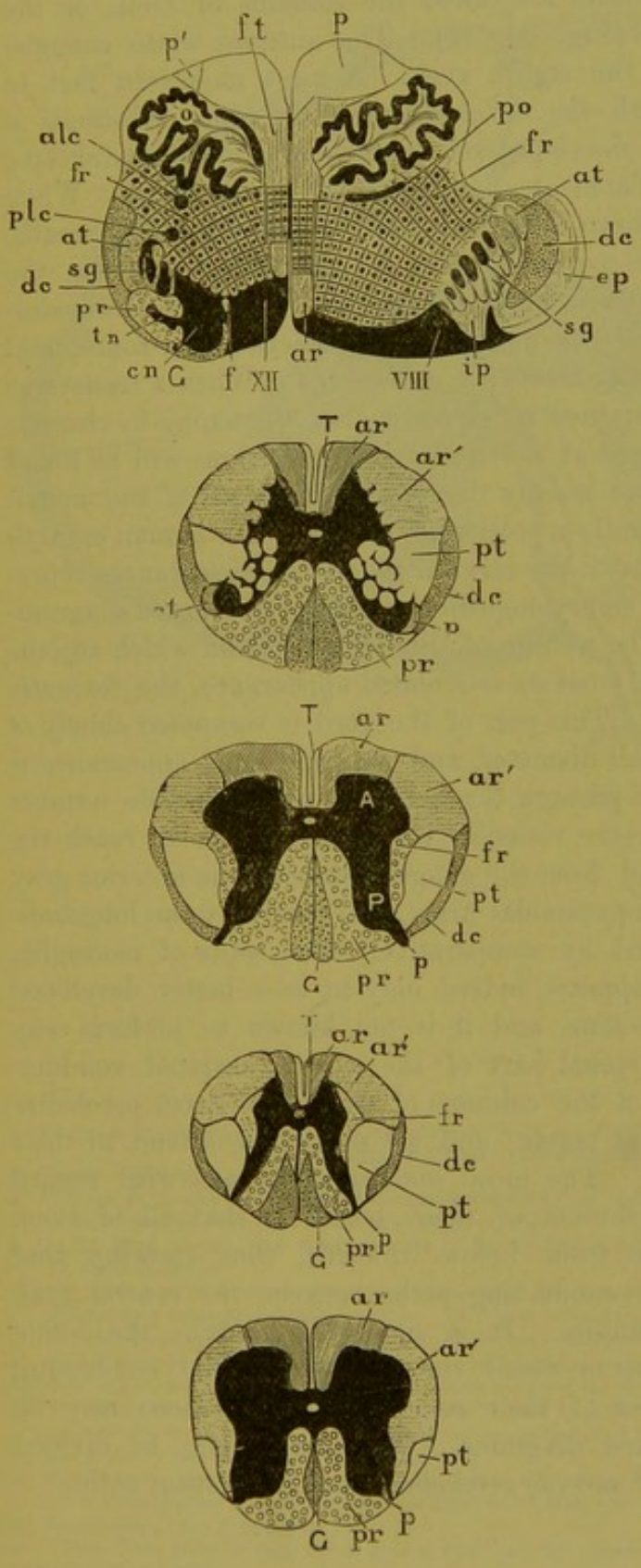


FIG. 35.

(After FLECHSIG.)

G, The column of Goll.
pr, the posterior root-zone.
p, the posterior root.
P, the posterior gray horn.
A, the anterior gray horn.
dc, the direct cerebellar tract.
pt, the lateral pyramidal tract
T, the anterior pyramidal tract.
fr, the formatio reticularis.
ar', the external portion of the anterior root-zone.
ar, the internal portion of the anterior root-zone.
at, the ascending root of the fifth nerve.
Sg, the substantia gelatinosa.
tn, the triangular nucleus
cn, the clavate nucleus.
P and P', the anterior pyramids of the medulla oblongata.
ft, the fillet.
po, the parolivary body.
o, the olivary body.
ep, the external division of the inferior peduncle of the cerebellum.
ip, the internal division of the inferior peduncle of the cerebellum.
alc and *plc*, anterior and posterior nuclei of the lateral column.
f, fasciculus rotundus.
VIII, posterior median nucleus of the auditory nerve
XII, the nucleus of the hypoglossal nerve.

median fissure. These fibres are called the columns of Goll, or the *postero-median* columns (Fig. 34, *G*). The anterior white commissure also appears about the eighth week. A most important fact to notice in connection with the development of the white substance is that the fibres when first developed are destitute of a medullary sheath, and only become medullated at a late period of development. When the cord of a human embryo is examined at the end of the fifth month, it will be found that the pyramidal fibres of the lateral columns, the fibres of the columns of Türck, and the columns of Goll are non-medullated, while the fibres of the anterior and posterior root-zones, and those of the direct cerebellar tracts are medullated. When a transverse section of the cord is examined in glycerin after hardening in chromic acid, the bundles composed of the non-medullated fibres will be found to transmit the light more readily than those composed of the medullated fibres, and consequently a section of the cord of a human embryo at the fifth month presents in the cervical region the appearances represented in Fig. 34. The embryological tracts are represented diagrammatically in Fig. 35. The portion of the lateral column which adjoins the gray matter is named, from its reticulated appearance, the *formatio reticularis* (Fig. 35, *fr*). This part of the cord is composed chiefly of longitudinal fibres of small diameter, and the reticulated appearance is given to it partly by the passage through it of a considerable number of horizontal fibres from the vesicular column of Clarke to reach the direct cerebellar tract, and from the ganglion cells of the anterior gray horns to reach the lateral pyramidal tract, and partly by the longitudinal fibres being separated by comparatively wide septa of neuroglia. The formatio reticularis appears indeed only to be a better developed part of the anterior root-zone, and it is not known to perform any special functions. The spinal part of the encephalo-spinal conducting paths then consists of the columns of Goll, the direct cerebellar tracts, and the pyramidal tracts, and we must now attend to their longitudinal distribution. The most noticeable feature with regard to the longitudinal distribution of these tracts is that all of them increase steadily in size from below upwards, thus showing that they must be regarded as conducting paths between the central gray tube and the higher ganglia. It is impossible, within the limits of this work, to describe in detail the encephalo-spinal conducting paths, but a general view of their course and connections may be obtained from the annexed diagrams. These paths may be divided into (1) the cerebro-spinal and (2) cerebello-spinal conducting paths.

FIG. 36.

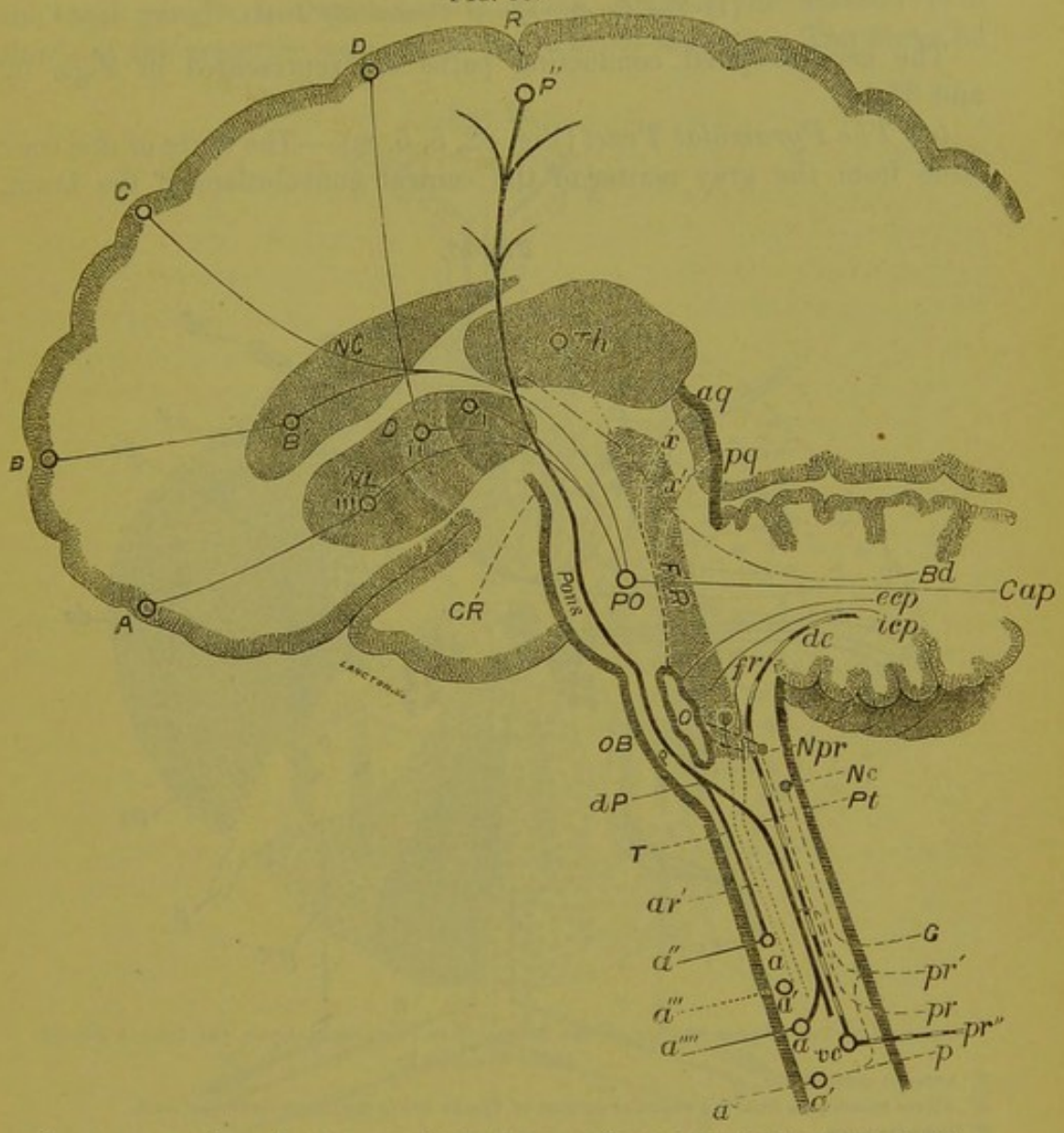


DIAGRAM OF THE GRAY MASSES OF THE SPINAL CORD AND BRAIN, SHOWING THE COURSE OF THE CONDUCTING PATHS (After FLECHSIG.)

R, Fissure of Rolando.

P'' P, *T* and *Pt*, Course of the fibres of the pyramidal tract from their origin in the central convolutions to their termination in the anterior gray horns (*a*, *a'*).

I, *II*, *III*, First, second, and third portions of the lenticular nucleus (*NL*). *NC*, Caudate nucleus. *Th*, Optic thalamus.

D, *C*, *B*, *A*, Points from which fibres issue connecting the cortex of the brain and basal ganglion, and also the gray substance of the pons (*PO*). *Bd*, Fibres connecting the cerebellum and optic thalamus; and *Cap*, those connecting the cerebellum and the gray substance of the pons.

aq, and *pq*, Anterior and posterior pair of corpora quadrigemina respectively.

x, Upper, and *x'*, lower fibres connecting the olivary body and the corpora quadrigemina.

FR, Formatio reticularis of the medulla oblongata, formed by fibres from the optic thalamus (*Th*), the internal division of the inferior peduncle of the cerebellum (*icp*), from the spinal cord (*fr*, *ar*, and *ar'*), and probably also from the clavate nucleus (*Nc*).

O, Olivary body; *ecp*, fibres of the restiform bodies connecting the olivary bodies and cerebellum; other fibres connect it with the triangular (*Npr*) and clavate (*Nc*) nuclei.

dp, Decussation of the pyramids.

pr', Fibres of the posterior roots which pass upwards and downwards into the gray substance, and pursue only a short course.

a, *a'*, *a''*, *a'''*, anterior roots.

p, *pr*, *pr''*, *G*, Fibres of the posterior roots

(1) *The Cerebro-spinal Conducting Paths.*

The cerebro-spinal conducting paths are represented in Figs. 32 and 36.

(*a*) *The Pyramidal Tract* (Fig. 32, 5, 5, *m*).—The fibres of this tract issue from the gray matter of the central convolutions of the brain,

FIG. 37.

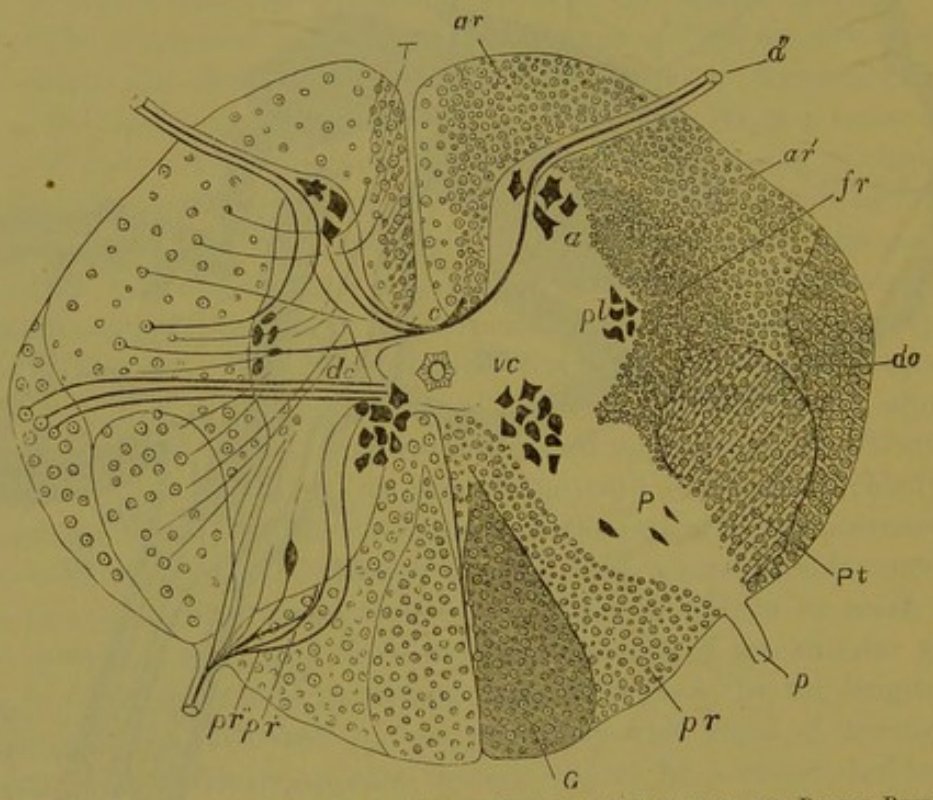


DIAGRAM OF TRANSVERSE SECTION OF THE SPINAL CORD IN UPPER HALF OF THE DORSAL REGION. (After FLECHSIG.)

C, Anterior commissure.
dc', Fibres which pass from the vesicular column of Clarke (*vc*) to the direct cerebellar tract.
P, Posterior horn.

FIGS. 36 and 37.—Letters common to both.

Pt, Pyramidal tract of the lateral column.
T, Columns of Türck.
dc, Direct cerebellar tract.
ar, Internal portion of the anterior root-zone.
ar', External portion of the anterior root-zone.
pr, Posterior root-zone.
G, Goll's columns.
fr, Reticular formation of the spinal cord.
a, Anterior gray horns of the spinal cord.

the axis-cylinder processes of the giant cells already described being continuous with the axis-cylinder of the fibres of this tract. These fibres form at first more or less separate bundles, which descend in the centrum semiovale of the hemisphere, and come together on reaching

the basal ganglia to form a bundle, which occupies the anterior two-thirds of the posterior segment of the internal capsule. The fibres lie

FIG. 38.

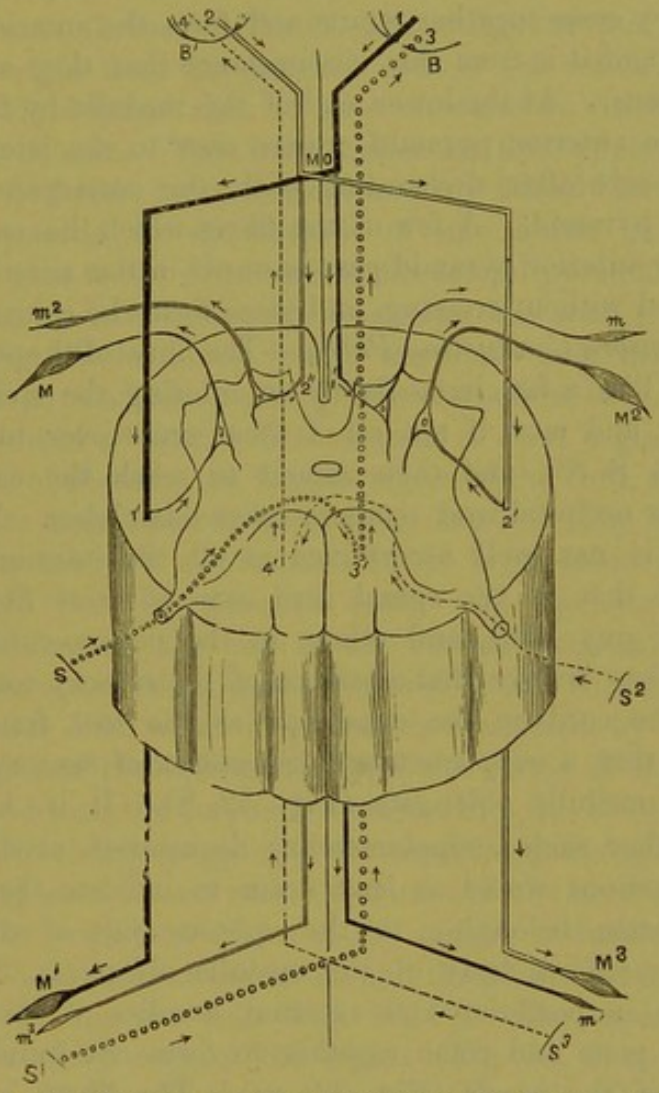


DIAGRAM OF THE SPINAL SEGMENT AS A CENTRE AND CONDUCTING MEDIUM (After BRAMWELL)

B, right, and B', left hemispheres of the brain; M O, lower end of the medulla oblongata; 1, the motor tract from the right hemisphere; it divides at M O, and the larger portion crosses over to the opposite side and passing down the lateral column it supplies the muscles M and M' on the left side of the body. The supply to M is given off at 1'. The smaller portion passes down the anterior column of the same side, and supplies the muscles m and m' on the right side of the body; 2, the motor tract on the left side of the body; the larger portion crosses over to the opposite side and supplies the muscles M^2 and M^3 on the right side of the body, while the lesser portion passes down the same side and supplies m^2 the muscles and m^3 on the left side of the body. S, S', sensory areas on the left side of the body; 3', 3, the main sensory tract from the left side of body; it passes up on the opposite side (right) of the cord in the postero-external column, and proceeds to the right hemisphere of the brain; S^2, S^3, sensory areas of the right side of the body; 4', 4, the main sensory tract from the right side of the body, proceeding up the left side of the cord to the left hemisphere of the brain. The arrows indicate the direction of the conduction.

between the caudate nucleus and optic thalamus on the inner, and the lenticular nucleus on the outer side, but without forming any connec-

tions with these ganglia, and pass still as one bundle through the middle third of the crusta of the crus cerebri. On reaching the pons the fibres become separated into several bundles by the transverse fibres of the middle peduncle of the cerebellum, but on reaching the medulla oblongata they come together again and form the anterior pyramid of the medulla, and it is from this circumstance that they are named the pyramidal tracts. At the lower end of the medulla by far the largest portion of the anterior pyramid crosses over to the lateral column of the opposite side after decussating with the corresponding fibres of the anterior pyramid. A few of the fibres which lie on the external aspect of the anterior pyramid pass onwards in the anterior column of the spinal cord without crossing, and these form the columns of Türck.

(*b*) *The Sensory Conducting Paths.*—The fibres of the posterior nerve-root diverge like a fan immediately on entering the spinal cord (Fig. 37, *pr'*, *pr''*), and most if not all of them cross over to the opposite side (Fig. 38, S S^2), and then ascend to reach the cerebral cortex chiefly of the occipital and temporo-sphenoidal lobes. The course of these fibres is not well ascertained at all the intermediate points. It is probable that in the spinal cord some of these fibres ascend in the posterior gray horn, and others in the postero-external column. Some anatomists believe that a portion of the sensory conducting path ascends in the cord on the same side as the root from which it is derived, and that a supplementary decussation of sensory fibres takes place in the medulla oblongata (Fig. 32, S). It is, however, very doubtful whether such a supplementary decussation exists, and pathological observations would at least seem to indicate that the sensory conducting paths belonging to the opposite side of the body pass through the restiform body of the medulla oblongata. These fibres pass upwards in posterior and external bundles of the longitudinal fibres of the pons and come together to form one bundle in the external third of the crusta (Fig. 32, *ps*). The fibres ascend as one bundle on the posterior third of the posterior segment of the internal capsule, and at this point they are joined by fibres which ascend from the optic tracts through the geniculate bodies and anterior tubercle of the corpora of the quadrigemina around the optic radiations of Gratiolet, and also by fibres from the olfactory tracts of the opposite side, which reach them most probably through the anterior commissure of the third ventricle and the optic thalamus. The sensory fibres of the internal capsule now radiate backwards, outwards, and upwards to reach the cortex of the occipital and temporo-sphenoidal lobes, and to a less extent that of the parietal lobe.

The connections of the intermediate ganglia with the central gray

tube have already been described, and these are also connected with the cortex cerebri by fibres which ascend in the corona radiata.

(2) *The Cerebello-spinal Conducting Paths.*

(*a*) *The Direct Cerebellar Tracts.*—The fibres of this tract appear to take their origin from the ganglion cells of the vesicular column of Clarke (Fig. 37, *dc*), and then to pass outwards to reach the surface of the lateral column. The tract makes its first appearance in the upper lumbar or lower dorsal region, and it gradually increases in size from below upwards, by the addition of new fibres. It lies on the surface of the cord to the outer side of the lateral pyramidal tract, and on reaching the medulla oblongata it occupies the surface of the restiform body (Fig. 35, *dc*), and finally the fibres pass through the inferior peduncle to reach the cortex of the cerebellum (Fig. 33, *dc*).

(*b*) *The Columns of Goll.*—The columns of Goll begin as an insignificant bundle of fibres in the lumbar region of the cord, and gradually increase in size from below upwards (Fig. 35, G). The inferior origin of the fibres is not well ascertained, but it is probable that they are directly derived from the posterior roots. On reaching the medulla the fibres terminate in the cells of the clavate nucleus (Fig. 33, *cn*). This nucleus is connected by arcuate fibres (Fig. 33, 10) with the olivary body of the same side, which in its turn is connected, by means of the inferior peduncle of the cerebellum, with the cortex of the cerebellum (Fig. 33, 7) and with the corpus dentatum (Fig. 33, 6) of the opposite side. The *intermediate ganglia of the cerebello-spinal system* are connected with one another and with the cortex of the cerebellum in various ways. The transverse fibres of the middle peduncle of the cerebellum (Fig. 33, 5) connect the cortex and the interposed gray matter of the pons of the opposite side (Fig. 33, P). The superior peduncle contains fibres which connect the cortex (Fig. 33, 2) and the corpus dentatum (Fig. 33, 3) with the red nucleus (Fig. 33, R) of the opposite side, and fibres (Fig. 33, 1) which pass near the red nucleus and through the optic thalamus to connect the cortex of the cerebellum with the cortex of the opposite hemisphere of the brain.

The connections of the cerebellar system with the anterior gray horns or the motor part of the spinal cord, are not well ascertained, but as the upward continuation of the anterior root-zone of the spinal cord almost surrounds the olivary body and the red nucleus, and passes near to the interposed gray matter of the pons, it is not improbable that connections may be formed between them. These supposed connections are represented in Fig. 33 by the lines 8, 8; 9, 9, and 9″.

CHAPTER II.

PHYSIOLOGICAL INTRODUCTION.

The law of evolution is, as the student has already learned, as applicable to the function as to the structure of the nervous system. It is, indeed, manifest that there must be a determinate relation between these two factors in development. The most general expression of this relationship is that unlike functions entail unlike structures; and, inversely, that unlike parts assume activities of unlike kinds. The whole of scientific pathology may be said to rest upon this law, for we assume that deranged nervous functions are caused by disorders, either molar or molecular, of nervous structures; and, inversely, that injured or diseased nervous structures are, or were, accompanied by corresponding disorders of nervous functions. In tracing the development of the structure of the nervous system, therefore, it was found impossible to avoid continual reference to its functions, and now in briefly discussing the functions of the system constant reference will have to be made to its structure.

The nervous system is, as we have seen, composed of cells and fibres. The cells are originators of motion, and, to some small extent, conductors also, while the fibres are conductors, and only in a very small degree originators of motion. But the unit of composition of a nervous system, which has assumed even a minor degree of complication, is a cell with a nerve fibre connecting it with a muscular fibre or work organ, or a cell with one fibre connecting it with the periphery of the body and another with a muscular fibre or work organ. In the first arrangement the cell gives out energy or discharges at periodical intervals, and the liberated energy being conducted by the efferent fibres to the work organ, gives rise to rhythmical contractions of the muscular fibre. This constitutes *automatic* action. In the second arrangement an impression made on the surface of the body sets up a disturbance which is conducted by an afferent fibre to the nerve-cells, where it liberates a store of energy which is now conducted outwards to the muscular fibre, secreting cell, or other work organ. This constitutes *reflex* action. Of these two mechanisms, the latter is by far the most important. The ganglia and strands of the sympathetic system appear to be simply an aggregation of reflex mechanisms connected together in various ways, and we shall, therefore,

at once proceed to discuss the functions of the spinal and encephalic systems.

1. FUNCTIONS OF THE SPINO-NEURAL SYSTEM.

(1) *Reflex Action.* — The ganglion cells of the anterior gray horns of the spinal cord are centres for reflex actions. The afferent fibres of the reflex loop start from the periphery of the body and pass upwards along the peripheral nerves and through the posterior roots and posterior gray horns to join the ganglion cells of the anterior horns, while the efferent fibres pass outwards through the anterior roots and peripheral nerves to reach the muscles. Two forms of reflex action may be distinguished, (*a*) the *superficial* and (*b*) the *deep reflexes*.

(*a*) The *superficial reflexes* are excited by stimulation of the skin and accessible mucous membranes.

(*b*) The *deep reflexes* consist of muscular contractions evoked by striking the muscles themselves, stretching their tendons, or tapping certain parts of the periosteum, and probably some of the fasciæ. Some physiologists believe that the tendon reactions are caused not by reflex, but by direct action, being supposed to be due to the sudden stretching of the muscular substance itself. It is, however, admitted by all that the integrity of the reflex loop is necessary for the production of these reactions, and we shall consequently speak of them in these pages as if they were proved to be of reflex origin. The various forms of the superficial and deep reflexes will be described in detail when the disorders of the reflex functions are under consideration.

(2) *Automatic Action.*—The spinal cord contains a considerable number of what have been regarded as automatic centres, but it is probable that many of them act in a reflex manner. The lumbar portion of the cord contains centres for the regulation of the acts connected with micturition, defecation, erection and ejaculation, and parturition. The medulla oblongata contains numerous centres which are more or less automatic in their actions. The following may be mentioned: (*a*) the respiratory centre, (*b*) the cardiac centre, (*c*) the vasomotor centre, (*d*) the diabetic centre, or more probably a centre for the regulation of the nutrition of the liver and the other abdominal viscera, (*e*) the centre for deglutition, (*f*) the centre for the movements of the stomach and œsophagus, (*g*) the centre for the regulation of the secretion of saliva, and (*h*) the centre for the dilation of the pupil.

(3) *Trophic Functions of the Cord.*—The ganglion cells of the anterior horns exercise a trophic influence on the muscles with which they are connected by efferent fibres. It is also probable that these

cells exercise a controlling influence upon the nutrition of the bones and joints, while the cells of the posterior gray horns constitute trophic centres for the skin and its appendages.

(4) *Vaso-motor and Secretory Functions.*—Vaso-motor centres exist in the cord, by means of which *tonus* of the muscular coat of the vessels is maintained. The secretions of the glands are also controlled by the spinal centres, and the peristaltic movements of the œsophagus, stomach, and intestines are regulated from the cephalic end of the central gray tube. Little is known, beyond conjecture, of the localiza-

FIG. 39

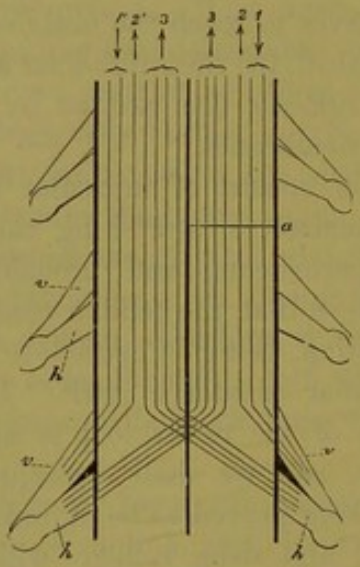


DIAGRAM OF THE COURSE OF THE PRINCIPAL CONDUCTING PATHS WITHIN THE CORD. (After ERB.)

1 and 1′, The motor and vaso-motor tracts passing through the anterior root (v), and remaining on the same side of the cord; 2 and 2′, Tracts which conduct the muscular sensibility, also passing through the anterior roots, and remaining on the same side of the cord; 3 and 3′, The tracts which conduct sensory impressions of touch, temperature, pain, and tickling. These enter the cord through the posterior roots, and cross to the other side, and pursue their course upwards on that side. Section of the right half of the cord (a) must interrupt conduction through the motor, vaso-motor, and musculo-sensory tracts (1 and 2) on the right side, and the cutaneous sensory tracts on the left side (3′).

tion in the cord of the centres of visceral innervation. That they are not situated in the anterior gray horns is rendered certain by the fact that the visceral movements, and the automatic actions of defecation, micturition, erection, and parturition, are unaffected in those diseases in which the lesion is limited to the anterior gray horns. The vesicular column of Clarke, the ganglion cells of which are bipolar like those of the sympathetic ganglia, may possibly contain the centres of visceral innervation.

(5) *Functions of the Cerebro-spinal Conducting Paths.*—A transverse lesion of the cord, if complete, gives rise to loss of sensation and

voluntary motion in all parts of the body which are innervated from the part of the cord which is below the level of this disease. If the lesion is limited to one lateral half of the cord, the sensations of touch, pain, temperature, and tickling are lost on the opposite side to the disease, while muscular sense and voluntary motion are lost on the same side. The annexed diagram (Fig. 39) will afford a sufficient explanation of this statement.

(6) *Functions of the Cerebello-spinal Conducting Paths.*—A transverse lesion of the spinal cord must doubtless arrest conduction through the cerebellar spinal conducting paths, but the symptoms which might be caused by injury of these paths are altogether obscured by the presence of voluntary paralysis. The cerebellar afferent conducting paths are, however, most probably interfered with when the posterior columns of the cord are separately diseased, and it is probable that the incoördination of movements, to be subsequently described under the name of ataxia, is caused by injury of these paths. Disease of the cerebello-spinal efferent conducting paths is not known as a separate affection.

2. FUNCTIONS OF THE ENCEPHALO-SPINAL SYSTEM.

All recent researches have, in my opinion, tended to confirm Mr. Herbert Spencer's hypothesis: "That the cerebellum is an organ of doubly compound coördination in space, while the cerebrum is an organ of doubly compound coördination in time." According to this theory, which has been adopted and elaborated by Dr. Hughlings Jackson, the cerebellum regulates the muscular contractions necessary for the maintenance of all our attitudes in space, while the cerebrum regulates the contractions necessary to effect all the changes of attitude which are made in response to the successive impressions made upon the organism in time. Now, so long as a particular attitude is maintained in opposition to gravity or other forces, the contractions of the various groups of muscles concerned must be *continuous* and in equilibrium with one another; while each change of attitude necessitates the overthrow of this equilibrium by the contractions of some groups of muscles preponderating over those of others, and thus change of attitude involves *alternate* muscular contractions and relaxations. Speaking broadly, then, the cerebellum regulates *continuous* or *tonic* muscular contractions, while the cerebrum regulates *alternate* or *clonic* contractions. It will be seen, therefore, that every compound muscular adjustment necessitates the coöperation of both these organs. No change of attitude can be effected by the cerebrum except in so far as a certain attitude was previously maintained by the cerebellum, and no steady movement can be produced by the alternate contractions of some groups of

muscles except in so far as other groups of muscles are maintained in a state of continuous contraction, and it may consequently be inferred that all the movements of the body are coördinated both in the cerebellum and cerebrum. But although the functions of the cerebellum and cerebrum are to a considerable extent coördinate, yet it is manifest that the former must act in subordination to the latter.

The degree of development to which an animal has attained may, indeed, be measured by its power of effecting multitudinous changes of attitude, and, were it only possessed of the power to maintain one unvarying attitude, its degree of organization would not require to be more complicated than that of a vegetable. But, in effecting these changes of attitude, the alternate contractions, under the guidance of the cerebrum, must take the lead, and any change which is produced in the relative strength of the continuous contractions, although regulated by the cerebellum, must be in strict subordination to the action of the cerebrum.

3. FUNCTIONS OF THE CEREBRO-SPINAL SYSTEM.

The central gray tube, with the peripheral nerves, constitutes, as we have seen, a system of simple coördination in time (reflex action), and we must now point out that the basal ganglia, when acting upon the central gray tube and peripheral nerves, form a system of compound coördination in time (instinctive action), and the cortex of the brain, when acting on the inferior centres, forms a system of doubly compound coördination in time (conscious action). Both conscious and instinctive subconscious movements are comprised under the name of psychical actions. Reflex actions consist, as we have seen, of three factors: (*a*) conduction to a nerve centre of an impression made on the surface; (*b*) reduction to order of these impressions in the centre; and (*c*) conduction of these outwards, with the muscular contractions resulting from them. But, as has been frequently stated by Mr. Herbert Spencer, four factors may be distinguished in every psychical action. To quote Mr. Spencer's own language: "There is (*a*) that property of external objects which primarily affects the organism—the taste, smell, or opacity; and, connected with such property, there is in the external object that character (*b*) which renders seizure of it or escape from it beneficial. Within the organism there is (*c*) the impression or sensation which the property *a* produces, serving as stimulus, and there is connected with it the motor change (*d*) by which seizure or escape is effected. Now, psychology is chiefly concerned with the connection between the relation *ab* and the relation *cd*, under all those forms which they assume in the course of evolution. Each of the factors

and each of the relations grows more involved as organization advances. Instead of being single, the identifying attribute *a* often becomes, in the environment of a superior animal, a cluster of attributes, such as the size, form, colors, motions, displayed by a distant creature that is dangerous. The factor *b*, with which this distant combination of attributes is associated, becomes the congeries of characters, powers, habits, which constitute it an enemy of the subjective factors; *c* becomes a complicated set of visual sensations coördinated with one another and with the ideas and feelings established by experience of such enemies, and constituting the motive to escape; while *d* becomes the intricate, and often prolonged, series of runs, leaps, doubles, dives, etc., made in eluding the enemy."

The account first given of reflex and psychical action shows that the former consists of a muscular adjustment made in response to a *present* impression; while the latter is a muscular adjustment excited by a present impression, but made to attain or avoid an *anticipated* impression. In reflex action the muscular adjustment is effected by means of afferent and efferent fibres and one centre, while in psychical action this mechanism must consist of at least afferent and efferent fibres, a reception and an emission centre with some means of intercentral communication. In the purely unconscious psychical actions, or the instinctive actions, the optic thalamus is supposed to be the central receptive organ, and the corpus striatum the central emission organ, the two being connected by white fibres which interlace with the longitudinal fibres of the internal capsule. The cortex of the cerebrum with its afferent and efferent fibres appears to be the organ for the regulation of the conscious psychical actions, although the fact that individuals sometimes sing songs or recite long passages of poetry during the unconscious stage which follows an epileptic attack, or that which results from the mesmeric state or from chloroform narcosis, seems to show that all actions, which, like spirits, are regulated from the cortex of the brain, are not necessarily attended by consciousness. A glance at the structure of the cortex of the brain (Fig. 30) will show that the small cells of the outer layers are best adapted to act as a receptive organ, while the caudate cells of the inner layers are best adapted to act as an emissive centre. The structure of the outer layers of the cortex in comparison with the inner layers, forcibly reminds one of the structure of the posterior gray horn of the spinal cord in comparison with that of the anterior horn. In the outer layers of the cortex, as in the posterior gray horn, the afferent fibres terminate without forming definite connections with the cells, which are in both cases, small, round, and destitute of processes, while in the inner layers of the cortex and in the

anterior gray horns, the cells, which are large and furnished with numerous processes, are definitely connected with the axis-cylinders of efferent fibres.

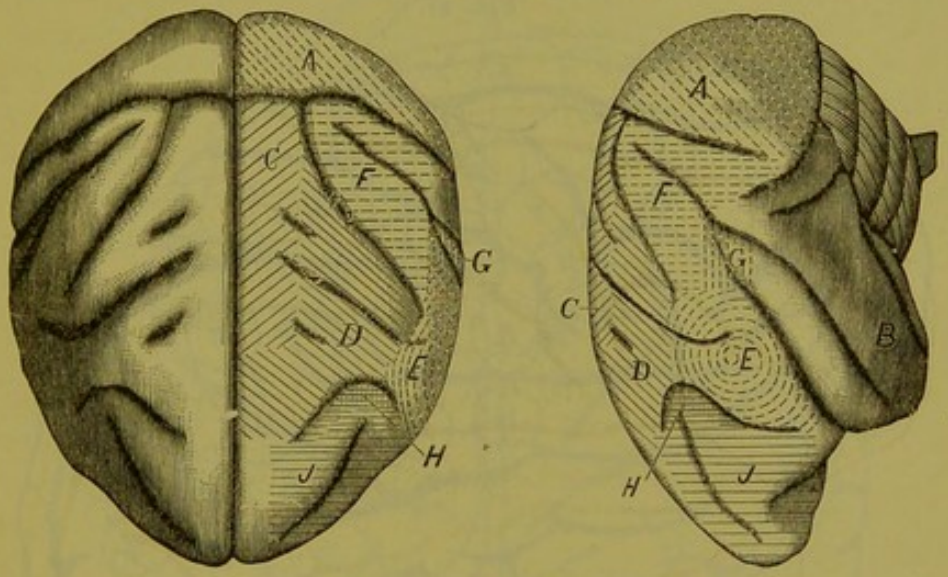
Now, although it is probable that the outer layers of the cortex of the whole cerebrum constitute a receptive organ, and the inner layers an emissive organ, yet, the outer layers of certain districts of the cortex have become specially adapted to act as receptive organs or sensory centres, and the inner layers of other districts have become specially adapted to act as emissive organs or motor centres.

(1) *The Sensory Cortical Centres.*—From his first experiment, Ferrier concluded that the centre for vision was situated in the angular gyrus and surrounding gray matter; the auditory centre in the superior temporo-sphenoidal convolution; the centres of taste and smell at the extremity of the temporo-sphenoidal lobe, and that of touch in the uncinate gyrus and the hippocampus major. Munk believes that the visual centre or area is of much larger extent than that assigned to it by Ferrier, and that it is situated in the occipital lobes. He maintains that removal of this area causes blindness, and that extirpation of small portions of it gives rise to blindness of localized areas of the retina. He believes that there are three visual spheres in the cortex corresponding to three visual areas in the retina. The external part of the retina of the left eye is connected with the external part of the cortical visual centre in the left hemisphere, while the internal and central portions of the retina of the right eye are respectively connected with the internal and central portions of the visual centre of the left or opposite hemisphere. The upper portion of the retina is connected with the front, and the lower part with the posterior aspect of the visual centre of the opposite hemisphere.

Removal of both visual centres causes, according to Munk, complete or *absolute* blindness, while partial removal of these areas causes incomplete, or what Munk calls *psychical* blindness, a state of vision first observed by Goltz, in which the animal sees and avoids objects, but fails to recognize the special properties of a piece of meat, for example, which renders it food to be eaten instead of an obstacle to be avoided. Munk regards the whole superior and internal surface of the cerebral hemisphere as constituting a sensory area, and the annexed diagrams (Fig. 40) indicate the manner in which he believes these centres to be distributed. Very elaborate experiments have been conducted by Drs. Ferrier and Yeo, to determine the exact seat of the centre of vision, and the most important conclusion they come to is that "destruction of both angular gyri and occipital lobes causes total and permanent blindness in both eyes without any impairment of the other senses or of motor

power." Blindness or hemiopia was caused by less extensive injuries, but partial or complete recovery always ensued. The experiments of Luciani show that visual disturbances follow extirpation, not only of the occipital, but also of the parietal, temporal, and frontal lobes. It may therefore be concluded that the localization of sensory centres is a diffused one, and that the whole surface of the cortex is possessed of more or less of sensory functions.

FIG. 40.



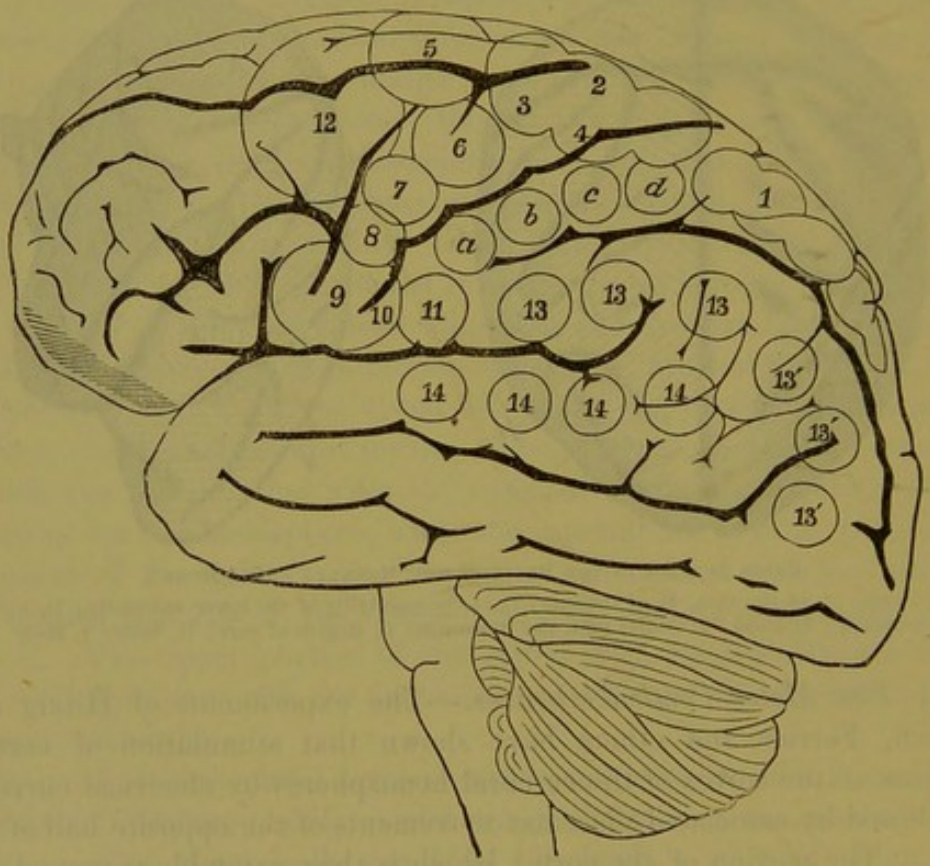
UPPER SURFACE OF THE BRAIN OF THE MONKEY. (After MUNK.)

Sensory Areas: A, of the eyes; B, of the ears; C, of the sensibility of the lower extremity; D, Anterior extremity; E, Head; F, Ocular muscular apparatus; G, Region of ears; H, Neck; I, Body.

(2) *The Motor Cortical Centres.*—The experiments of Hitzig and Fritsch, Ferrier, and others have shown that stimulation of certain portions of the cortex of the cerebral hemispheres by electrical currents is followed by associated muscular movements of the opposite half of the body. The portion of the cortex which is thus excitable is named the *motor area.* This area corresponds generally with the part supplied by the Sylvian artery, and also with the area in which the *giant cells*, already described, have been discovered. The portions of the cortex—the areas supplied by the anterior and posterior cerebral arteries—which do not respond to electrical excitation are called *latent areas.* It has also been found that extirpation of a localized portion of the motor area causes paralysis of the muscles which are thrown into a state of spasm by electrical excitation of this portion. The annexed diagrams (Figs. 41 and 42) show the topographical distribution of these motor centres on the outer and superior surfaces of the cerebral hemispheres without further description. The experiments of Schafer and Horsley

have shown that a portion of the cortex of the internal surface of the cerebral hemisphere is excitable. The excitable portion is limited to the marginal convolution, and extends as far forwards as the junction of the middle and posterior thirds of the superior frontal convolution, and as far backwards as a point opposite the centre of the parietal lobule. Speaking in general terms, stimulation induces contractions of the muscles of the trunk and of the large muscles inserted in the shoulder-blade and shoulder, and those inserted about the hip-joint.

FIG. 41.



FIGS. 41 and 42.—SIDE AND UPPER VIEWS OF THE BRAIN OF MAN. (After FERRIER.)

These figures are constructed by marking on the brain of man, in their respective situations, the motor areas of the brain of the monkey as determined by experiment, and the description of the effects of stimulating the various areas refers to the brain of the monkey.

1 (On the posterior-parietal lobule), Advance of the opposite hind limb as in walking.

2, 3, 4 (Around the upper extremity of the fissure of Rolando), Complex movements of the opposite leg and arm, and of the trunk as in swimming.

a, *b*, *c*, *d* (On the ascending parietal convolution), Individual and combined movements of the fingers and wrist of the opposite hand. Prehensile movements.

5 (At the posterior extremity of the superior frontal convolution), Extension forward of the opposite arm and hand.

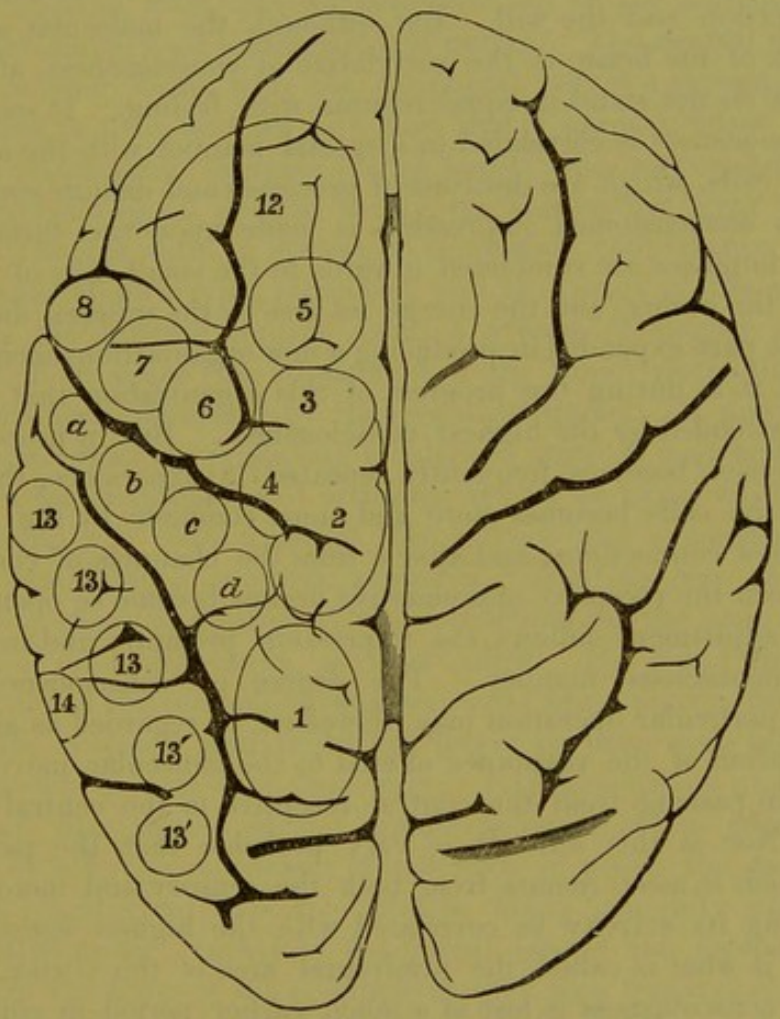
6 (On the upper part of the ascending frontal convolution), Supination and flexion of the opposite forearm.

7 (On the median portion of the ascending frontal convolution), Retraction and elevation of the opposite angle of the mouth by means of the zygomatic muscles.

8 (Lower down on the same convolution), Elevation of the ala nasi and upper lip with depression of the lower lip on the opposite side

(3) *The Intercentral Connections.*—The central ends of the individual sensory mechanisms are unified by a collective centre—the *sensorium commune*—which is the general centre of nervous connections on the afferent side. It may also be supposed that the individual motor centres are unified by a general centre of nervous connections which may be regarded as a *motorium commune*. Between the sen-

FIG. 42.



9, 10 (At the inferior extremity of the ascending frontal and posterior extremity of the third frontal convolution), Opening of the mouth with (9) protrusion and (10) retraction of the tongue. *Region of Aphasia.*

11 (At the inferior extremity of the ascending parietal convolution), Retraction of the opposite angle of the mouth, the head turned slightly to one side.

12 (On the posterior portions of the superior and middle frontal convolutions), Eyes opening widely, pupils dilating, and the head and eyes turning towards the opposite side

13, 13′ (On the supramarginal lobule and angular gyrus), The eyes moving towards the opposite side with an upward (13) or downward (13′) deviation. Pupils generally contracting. (Centre of Vision.)

14 (On the inframarginal or superior temporo-sphenoidal convolution), Pricking up of the opposite ear, head and eyes turning to the opposite side, and pupils dilating largely. (Centre of hearing.)

Ferrier, moreover, places the centres of taste and smell at the extremity of the temporo-sphenoidal lobe, and that of touch in the gyrus uncinatus and hippocampus major.

sorium commune and the motorium commune there is no distinct line of demarcation, nor is there any definite boundary between these highest centres and the individual sensory and motor centres. All of them run indistinguishably into one another, and all of them are represented by the cortex. The activity of the individual sensory centres is the correlation of sensation and feeling, and of the highest sensory centre of perception—emotion, and the highest operation of the intellect, while the activity of the motor centres is the correlation of conscious exertion and the will. But although the molecular activity of the cortex of the brain is the correlative of consciousness, all parts of the cortex do not stand in equal relation with feeling. It seems to me that consciousness is correlated in a special manner with the activity of the small cells, which are destitute of processes and definite connections. When an unaccustomed impression is made upon the surface of the body the impulses are conducted inwards to the small cells of the outer layers of the cortex, and the energy set free by the unusual disturbance is in great part expended in producing a new organization amongst these cells, and it is during the progress of this organization that psychical action is attended by the highest consciousness. But in proportion as this impression becomes frequently repeated in experience, the organization of the cells becomes more and more complete by the establishment of new connections, and after a time the channels of communication between the receptive and emissive organs become so open that the muscular adjustment follows the impression promptly and in almost a wholly unconscious manner. The degree of consciousness which attends a particular operation may, therefore, be regarded as an expressive indication of the resistance offered to the molecular movements or impulses in passing from the central receptive to the central emissive organs. Nor is this all. It is very probable that the part of the centre which is most remote from both the sensory and motor centres will, during its activity be correlated with the highest consciousness. This part is what is called the præfrontal area of the cortex, and the fact that consciousness is lost at a much earlier period in epileptiform attacks caused by lesions of this area than in the attacks which are caused by lesions in other areas of the cortex, speaks much in favor of the supposition just advanced.

The different cortical sensory and motor centres are connected with one another in various ways by systems of white fibres. These systems are the longitudinal or collateral fibres, consisting of the arcuate fibres or fibræ propriæ, fibres of the gyrus fornicatus, longitudinal septal fibres, the fasciculus uncinatus, the longitudinal inferior fasciculus, the longitudinal fibres of the corpus callosum, and the perpendicular occipital

fasciculus described by Wernicke, and the transverse or commissural fibres, which connect similar points of the convolutions of the two hemispheres, and which consist of the transverse fibres of the corpus callosum, and the fibres of the anterior and posterior commissures of the third ventricle.

4. FUNCTIONS OF THE CEREBELLO-SPINAL SYSTEM.

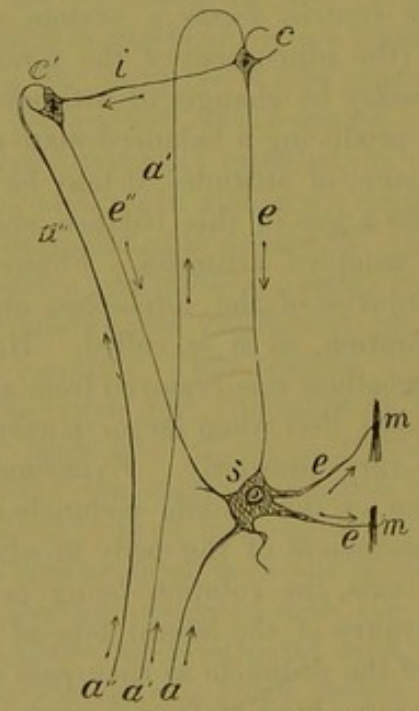
The cerebellum must, like the cerebrum, act on the muscular system through the spinal cord and peripheral nerves. The central gray tube, as we have seen, forms with the peripheral nerves a system of simple coördination in time, and we now find that it likewise forms a system of simple coördination in space (*reflex tonus*); the intermediate ganglia of the cerebello-spinal system acting on the central gray tube and peripheral nerves form a system of compound coördination in space (maintenance of unvarying attitudes), and the cortex of the cerebellum acting on the inferior centres forms a system of doubly compound coördination in space (the adjustment of the tonic contractions of the muscles rendered necessary by changes of attitude). If now the cerebellum is an organ for producing a balanced state of contraction of the muscles in the maintenance of attitudes, it may be expected that injury of this organ will cause a loss of this balance and a consequent difficulty in maintaining complex attitudes. Flourens was the first to observe the effect of injuries of the cerebellum on the maintenance of attitudes, or on equilibration, as it is called. He found that when a small portion of the cerebellum was removed from a pigeon, the animal's gait became unsteady, and that when larger portions were taken away, the movements became very disorderly. Experiments on animals have also shown that section of the middle peduncle of the cerebellum is followed by a forced movement of the body in which the animal rolls round its longitudinal axis, the rotation being generally towards the side operated upon. Injury of the lateral lobe of the cerebellum, and probably of the fibres of the peduncle as they pass transversely through the pons, produces the same kind of forced movement as section of the middle peduncle. Nothnagel concludes from experiments on rabbits that lesions which injure the fibres uniting the two sides of the organ occasion the greatest amount of motor disturbance. Ferrier found that electrical stimulation of the cortex of the cerebellum in animals caused movements of both eyes with associated movements of the head, limbs, and pupils. Our relations to the external objects in space are largely determined by the sense of vision, which is better adapted than any of the other senses for conducting a large number of simultaneous impressions from the periphery to the centres, and it may consequently

be expected that the organs of vision will stand in a peculiarly intimate relation to the chief organ for regulating the attitudes of the body. The semicircular canals, with the portion of the auditory nerve which supplies them, appear to form a special peripheral organ for determining the attitudes of the body through the cerebellum.

5. COÖPERATION OF THE CEREBRO-SPINAL AND CEREBELLO-SPINAL SYSTEMS.

According to the theory which has just been advanced the multitudinous adjustments of the body, both in time and space, are regulated by the combined action of the cerebrum and cerebellum acting through the spinal cord and peripheral nerves. The coöperation of these organs in the regulation of motor actions is, however, generally of an antagonistic

FIG. 43.



SCHEMA OF ENCEPHALO-SPINAL ACTION.

s, Motor ganglion cell of spinal cord; *c*, Ganglion cell of cortex of cerebrum, and *c'*, of cortex of cerebellum; *a*, *a'*, *a''*, Afferent fibres to the spinal cord, and to the cortices of the cerebrum and of the cerebellum respectively; *e*, *e*, Efferent fibres from the spinal ganglion cell to *m*, *m'*, the muscles; *e'* and *e''*, Fibres from the cerebral and cerebellar cells respectively to the spinal ganglion cell; *i*, Intercentral fibre connecting the cerebral and cerebellar cells. The arrows indicate the direction of the conduction.

kind. The cerebellum tends to maintain an unvarying attitude, while the cerebrum, in initiating a change of attitude, must act by overthrowing the balance of the muscular contractions which maintain this attitude. The overthrow of this equilibrium can be effected by the cerebrum in

either of two ways, either *positively* by an increase of nervous impulses to certain groups of muscles, or *negatively* by arresting or inhibiting in the spinal centres the cerebellar influx to their antagonists. Now it is manifest that the latter method would be much more economical than the former, and consequently there is every reason to believe that the cerebrum does act largely by inhibiting the action of the cerebellum, although it is also certain that it must exercise a positive control over the various muscular contractions. The conjoint action of the central gray tube, the cerebrum, and cerebellum is represented in the accompanying diagram (Fig. 43) under the simplest conditions. A ganglion cell of the spinal cord is represented by *s*, of the cerebrum by *c*, of the cerebellum by *c'*. The afferent conducting paths from the periphery to the spinal cord, cerebrum, and cerebellum are represented by *a*, *a'*, *a''* respectively. The efferent conducting path between the cerebrum and spinal cord is represented by *e'*, between the cerebellum and cord by *e''*, and between the cord and muscles by *e*, while *m m* represent the muscles themselves, and the arrows indicate the direction of the currents. Now, when an impression is made upon *a*, it is conveyed to *s*, and reflected through *e e* to *m m*, this constituting a simple reflex action. When an impression is made upon *a''*, the impulse is conveyed to *c'* and through *e''* to *s*, and through *e e* to *m m*, producing a continuous contraction of the muscles. But when an impression is made upon *a'*, an impulse is conveyed to *c* and downwards, through *e* to *s*. Now, the impulses conveyed through *e'* to *s* may produce, when of a certain degree of intensity, only an arrestive or inhibitory action on the impulses conveyed to *s* through *a* and *e''*, while an additional degree of intensity enables it to pass through *s* and *e e* to *m m*, and to produce clonic muscular contractions. It is also probable that the cerebrum may exercise both an inhibitory and excitative action on the cerebellum through the intercentral fibres (*i*), which connect the centres directly with each other. This hypothesis was first stated by Dr. Hughlings Jackson, and he has since applied it, with his usual subtle and generalizing power, to the explanation of various pathological phenomena. As this is an exceedingly important theory, it will be as well to illustrate the actions of the cerebrum and cerebellum by reference to the muscular contractions necessary for the maintenance of the erect posture and for locomotion.

(*a*) *The Erect Posture.*—In the erect posture the weight of the body is borne by the plantar arches, and the body is maintained by a series of muscular contractions in such a position that the line of gravity falls within the area of the feet. In this position the line of gravity of the head falls in front of the occipital articulation, that of the combined

Fig. 44.

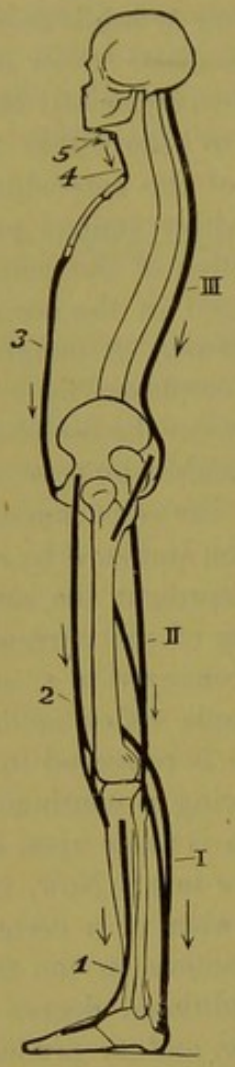


Diagram Illustrating the Attachments of some of the most Important Muscles which keep the Body in the Erect Posture. (After Huxley.)

I. Muscles of the Calf. II. Those of the back of the Thigh. III. Those of the Spine, which tend to keep the body from falling forwards. 1, the Muscles of the front of the Leg; 2, those of the front of the Thigh; 3, those of the front of the Abdomen; 4, 5, those of the front of the Neck, which tend to keep the body from falling backwards. The arrows indicate the direction of action of the muscles, the foot being fixed.

head and trunk passes behind a line joining the two hip-joints, that of the combined head, trunk, and thighs falls a little behind the knee-joints, and the line of gravity of the whole body passes in front of the line drawn between the two ankle-joints. This statement of the direction of the line of gravity shows that when the foot is made the surface of support, the body would fall forwards unless prevented by contraction of the muscles of the calf (Fig. 44, I). "But this action," says Prof. Huxley, "tends to bend the leg, and to neutralize this and keep the leg straight the great muscles in front of the thigh (Fig. 44, 2) must come into play. But these, by the same action, tend to bend the body forward on the legs; and if the body is to be kept straight they must be neutralized by the action of the muscles of the buttocks and of the back (Fig. 44, III)." It will be seen, however, that since the centre of gravity of the combined head and trunk falls a little behind the line joining the hips, the muscles of the buttocks, although strongly contracted in effecting the erect position, do not require to contract in order to maintain it.

The muscles of the calf, those of the front of the thigh, and the erector spinæ are therefore the most active muscles in maintaining the erect posture, and these are, according to the hypothesis, maintained in a state of tonic contraction, mainly by the cerebellum.

(*b*) *Walking.*—At each step in walking there is a moment at which the body rests vertically on the foot of one leg (say the right), which is then called the "*active leg.*" The other (left), which is now called the "*passive leg,*" is at this time inclined obliquely, with the heel raised and the toe resting on the ground. The left leg, slightly flexed to avoid contact with the ground, is now swung forwards like a pendulum, the length of the swing or step being determined

by the length of the leg, the left toe is brought to the ground, and the step is finished. The left leg, which was previously passive, now gradually becomes straight and rigid, and the body is moved forwards on the left toe as a fulcrum; while the right leg, which was previously active, assumes an inclined position, with the heel raised and the toe resting on the ground, so that it is ready to swing forwards, and then once more to assume the rôle of activity, while its fellow becomes in its turn passive again. During the forward movement the centre of gravity of the body describes a curve, the convexity of which is upward; hence in successive steps the centre of gravity, and with it the top of the head, describes a series of curves, with their convexities upwards.

In standing on both feet the line of gravity falls between them, but in walking it must be alternately shifted from one foot to the other, in order to balance the body on the active leg. While the left leg, for instance, is passive and swinging, the line of gravity falls within the area of the right foot, and passes through the right lateral half of the pelvis, and as the left foot becomes active the centre of gravity is shifted to the opposite side, and the line of gravity passes through the left lateral half of the pelvis to the left foot. In walking, therefore, the centre of gravity describes not only a series of vertical but also a series of horizontal curves, so that the curve described by the head is composed of vertical and horizontal factors. In slow walking there is an appreciable time during which both feet are on the ground; the one being planted so as to become active before the other has ceased its activity. In fast walking this period is very short, the one leaving the ground the moment the other touches it, while in running there is an interval during which neither foot is on the ground.

Let us now attend to the muscles, the contraction of which effects the changes of attitude necessarily involved in walking. Suppose that we start with the right leg in the vertical position, with the line of gravity passing within the line of the right foot, and the left partially raised from the ground. The first indication of a forward movement must be effected by a contraction of the flexors of the foot on the leg, which, as the toe is fixed, bends the leg and with it the whole body forwards. This contraction fixes the upper end of the tibia, the leg being bent forwards at an acute angle with the foot, and the femur is kept extended on the tibia by a rigid contraction of the muscles of the front of the thigh. The lower end of the femur and upper end of the tibia are now rendered fixed points, the line of gravity is rapidly passing forwards from the middle of the foot to the toe, the weight is thus taken off the heel, and contraction of the muscles of the calf causes its elevation.

But the line of gravity is now passing through the toe, in front of the knee, and in front of the centre of the hip-joint, so that the muscles of the back of the thigh and those of the buttocks must contract strongly or the body would be flexed on the thighs, while the erectors of the spine must be sufficiently contracted to keep the different segments of the body in a rigid condition. It is manifest that as soon as the line of gravity passes in front of the centre of the hip-joint and through the toe, although muscular action may maintain the different segments of the body extended, no muscular action can prevent the body as a whole from falling forwards on the toe as a pivot, and the body would fall unless the left foot were now in a position to be planted on the ground in front of the line of gravity, and ready to assume the rôle of the active leg. Before, however, the left leg can become active, the line of gravity must be transferred to the left foot, and before the right foot can be made to swing it must be shortened so as to avoid contact with the ground. These operations are so important as to require careful study.

(*c*) *The transference of the centre of gravity from the passive to the active leg* is largely effected by the contraction of the abductors of the thigh, especially by the gluteus medius, contraction of which, the left thigh being fixed, causes the pelvis to rotate vertically on the hip-joint; so that the centre of gravity, and with it the head, describes a curve to the left, with its convexity upwards, a movement which at the same time slightly elevates the pelvis and with it the hip-joint of the opposite side. The slight elevation of the right hip-joint not only transfers the centre of gravity to the left, but also increases the distance of the centre of movement (hip-joint) of the passive leg (right) from the ground, and thus prepares for the forward swinging of the right leg. The contraction of the abductors is accompanied by a contraction of their antagonists—the adductors, which not only gives steadiness to the pelvis but holds the latter in readiness to counteract at once any tendency to overaction on the part of the former, by which the line of gravity would be carried beyond the middle of the foot. The curve described by the head owing to contraction of these muscles would indeed be much greater than it is were it not compensated by contraction of other muscles. At the time that the abductors of the left leg contract, and thus rotate the pelvis, the centre of gravity, and head to the left, the erector spinæ of the right side enter into a somewhat additional contraction producing a compensating curve to the right, so that the head does not deviate to the left during the transference of the centre of gravity to the left foot to anything like the extent that might be expected.

(*d*) *Swinging of the Passive Leg.*—It has just been said that when the left leg becomes active the pelvis rotates vertically on the left hip, so that the opposite hip-joint is slightly elevated to an extent sufficient to take the weight of the body from the right toe, but inasmuch as the right foot is, at the time it is about to become passive, extended obliquely, with the toe depressed, while the left is placed nearly vertically, the former is much too long to swing past the other without touching the ground, and the slight vertical rotation of the pelvis just described does not give the requisite elevation for this purpose. In order to swing forwards, therefore, the right leg is still further shortened by flexion of its various segments on the body and on one another. The thigh is slightly flexed on the body, the leg on the thigh, and the foot on the leg. Of these movements the slight elevation of the toe caused by dorsal flexion of the foot is by far the most important and special; it is this movement which distinguishes the walk of the adult from that of the infant, the latter advancing the passive foot not by a pendulum motion, but by a voluntary effort in which the leg and foot are raised from the ground by the flexion of the thigh on the body. It may also be mentioned that the adductors of the thigh manifest a very special action in assisting to cross one leg over the other—an action which cannot be effected by the lower animals, or by the human infant, and hence these muscles must also be regarded as being in an especial manner under cerebral influence. The cerebro-spinal influence is therefore manifested in the active leg during locomotion by securing a strong contraction of the anterior flexors of the foot, and of the flexors of the leg on the thigh along with the abductors so as to fix and rotate the pelvis vertically, while it is manifested in the passive leg partly by contraction of the flexors of the thigh on the body, partly by contraction of the flexors of the leg on the thigh, and partly by flexion of the foot on the leg, the flexion in all these instances being probably due less to active contraction than to relaxation of the antagonistic muscles. The transference of the line of gravity to the active leg also takes part in this action by removing the fixed point, from which the muscles of the passive leg act, from the foot to the pelvis.

(*e*) *The Act of Acquiring the Erect Posture.*—Now, if the changes of position which take place in walking are due to the predominance of cerebro-spinal over cerebello-spinal action, this is no less true with respect to the successive changes of posture requisite to raise the body from the recumbent to the erect posture. Suppose a man is lying in the prone position, and then gets up on his hands and knees. When the knees are raised by muscular action, so that the body is supported by the tips of the fingers and the toes, while the centre of gravity falls

midway between the anterior and posterior extremities, this constitutes what I may call the *quadrupedal* position. In this position the toes constitute the fixed point for the posterior extremities, and the muscular strain rests upon the flexors of the phalanges, their contraction being necessary to maintain the rigidity of the plantar arch. The extensors of the foot on the leg must contract to prevent flexion of the leg at the ankle, the extensors of the leg on the thigh must contract in order to prevent the thigh being flexed on the leg, and the extensors of the body on the thigh must also contract in order to prevent the former being flexed on the latter. A further contraction of these same muscles drags the centre of gravity of the body upwards and backwards, the weight is taken off the anterior extremities, and the body assumes the semibipedal posture, in which the line of gravity passes between the feet in the line which joins the toes, considerably in front of the line which joins the ankles, behind that which joins the knees, and in front of that joining the hip-joints. It is manifest that the contractions of the muscles of the sole, those of the calf, front of the thigh, those of the gluteal region and the erectors of the spine, must largely predominate over their antagonists in order to maintain this position, and that this predominance must be maintained until such time as the heel touches the ground, when the line of gravity passes from the toes to the centre of the plantar arch, and behind the line joining the centre of the two hip-joints. The vertical position is then maintained mainly by means of the bones and ligaments, aided only by a slight degree of muscular contraction. Now, the bipedal erect posture has only been attained from the recumbent position by passing through an infinity of intermediate postures, and according to the hypothesis the cerebello-spinal system has had to maintain each posture attained by striking a balance between the tensions of the extensors and flexors of the body, the latter being aided by gravity; while the cerebro-spinal system continually changes each attained posture by overthrowing this balance in favor of the extensors. In passing from the bipedal erect posture through the semibipedal and quadrupedal to the recumbent posture a reverse process takes place, the cerebro-spinal system at each new position acquired, inhibits the action of the cerebello-spinal system on the extensors, so that contraction of the flexors assisted by gravity is allowed gradually to predominate.

But if this hypothesis of the joint, although opposite, action of the cerebrum and cerebellum acting through the spinal cord be true at all, it must be accepted in its fullest extent. If, for instance, it be true that the passage from what I have called the *quadrupedal* to the *bipedal* posture in the human subject is due in the individual to the

predominance of cerebral influx to the extensors over their antagonists, this is no less true with respect to the race. In the gradual development of man from the lower animals the same forces have been at work. From the semibipedal position assumed by a dog attempting to stand on its hind legs, and the imperfect bipedal attitude of the monkey to the perfect bipedal posture of man, the transition must have been effected by the gradual predominance of the extensors over their antagonists through cerebro-spinal influence. In all these processes it will be seen that the flexors of the body are aided in their action by the force of gravitation, while the extensors have to overcome this force by their action, hence the latter must be capable of much more powerful contraction than the former, and are consequently more liable to have more powerful discharges sent to them both from the cerebro-spinal and cerebello-spinal systems.

When, therefore, both the extensors and flexors of the head, trunk, and lower extremities are contracted to their utmost capacity, the action of the former must predominate over that of the latter; so that the segments of the lower extremities will be extended upon one another, and the body will be arched with the concavity directed backwards, as occurs during the paroxysms of tetanus. But the hand being mainly an organ of prehension, the principal functions of the anterior limbs of man is to pull objects towards the trunk, the latter being the fixed point during their activity, hence the flexors of the upper extremities must be more strongly developed than the extensors. And when the hands become fixed, as in climbing, the most powerful contractions are obtained when they are in a position to drag the body towards the fixed position, and not, as in the case of the lower extremities, when the body is thrust upwards and away from it; hence, when the muscles of the upper extremities are contracted to their utmost capacity, flexion will predominate over extension.

(*f*) *Fundamental and Accessory Portions of the Nervous System.*—Before leaving this portion of our subject I should like to establish one more distinction. Structure being the correlative of function, the multiplicity and complexity of the movements which distinguish man from the lower animals must be accompanied by a corresponding degree in the intricacy and variety of the structural arrangements of his nervous system. The main movements which distinguish man from the lower animals are those concerned in attaining and maintaining the erect posture, the varied movements of the hands as organs of prehension, the movements of voice and articulation concerned in speech, and those which are active in the production of facial expression. All these movements must, therefore, be represented in the human nervous system

by structural arrangements superadded to those which man possesses in common with the highest of the lower animals. Indeed, all the complex movements first mentioned are acquired considerably after the birth of the human infant, and we may consequently expect that the structural arrangements corresponding to them either do not exist at birth or exist only in an embryonic condition.

The portions of the nervous system which man possesses in common with the lower animals, and which are well developed in the human embryo at nine months, I shall call the *fundamental* part, and the portions which have been superadded in the course of evolution, which differentiate the nervous system of man from that of the highest of the lower animals, and which are either absent in the human embryo, or exist only in an embryonic condition, I shall call the *accessory* part of the nervous system.

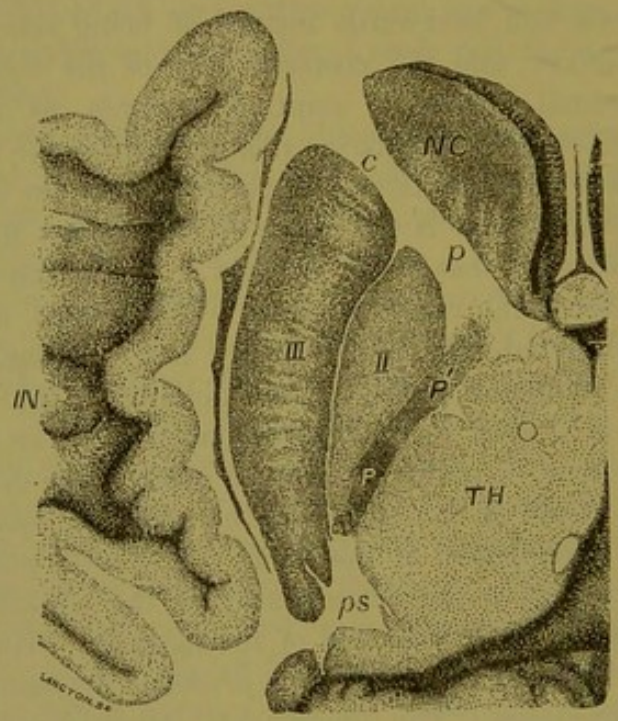
The fundamental portion of the human nervous system coördinates the fundamental functions which man possesses in common with the lower animals, but the accessory portions can only be said to regulate the accessory functions in a peculiar sense. The accessory structure constitutes indeed a new complexity of mechanism superadded to that already existing, a complexity rendered necessary for the regulation of the intricate and multiform actions which distinguish man from the lower animals. In the development of the accessory system, small round cells and non-medullated fibres appear at a comparatively late period in the development of the embryo, and the presence of these simple elements may be regarded as the structural counterpart of a new modification or specialization of function. Specialization of function has hitherto been connected with the gradual development of medullated from non-medullated fibres, and of large caudate from small round cells; but now it appears that specialization of function is to be connected with the development of embryonic cells and fibres. There is, however, no contradiction between the two statements. The embryonic cells and fibres of the accessory system do not of themselves indicate any specialization of function. These cells and fibres are, indeed, mere complications of an already existing mechanism, and it is this alone which entitles them to be regarded as true indicators of a newly acquired specialization of function; they are, in short, mere modifications of an already existing structure corresponding to newly acquired modifications of previously existing muscular adjustments. There can be no doubt that the fundamental and accessory portions of the nervous system will be so mingled together that it will be almost impossible to separate the two, but whether they can be distinguished from one another morphologically or not, the mental distinction is a

valuable one, and it will be found of importance to remember that in man the cephalic ganglia, the central gray tube, the conducting paths, and even the peripheral nerves, *must* contain fundamental and accessory cells and fibres.

A comparison of the brain of man with that of the monkey will afford a rough test by which we may distinguish the accessory portions of the former of the two. Passing over such obvious considerations as distinctions of size and weight, the first important difference we note is the great relative increase in the size of the frontal lobe in the brain of man. The relative increase of this lobe causes the posterior lobes to be thrust further backwards over the cerebellum, the fissure of Rolando to slant upwards and backwards instead of being vertical, as in the brain of the monkey, and the posterior limb of the Sylvian fissure to become longitudinal instead of slanting upwards, as in the Simian brain. It is likely that the large relative size of the frontal lobe in man is connected with his mental superiority over the monkey. The next difference we observe is the depth of the fissures in the brain of man as compared with that of the monkey. In the course of development the summits of the convolutions are first formed, and they alone are directly connected with the incoming and outgoing fibres of the cerebro-spinal conducting paths. The infolding of the cortex, which forms a sulcus, is a later result of development, and, as the superficial extent of the cortex becomes greater and greater, the deeper the sulci become, so the depth of the sulci may be taken to some extent as a measure of the development of the brain. The next point we note is the great complexity of the arrangement of the convolutions in the brain of man as compared with that of the monkey. The convolutions of the human brain are divided into *primary* or *fundamental* and *secondary* or *accessory*. The fundamental convolutions in man are distributed along the margins of the great longitudinal fissure and other primary fissures, like the Sylvian fissure and the fissure of Rolando, and their disposition corresponds closely with the arrangement of the convolutions in the brain of the monkey. The accessory convolutions of man, which are rather irregular in their distribution, may be regarded as structures superadded in the course of evolution. These convolutions are connected with the primary convolutions by arcuate fibres, and are not directly connected with the ascending and radiating fibres of the internal capsule, and consequently their activity is likely to be correlated with mental operations. Another remarkable feature in which the human brain differs from the brain of animals is the manner in which the Island of Reil is completely surrounded and hidden out of view by deep convolutions. This is brought about by

the large development of the posterior extremity of the inferior frontal, of the inferior extremities of the ascending frontal and parietal convolutions, and of the supra-marginal, angular, and temporo-sphenoidal gyri. The consequence of the large development of these convolutions is that the cortex is deeply folded over the Island of Reil, this fold forming a kind of hood, which has been named the operculum. It appears to me that the cortex of the Island of Reil, starting from the gray matter of the anterior perforated space, is the embryonic part of the cortex of the brain, just as the central gray column is the embryonic

FIG. 45.



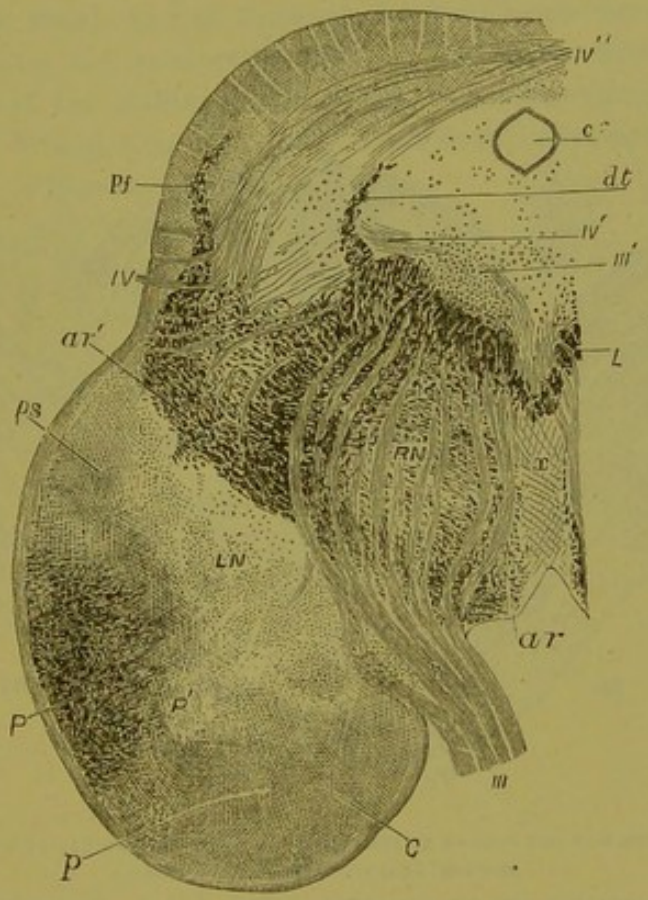
HORIZONTAL SECTION OF THE BASAL GANGLIA AND INTERNAL CAPSULE OF A NINE MONTHS' EMBRYO.

II, III, Second and third segments of the nucleus respectively; *NC*, Caudate nucleus; *TH*, Optic thalamus; *IN*, Island of Reil; *ps*, Peduncular sensory tract and optic radiations of Gratiolet; *P*, Fundamental, *P'*, Mixed, and *p*, Accessory portion of pyramidal tract; *C*, Fibres from the corpus callosum (?).

portion of the gray matter of the spinal cord. On the supposition that the portion of the Island of Reil which lies in the line of distribution of the Sylvian artery is the embryonic portion of the convolutions of the motor area of the cortex, it may be expected that the earlier formed portions of these convolutions will be thrust upwards towards the great longitudinal fissure, while the later formed portions will approach nearer and nearer to the root of the artery. According to this supposition, therefore, the fundamental portions of the convolutions supplied by the Sylvian artery will be found near the great longitudinal fissure, and the

accessory portion low down near the root of the artery, the last portion being formed by the operculum. This opinion corresponds closely with the topographical distribution of the motor centres as determined by experiment and pathological observation. The movements of the muscles of the trunk, which must be regarded as the fundamental movements, are regulated from the marginal convolutions of the longitudinal

Fig. 46.



Transverse Section of the Crus Cerebri on a level with the anterior pair of Corpora Quadrigemina, from a nine months' embryo. (Modified from Krause.)

cc, crusta; *P*, pyramidal tract; *p*, accessory portion of the pyramidal tract; *LN*, locus niger; *RN*, red nucleus of the tegmentum; *L*, posterior longitudinal fasciculus; *ar* and *ar'*, upward continuation of the internal and external portions respectively of the anterior root-zone of the spinal cord; III, third nerve; III', nucleus of the third nerve; IV, fourth nerve; IV', nucleus of the fourth nerve; IV'', crossing of the fibres of the fourth nerve to opposite sides; *dt*, descending root of the trigeminus; *cc*, aqueduct of Sylvius; *x*, crossing of the fibres of the superior peduncles of the cerebellum; *pf*, fasciculus of medullated fibres proceeding from the fillet to the anterior pair of corpora quadrigemina.

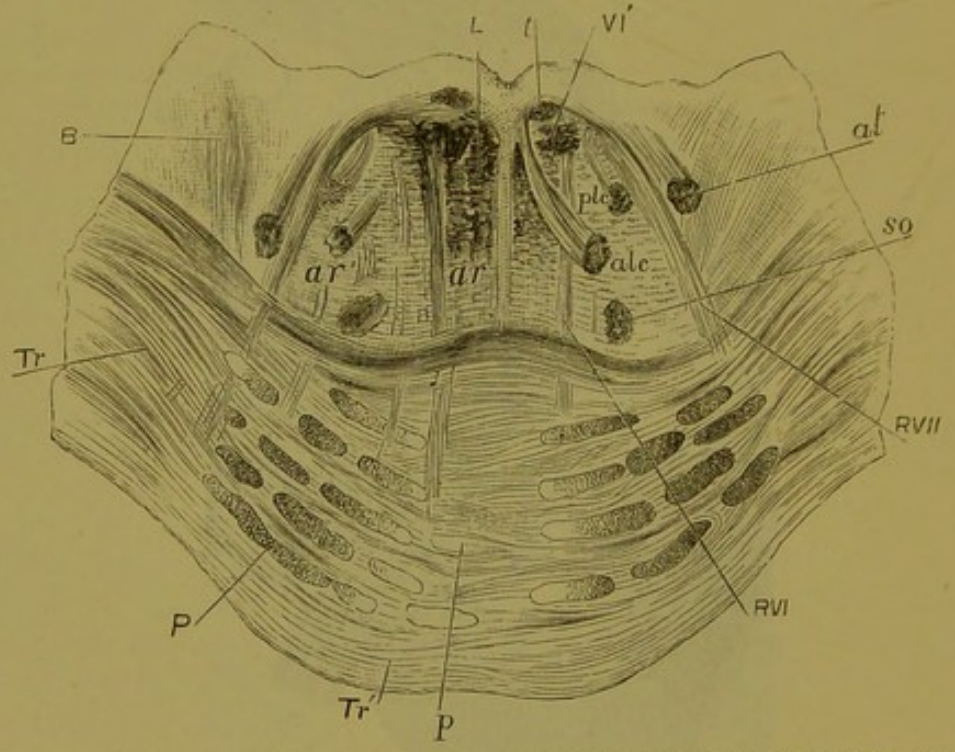
sulcus; while the movements of the lips and tongue, and those of the small muscles of the hand, which are the chief accessory movements, are regulated from the operculum.

In endeavoring to discriminate between the fundamental and accessory portions of the conducting paths, the most efficient practical test

is to regard all those fibres which are fully medullated at birth as belonging to the fundamental system; and those which are non-medullated at birth, but afterwards acquire this sheath, as belonging to the accessory system.

In following the course of the pyramidal tract downwards from the cortex, its fibres come together to form one bundle in the anterior two-thirds of the posterior segment of the internal capsule. When the internal capsule is examined at birth the pyramidal tract consists of a

FIG. 47.



TRANSVERSE SECTION OF THE PONS ON A LEVEL WITH THE ABDUCENS AND FACIAL ROOTS, FROM A NINE MONTHS' EMBRYO. (Modified from ERB.)

The right half represents a section made a little lower than the left. *P*, pyramidal tract; *p*, accessory portion of the pyramidal tract; *Tr* and *Tr'*, transverse fibres of the pons; *so*, superior olivary body; *alc* and *plc*, anterior and posterior nuclei of the lateral column respectively, representing the nucleus of the facial nerve; RVII, root of the facial nerve; VI', nucleus of the sixth nerve; RVI, root of the sixth nerve; *at*, ascending root of the trigeminus. *B*, The internal division of the peduncle of the cerebellum as it passes from the cerebellum; *L*, posterior longitudinal fasciculus; *ar* and *ar'*, the upward continuation of the internal and external divisions of the anterior root-zone of the spinal cord; *t*, fasciculus teres.

posterior portion (Fig. 47, *P*) in which all the fibres are medullated, a middle portion (Fig. 45, *P'*) in which medullated and non-medullated fibres are mixed, and an anterior portion (Fig. 45, *p*) in which all the fibres are non-medullated. In the crusta the medullated fibres lie in the outer portion of the middle third (Fig. 46, *P*), the mixed fibres in the inner portion of the middle third (Fig. 46, *P'*), and the non-medullated fibres in the inner third (Fig. 46, *p*). In the pons the medullated

fibres lie in the anterior and external longitudinal bundles (Fig. 47, *P*), and the non-medullated lie in the anterior and internal bundles (Fig. 47, *p*), while the bundles of mixed fibres lie between these. In the anterior pyramid of the medulla the medullated fibres occupy the posterior and external parts of the pyramid (Fig. 28, *P*), and the non-medullated the anterior and inner side (Fig. 28, *p*), while the area of mixed fibres lies between them. It is probable that the fibres of the medullated area form the conducting paths of the movements of the muscles of the trunk and of those of the lower extremities, the fibres of the mixed area the conducting paths of the movements of the hand, and the fibres of the non-medullated area the conducting paths of the movements of articulation and facial expression, movements which are not organized until some time after birth.

CHAPTER III.

GENERAL MORBID ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM.

I. GENERAL MORBID ANATOMY.

Disease of the nervous system is caused (1) by defects in its development or evolution; or (2) by a reversal of the process of evolution, or, in other words, by dissolution.

1. Defects of Development.

a. Congenital Defects of Nerves.

When portions of the body are incompletely formed, the nerves are defective or absent. When, for example, the nose is arrested in its development, and when no eyes have been formed, not only are the optic nerves absent, but the third, fourth, and sixth pairs are also deficient. In a cyclops, in which the face was almost entirely wanting, no facial nerve could be found, and when the tongue is wanting the lingual branches of the fifth and the hypoglossal nerves are absent. In monsters in which one or more of the extremities are imperfect, there is a corresponding deficiency in the nerves of that limb or limbs.

b. Congenital Defects of the Spinal Cord.

(1) *Amyelia*, or absence of the spinal cord. Only met with when the brain is also absent.

(2) *Atelomyelia*, or imperfect development of the spinal cord. The upper end of the cord is lacking or imperfectly developed, the brain being also absent (*anencephalia*), or the head defective (*acephalia*). The medulla oblongata is absent or exists only in a rudimentary form.

(3) *Diastematomyelia* is a condition in which the two lateral halves of the cord either do not unite, or unite only throughout a portion of their extent. This malformation occurs with anencephalia.

(4) *Diplomyelia*, or duplication of the spinal cord, appears in the various forms of double monsters.

(5) *Anomalies in the length and thickness* of the cord are sometimes observed, the organ being sometimes thick and voluminous, and at other times thin and small. It descends at times to the third lumbar vertebra, and ends at other times opposite the eleventh or twelfth dorsal. A small size of the spinal cord has been met with in hereditary ataxia.

(6) *Asymmetry of the gray substance* has been observed in cases of pseudo-hypertrophic paralysis, and more especially in cases of congenital absence of one or more of the extremities.

(7) *The pyramidal tracts* are absent in anencephalous monsters, and imperfectly developed in some cases of congenital *parencephalus*. Each anterior pyramid of the medulla may also send the mass of its fibres into the spinal cord, either entirely crossed or only partially crossed, or down the anterior columns almost entirely uncrossed.

(8) *Hydromyelus congenitus*, or *hydrorrhachis interna*, is a condition in which there is a congenital enlargement of the central canal of the cord, which is converted, in the slighter degrees of the affection, into a cavity varying from the size of an ordinary knitting-needle to that of a crow's quill. The dilated canal may extend the whole length of the cord, be restricted to certain portions, assume a moniliform appearance, or present the appearance of a double canal by the growth of adhesions between the anterior and posterior walls. In the higher grades of congenital hydromyelus the spinal cord either disappears entirely, or becomes split into two halves for a greater or less distance, while the cavity of the central canal communicates freely with the cavity of the spinal arachnoid; the hydrorrhachis interna is then merged into hydrorrachis externa, as not infrequently happens in spina bifida.

(9) *Spina bifida* consists of an abnormal accumulation of fluid within the cavity of the spinal arachnoid, associated with a greater or less deformity of the spinal canal.

c. Congenital Malformations of the Skull and Brain.

(1) *Anencephalia.*—The upper portion of the skull and brain is entirely absent, and is occasionally associated with amyelia.

(2) *Hemicrania.*—The anterior portion of the skull is absent, and the brain deficient.

(3) *Hemicephalia.*—The lateral half of the brain and skull is deficient.

(4) *Notencephalus.*—The upper part of the skull is deficient, and the vertebral column is not entirely closed in, the brain developing in the vertebral canal instead of in the skull.

(5) *Hydrencephalocele.*—The bones of the skull are deficient, an opening being left through which a soft fluctuating tumor projects. The walls of the tumor consist of the soft coverings of the skull and the distended membranes of the brain. The tumor communicates with the general ventricular cavity of the brain, and can generally be emptied on steady pressure.

(6) *Encephalocele.*—The bones of the skull are deficient, and through the opening thus caused a portion of the brain projects, forming a broad, flat, solid tumor. The tumor frequently occupies the forehead, orbit, or side of the nose.

(7) *Parencephalus.*—A portion of the hemisphere is deficient, but, as it is not always caused by a congenital arrest of development, it will be subsequently described.

(8) *Microcephalus* is a condition in which the brain as a whole, and probably the nervous system generally, is arrested in its development. It occurs in idiots, and the brain is not only small in size, but its convolutions also retain the simplicity of arrangement which characterizes the Simian brain.

(9) *Neuropathic constitution* is a condition in which the nervous inheritance of the subject is shown to be defective by a functional incapacity to respond adequately to the ordinary conditions of life, but without this defect being apparent to our means of investigation in the structure of the nervous system. The predisposition to a particular disease may be *special* and *direct*, or *general* and *indirect*. Migraine, for example, may be transmitted directly from a mother to her daughter, and the attacks of headache generally appear in both about puberty. In such a case the transmission is direct from mother to daughter, and the transmitted disease is limited to a particular affection. But the inheritance in this case is also *immediate* as well as direct and special, inasmuch as the disease is directly transmitted from parent to child. But, in a large number of diseases, the inheritance is *remote*, the transmitted affection being derived not immediately from the parent, but from a grandparent, or a still more remote ancestor. The phenomena of *atavism* or *reversion*, as the remote inheritance is called, are, indeed, very conspicuous in the transmission of a large number of nervous diseases.

In other cases a vulnerable nervous system, named a *neurotic* or *neuropathic* disposition, is transmitted, which favors the appearance of nervous disease. In such cases the tendency to any particular disease is *indirect;* and it is also *general*, inasmuch as one member of a family may suffer from neuralgia, another from chorea, paralysis, hysteria, epilepsy, or insanity, while others may manifest a tendency to uncon-

trollable alcoholic excesses. At other times the predisposition to nervous disease is still more *indirect*. One man dies of disease of the brain, as his father did before him, at a particular age, but it is because both have inherited gout, which has induced early arterial degeneration, which in its turn has ended in rupture of a bloodvessel in the brain. In another family several children die about the same age after convulsions and coma, but it is because they have inherited a strong tendency to tubercular disease, and not from inherent weakness of the nervous system. The inherited tendency to certain nervous diseases is strongly influenced by age, sex, and race; but this point need not detain us at present.

2. Dissolution of the Nervous System.

The law of dissolution is the converse of evolution, and may be enunciated as a progressive disintegration both of structure and function, in which there is a passage from the complex to the simple, from the multiform to the uniform, and from the special to the general. Before discussing further the application of the law of dissolution to the phenomena of the diseases of the nervous system, it will be useful to classify the lesions to which the nervous system is liable—the term *lesion* being used here as a generic expression to indicate any morbid alteration of tissue, whether this alteration be or be not attended by such structural changes as can be recognized after death by our means of research.

Classification of Lesions of the Nervous System.

Lesions of the nervous system may be classified (1) according to their nature; (2) according to their form; (3) according to the functional disturbances they produce.

(1) Classification According to the Nature of the Lesion.

(1) *Inflammation.*—Every part of the nervous system is subject to inflammation, which, like inflammation of other tissues, may be acute, subacute, or chronic, with respect to its course and development. When the affection is acute it generally ends in complete disintegration of the affected tissue, which, on being mixed up with fluid and morphological elements effused from the bloodvessels, presents a pulpy mass technically called *softening*. When the inflammatory process is chronic the tissue undergoes degeneration; but, inasmuch as degeneration may occur independently of inflammation, both kinds may be included in one group.

(2) *Degenerations.*—Degenerations of nervous tissues are of various kinds; but inasmuch as in all of them the nervous tissue, instead of undergoing softening as in acute inflammation, becomes somewhat denser than usual, they are called *scleroses.* From the color of the altered tissue, it is sometimes called *gray degeneration.* Both inflammations and degenerations include affections which have begun in the connective tissues or neuroglia, the vessels and their adventitiæ, or the blood itself, as well as those which are primarily of parenchymatous origin.

(3) *Vascular Lesions.*—Besides the vascular lesions which accompany all inflammatory and degenerative processes, other very important diseases in connection with the vessels must be mentioned.

(*a*) *Hyperæmia and Anæmia.*—The vessels are at times actively dilated so that an undue quantity of blood is sent to portions of the nervous system, while they are at other times contracted so that the normal quantity is diminished. The hyperæmia may at times be active and due to high arterial tension and active dilatation of the arterioles, while at other times it is passive, and is then caused by some obstruction of the blood along the large veins of the body. The anæmia to which the nervous system is subject does not differ from the anæmia of other organs, and may, therefore, be due to an alteration of the quality as well as the quantity of the blood.

(*b*) *Hemorrhage.*—Rupture of vessels with consequent hemorrhage is a very common cause of disease of the central nervous organs, and especially of the cerebrum. The rupture may at times be due to accidental injury, but more frequently it occurs in the degenerative period of life, and is then caused by various degenerations of the coats of the vessels, such as atheroma and the fibrosis which accompanies Bright's disease.

(*c*) *Embolism and Thrombosis.*—The morbid processes of embolism and thrombosis, when they occur in the nervous system, are essentially the same as in any of the other organs of the body. The sudden arrest of the circulation, caused by obliteration of an artery, causes intense anæmia and loss of function of the part to which its branches are distributed. The centre of the ischæmic region usually undergoes necrobiosis, and its substance becomes altered into a soft pulpy mass closely resembling inflammatory softening.

(4) *Toxic Lesions.*—Various chemical agents circulating in the blood induce morbid alterations of the nervous tissues.

(5) *Traumatic Injuries.*—Wounds, contusions, and other traumatic influences cause so many alterations of nervous tissues that their results deserve to be mentioned amongst the morbid lesions of the nervous system.

(6) *Compression of Nervous Tissues.*—The nervous tissues are frequently subjected to sudden or gradual compression from various causes. One of the most frequent causes of compression is the gradual encroachment on the tissues of new formations, no matter whether the growth be outside the nervous tissues or *extra-neural*, or in the substance of the tissues or *intra-neural*. Other causes of compression are fractures of the cranium, dislocations, fractures and curvatures of the vertebræ, the formation and subsequent enlargement of abscesses and aneurisms, and the growth of cysticerci and other parasites. Inflammatory effusions and thickenings of the membranes of the brain or cord, or of the sheaths of nerves, also injure the nerve tissues by compression as well as by extension of the morbid process to the nervous tissues themselves.

(2) Classification According to the Form of the Lesion.

(1) *Circumscribed or Focal and Diffused Lesions—Systematic Diseases.*—When a lesion is circumscribed within definite limits it is called a *focal lesion*, and when it extends over an indefinite area with irregular limits it is called a *diffused lesion*. When the lesion is limited to a portion of the nervous system which possesses a distinct functional unity, it is called a *system-disease* or a *systematic lesion;* and when several physiological tracts are implicated, it is called a *mixed* disease, or *indiscriminate* lesion. A lesion limited to the pyramidal tract in the spinal cord is a good example of a systematic lesion, and a transverse myelitis of an indiscriminate lesion.

(2) *Molecular, Molar, and Histological Lesions.*—In order to study these diseases under the simplest conditions, let us suppose that the sciatic nerve of a frog is isolated, with the gastrocnemius muscle attached. On being stimulated by a strong faradic current, the muscle immediately contracts; but a second shock through the nerve is powerless to induce a contraction—the nerve is paralyzed. During the passage of the nerve from almost perfect health to complete, though temporary, paralysis, the change which it has undergone is such as cannot be detected by the most refined chemistry, or by the aid of the highest powers of the microscope. In such case we assume that the molecules of the axis-cylinder have come to a condition of stable equilibrium; hence the cause of the loss of function may be described as a *molecular lesion*. It need scarcely be added that a molecular lesion may also give rise to excess, as well as diminution, of functional activity. The lesion which I have called *molecular* has also been termed *functional*, from the fact that the part affected, while giving rise to definite functional disturbances, does not present any apparent structural changes.

But if a portion of the sciatic nerve be crushed or cut, the conductivity

of the nerve is destroyed at the point of injury, and a faradic current applied to the nerve on the central side of the injury will not cause the muscle to contract. The loss of function in this case is caused by a lesion, which can be recognized by the naked eye; hence it may be called a *molar lesion*. Dr. Hughlings Jackson has proposed to call the molar lesion by the name of "coarse disease," and the molecular lesion by the name of "fine disease"—names which, at least, possess the merit of explaining themselves. Between the fine or the molecular lesion on the one hand, and the coarse or molar lesion on the other, another variety may be interposed. When the morphological elements of the nervous tissues themselves, or of the tissues by which the nervous elements are surrounded, undergo alterations which can be recognized by the aid of the microscope, the morbid change of structure may be called a *histological lesion*.

(3) Classification according to the Alterations of Function Produced by the Lesion.

(1) *Irritative and Depressive Lesions.*—When the morbid alteration is attended during life with excess of functional activity, it is inferred that the lesion is one of an *irritative character;* or, in other words, it is inferred that the irritability of the cells and fibres of the part affected is increased. The opposite condition, in which the irritability is diminished or abolished, deserves a special name and may be called a *depressive lesion*.

(2) *Discharging and Destroying Lesions.*—The morbid alterations which are attended by paroxysmal and excessive liberations of energy have been called by Dr. Hughlings Jackson *discharging* lesions. We have seen that the nerve cells are the main generators and accumulators of energy, hence these lesions always implicate the gray substance, although it is not always easy to draw a sharp line of distinction between discharges of energy from gray substance and those which result from irritation of nerve fibres. When the affection is accompanied by a distinct destruction of nerve tissue, such as occurs in hemorrhage into the substance of the brain, Dr. Hughlings Jackson has named it a *destroying lesion*.

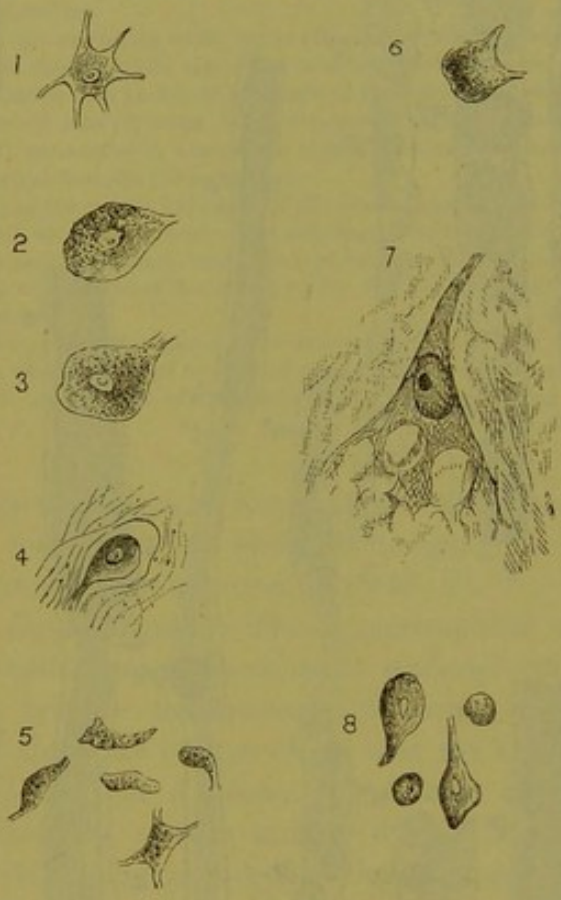
(1) *Dissolution of Nerve Cells and Fibres.*

a. Morbid Changes of the Ganglion Cells.

(1) *Hypertrophy.*—In acute inflammation the ganglion cells become swollen, their contents are cloudy and granular, and often pigmented, and their processes also participate in the same changes (Fig. 48, 2).

(2) *Shrinking.*—In the acute diseases of the gray substance of the cord the fluid contents of some of the ganglion cells appear to escape, the cell-wall shrinks around the nucleus and a small quantity of yellow pigment, and the cell is contracted into a shrivelled mass, which only presents slight traces of its former structure (Fig. 48, 4). At a

FIG. 48.



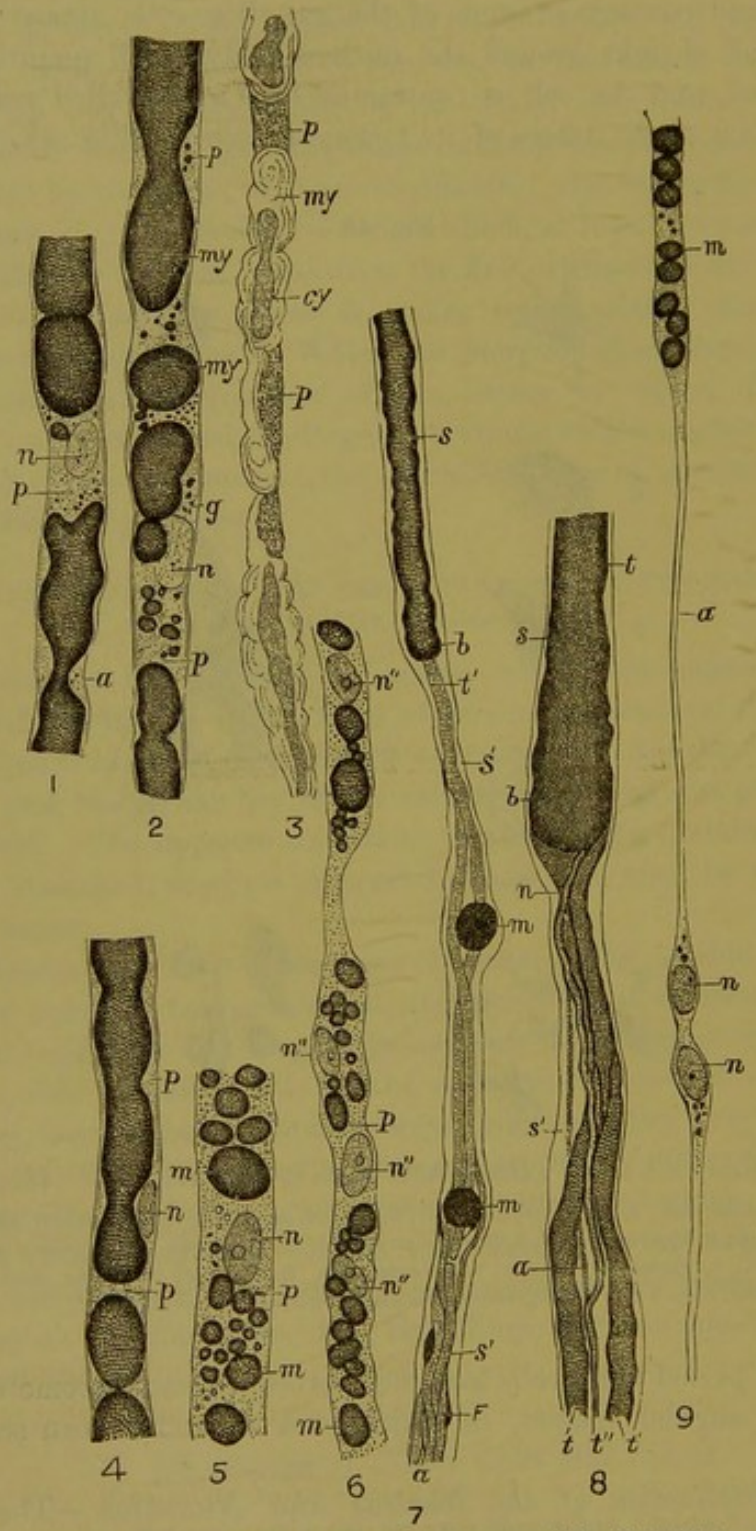
GANGLION CELLS OF THE ANTERIOR GRAY HORNS OF THE SPINAL CORD. (YOUNG.)

1, Healthy caudate cell; 2, Hypertrophied cell; 3, Yellow degeneration (the yellow color cannot be represented here); 4, Shrivelled cell; 5, Chronic atrophy, a group of cells from a case of pseudo-hypertrophic paralysis; 6, Pigmentary atrophy; 7, Vacuolation, from a case of canine chorea (GOWERS); 8, Chronic atrophy, from a case of progressive muscular atrophy—"yellow atrophy."

subsequent period these cells lose their processes and become converted into small angular masses, in which even a nucleus can scarcely be detected.

(3) *Multiplication of the Nucleus and Nucleolus.*—The nucleus and nucleolus may at times be observed either to have divided into two, or to exhibit an hour-glass contraction indicating that the process of division has commenced.

FIG. 49.



ALTERATIONS IN NERVE FIBRES AFTER SECTION. (After RANVIER.)

1 and 2. Two nerve fibres from the peripheral segment of the sciatic nerve of a hare fifty hours after section, examined after maceration for twenty-four hours in a solution of perosmic acid; (*n*) nucleus of interannular segment, swollen and detached from the sheath of Schwann; (*p*) mass of protoplasm, in which fat granules and drops of myeline (*g* and *my*) may be observed. The medullary sheath is completely interrupted at the level of the nucleus, while at *a* it has undergone strangulation.

3. Appearance presented by the peripheral fibres four days after section of the sciatic nerve of a hare, originally hardened in a solution of bichromate of ammonia and stained by picrocarmine; (*cy*) fragments of the axis-cylinder retracted, somewhat tortuous and embedded in a mass of myeline (*my*); (*p*) protoplasm swollen and granular.

4. Fibre same as 3, but originally colored by picrocarmine after maceration in perosmic acid; (*n*) nucleus compressing and partially interrupting the medullary sheath and the axis-cylinder; *p*, protoplasm.

5 and 6. Fibres from the peripheric portion of the sciatic nerve of a pigeon three days after section (same method of preparation as 4). 5. Median portion of an interannular segment presenting a single swollen nucleus (*n*) surrounded by a mass of protoplasm (*p*). 6. Presents four nuclei (*n'' n'' n'' n''*) in a single interannular segment. The protoplasm (*p*) which surrounds them is not segmented, but contains masses of myeline in its interior.

7 Fibres from the central end of the sciatic nerve of a hare ninety days after section (same method of preparation as 4). Dark upper portion represents primitive nerve fibre surrounded by the sheath of Schwann (*s*), and terminating by a knobby enlargement of its medullary sheath (*b*). From the extremity of this termination a second tube (*t'*) issues, which divides and subdivides until it forms a bundle of very fine medullary fibres (F), surrounded by a secondary sheath (*s'*) emanating from the sheath of Schwann; *m*, drops of myeline derived from the old nerve fibre.

8. A large nerve fibre of the central extremity of the pneumogastric nerve of a hare seventy-two days after section—maceration in perosmic acid. The medullary sheath (*t*) terminates by a knobby extremity (*b*), and from this extremity secondary medullated nerve tubes (*t' t''*) issue, as well as fibres without myeline; (*s*) the sheath of Schwann of the primary fibre forming secondary nerve sheath (*s'*) for the nerve fibres which issue from it.

9. A nerve tube of the peripheric segment of the pneumogastric of a hare six days after section. The portions *a a*, which are neither occupied by drops of myeline nor by nuclei, are collapsed, and the tube is contracted at this level, *n n*, nuclei of the interannular segment, having undergone proliferation; *m m*, drops of myeline.

(4) *Vacuolation.*—Two or three large spherical air-spaces named *vacuoles*, may sometimes be observed in ganglion cells which have undergone a granular degeneration (Fig. 48, 7).

(5) *Colloid Degeneration.*—The hypertrophied cells of the early stage of inflammation may subsequently undergo colloid degeneration. Their processes become transparent, glistening, and brittle, while a large portion of them are broken off so that the cells assume a rounded form. The cell-wall has a glassy appearance, and assumes brilliant tints when stained by various aniline dyes. The colloid appearance may be the result of post-mortem changes, and consequently considerable caution must be exercised in accepting them as evidence of disease.

(6) *Pigmentary Degeneration.*—The best examples of pigmentary degeneration are seen in the chronic diseases of the spinal cord. The cell-wall becomes contracted around a mass of dark granular pigment, the nucleus and nucleolus are indistinct or obliterated, the processes are atrophied, and many of them have disappeared (Fig. 48, 6).

(7) *Atrophy.*—In chronic diseases the cell-wall becomes dense and contracted, the processes are broken off, and the remnant of the cell is contracted into a small angular mass, without recognizable nucleus or nucleolus, and finally all traces of the cell may be lost (Fig. 48, 5 and 8).

(8) *Calcareous Degeneration.*—This form of degeneration is observed on rare occasions.

Although the illustrations of the morbid alterations of ganglion cells have been taken from disease of the caudate cells of the anterior gray horns of the spinal cord, yet essentially the same changes are met with in the ganglion cells of other parts of the nervous system, and notably in those of the cortex of the brain. It is scarcely necessary to point out how these various alterations conform to the law of dissolution. These alterations, taken as a whole, are a passage from the complexity and multiformity of the caudate cells with their numerous processes and well-defined connections to the simplicity and uniformity of the round cell without processes or definite connections.

b. Morbid Changes of Nerve Fibres.

(1) *Wallerian Degeneration.*—The most notable morbid alterations of nerve fibres are best studied in the peripheral ends of divided nerves. When the peripheral portion of a divided nerve is examined two days after section, the medulla of the divided fibres is found to be coagulated, opaque, granular, and broken up into cylindrical masses (Fig. 49, 1, 2). The nucleus (*n*) of the interannular segment has become increased in size, and contains a large and well-marked nucleolus. The protoplasm which surrounds the nucleus becomes so abundant and well developed at the level of the nucleus, that it fills the calibre of the nerve tube, and completely interrupts the medullary sheath. The protoplasm at this level, and at other points where it also accumulates, becomes filled with fine fat granules into which the myeline has been converted, and a similar granular débris may be observed outside the sheath of Schwann, and in the substance of the cells of the endoneurium. During the next two or three days the segmentation of the medullary sheath proceeds, and the cylindrical masses become broken up into globular masses (Fig. 49, 5, 6), which, at the end of the first week after section, are converted into drops of variable size, amongst which a progressively increasing number of fine fat granules may be observed. At this period the altered medulla occupies a larger space than in health, so that the fibres appear broader than usual, but their outlines are somewhat irregular and wavy. As the morbid processes advance the medulla becomes gradually converted into fat granules, which are finally absorbed. The axis-cylinder is said by some observers to persist for a long time after the medullary sheath has disappeared, but Ranvier asserts that the protoplasm collects at the level of the interannular nucleus to such an extent that it compresses, and finally intersects the axis-cylinder (Fig. 49, 3, *p c y*), which may also at a subsequent period be cut across by the accumulation of protoplasm at other levels. On the fourth day after section the nucleus, which is situated near the

middle of an interannular segment, contains a large and distinct nucleolus (Fig. 49, 5, *n*), which may present an hour-glass contraction, or be divided into two. After a time the nucleus exhibits a similar transformation, and ends by becoming completely divided into two nuclei, each of which may subsequently undergo subdivision, and these four nuclei may be found in one interannular segment (Fig. 49, 6, *n'' n'' n'' n''*). At a later period of the degenerative process the greater portion of the medulla is absorbed, although some globular masses may accumulate at certain points in the length of the fibre (Fig. 49, 6, *m*), the process of multiplication of nuclei ceases, and even the axis-cylinder disappears from considerable portions of the length of the fibre. The result of this process is that the sheath of Schwann is completely empty of its contents at certain points, and collapses so that the degenerated fibre appears very slender (Fig. 49, *a a*). The calibre of the tube is distended at intervals by elongated nuclei arranged in a series (Fig. 49, 9, *n n*), by fragments of the axis-cylinder, or by globular masses of altered myeline (Fig. 49, 9, *m*), and the degenerated nerve tube now appears as a delicate pale band with irregularly undulating contour. With the disappearance of the medullary sheath the degenerated nerve loses its white color and assumes a gray appearance, the fibres shrink, and the nerve looks small and wasted. This process is probably accompanied by proliferation of the cells of the endoneurium, or even of the perineurium, and in long-standing cases the newly found tissue undergoes cicatricial shrinking, or cirrhosis, rendering the texture of the degenerated nerve denser and adding to its atrophied appearance.

(2) *Regeneration of Nerves.*—Under favorable circumstances, the process of dissolution is arrested and a new evolution begins. If the ends of the divided nerve are maintained in apposition during the reparative process, it is probable that the axis-cylinders of the central and peripheral ends may become connected before any serious degenerative changes have occurred in the latter of the two segments. But when the ends are not in apposition, the nerve tubes of the peripheral segment become degenerated in their entire extent, and the subsequent regeneration is effected by an active growth of the nerve tubes of the central segment. Several ways are described by Ranvier in which the central ends give rise to new nerve fibres, but only one or two of the more common of these will be mentioned here. The central tube terminates by a slight enlargement of one of the nodes (Fig. 49, 7), and from this extremity a nerve tube (*t'*) issues, which, although thin, is furnished by a medullary sheath (*s'*) and interannular nucleus. This tube subdivides into two others of almost the same size as itself, and each of these in its turn subdivides into two new nerve tubes, so that the old sheath

of Schwann becomes distended by a bundle of new fibres (Fig. 49, 7, *F*). Rounded masses of altered myeline (*m*) are often observed to lie at intervals between the old sheath and the young fibres. At other times several nerve tubes (Fig. 49, 8, *t t′ t″*), some of them possessing distinct medullary sheaths, while others consist of naked axis-cylinders, issue from the extremities of the central fibres, and these also extend towards the periphery. These new fibres, on reaching the peripheral segment, penetrate for the most part into the interior of the degenerated tubes; but some of them, according to Ranvier, insinuate themselves between the old sheath and the substance of the endoneurium. The duration of the process of regeneration varies according as there is simple division of the nerve or a portion is resected. It is also influenced by numerous other circumstances, the most important of which is the length of the peripheral part of the divided nerve, restoration being so much the slower the longer that portion of the nerve is. Schiff found complete reunion of divided nerves in young animals in from seven to fourteen days, and Paget found in two cases of complete division of nerves traces of returning sensibility in fifteen days. Sensory functions are, however, restored considerably sooner than motor functions, and, after division of the facial nerve, the return of motor power in the facial muscles only takes place after the lapse of two or three months. If a portion of the nerve is resected the process of repair takes a much longer time, and if the portion of nerve removed exceeds two inches regeneration is not likely to take place. The nerve fibres of the white substance of the spinal cord and brain are found to undergo a degeneration essentially similar in kind to that just described as occurring in peripheral nerves. That degeneration of nerve fibres conforms to the law of dissolution, and their regeneration to that of evolution, is too obvious to require pointing out in detail.

(3) *Hypertrophy of the Axis-cylinder.*—In myelitis the axis-cylinders of many of the fibres appear on transverse section two or three times the normal size, but it is seen on longitudinal section that the swelling does not extend the whole length of the axis-cylinder, the fibre now presenting a varicose appearance.

(4) *Calcareous degeneration* of the fibres of the spinal cord has been exceptionally observed.

(2) *Dissolution of the Nervous Tissues.*

The morbid changes of the nervous tissues manifest themselves in (1) the parenchymatous elements; (2) the connective tissue or neuroglia; (3) the bloodvessels; and (4) the blood.

(1) Morbid Changes of Parenchymatous Elements.

The morbid changes of the parenchyma of the nervous system are the same as those already described as occurring in the cells and fibres, the kind of alterations which take place depending upon whether the process is acute or chronic, or it be caused by an irritative or degenerative lesion, or upon various other circumstances.

(2) Morbid Changes of the Neuroglia and Connective Tissue.

(*a*) *Hypertrophy and Hyperplasia of the Connective Tissue.*—In inflammation of nervous tissues the septa of the connective tissue become swollen, while its nuclei and those of the neuroglia are largely increased in number. It is also probable that the leucocytes, which have migrated from the vessels, may subsequently become organized, and thus increase the volume of the connective tissue.

(*b*) *Interstitial Sclerosis.*—When hyperplasia of the connective tissue has once taken place, the newly formed tissue may undergo cicatricial contraction, and thus lead to the destruction of the nervous elements, by a kind of *cirrhosis* or *interstitial sclerosis.*

(*c*) *Glüge's Corpuscles.*—These corpuscles are large globular cells which are distended with granular contents; they are met in the nervous system of the embryo in considerable numbers, but are only sparsely distributed in that of the adult, except in cases of disease, and then are supposed to be derived from fatty degeneration of the cells of the connective tissue and neuroglia, the white corpuscles of the blood, and the endothelial cells of the capsules of the ganglion cells of blood-vessels.

(*d*) *Amyloid Corpuscles and Colloid Bodies.*—Amyloid corpuscles (corpora amylacea) are small, round, concentrically laminated bodies, most of which turn blue or bluish-gray when acted on by iodine, and assume a beautiful blue tint on the addition of sulphuric acid. Colloid bodies are irregular masses, consisting apparently of changed myeline, and assuming beautiful tints on being stained with logwood or some of the aniline dyes. Neither of these bodies affords trustworthy evidence of disease, inasmuch as they may probably result from post-mortem mortem changes.

(*e*) *Deiter's cells* appear to be much increased in number in inflammatory diseases of the nerve centres.

(3) Morbid Alterations of the Vessels.

(*a*) *Intravascular Changes.*—The vessels are at times greatly distended with blood, but this distention may have occurred from the mode of dying, or from hypostatic congestion after death.

(*b*) *Capillary Extravasations.*—In the early stage of inflammation of nervous tissues the affected part assumes a reddish color, and becomes studded by a number of capillary extravasations, each about the size of a pin's head, these being sometimes so numerous that the part presents the appearance of a hemorrhagic infarct.

(*c*) *Thickening of the walls of the vessels* of the nervous system is found in chronic Bright's disease similar to that which occurs in the vessels of the body generally in that disease.

(*d*) *Perivascular Changes.*—The most important perivascular changes observed in disease of the nerve centres are caused by migration of the white corpuscles of the blood into the perivascular lymph spaces and surrounding tissues. The number of leucocytes surrounding a vessel may sometimes be so great as to constitute what has been called a *miliary abscess.*

(*e*) *Atheroma and Aneurism.*—The vessels of the brain are as subject to atheromatous changes as those of the body generally, and these changes are very prone to occur in syphilitic subjects at a comparatively early age. When the cerebral arteries are diseased the smaller branches often undergo saccular dilatations, which have been named by Charcot *miliary aneurisms.* They are said to result from a kind of arterial sclerosis of the nature of a chronic periarteritis, consisting of multiplication of the nuclei of the lymph sheaths and adventitia, with atrophy of the muscular coats. These aneurisms are liable to rupture, and thus give rise to massive hemorrhages in the brain. Atheroma of the vessels may also lead to aneurisms of the medium sized and larger arteries of the brain, which may compress and destroy the nervous substance like other tumors, or cause sudden destruction of large portions of it by rupturing and giving rise to massive hemorrhages.

(*f*) *Occlusion of Bloodvessels.*—In valvular diseases of the heart a fibrinous mass may be washed off from the left cavities of the heart or their valves, or from the pulmonary veins, or an atheromatous aorta, and may be lodged in one of the arteries of the brain, the left middle cerebral artery being the one most liable to be occluded. This process is termed *embolism.* The embolus sometimes consists of a cancerous nodule washed from the pulmonary vessels in cancer of the lungs, or of a syphilitic nodule which had projected into the interior of one of the arteries of the neck or brain. A cerebral vessel may be occluded by the local formation of a clot, a process which constitutes *thrombosis.*

(4) Morbid Changes of the Blood.

In cases of pyæmia, morbid products are conveyed in the blood, and metastatic abscesses may form in the brain and other parts of the ner-

vous system, and morbid changes are apt to occur in the nervous tissues in blood diseases like anæmia, leucocythæmia, and the specific fevers, and in those cases in which the blood contains a chemical poison like strychnine, lead, and alcohol.

(5) New Formations.

(*a*) *Neuromata.*—Growths in nerves may be divided into (j) true and (jj) false neuromata.

(j) *True neuromata* consist of a growth of nerve fibres mixed with a connective-tissue formation. True neuromata have been divided into two varieties according to the character of the nerve fibres found in them. In one form the fibres are medullated, and consequently it has been called by Virchow *neuroma myelinicum;* whilst in the other form the fibres are non-medullated, and the tumor has been named by the same author *neuroma amyelinicum.* True neuromata have also been divided into several varieties according to the amount and character of the connective-tissue basis and to the degree of vascularity, the more usual names applied to them being *fibro-neuroma*, *glio-neuroma*, *myxo-neuroma*, and *neuroma teleangiectodes.* True neuromata occur most frequently in spinal nerves, rarely in sympathetic nerves, and still more rarely in one of the cerebral nerves. They vary from the size of a millet-seed to that of the closed fist. Neuromata consisting of both gray and white matter have occasionally been found in the brains of lunatics, being situated on the surface of the ventricles.

(jj) *False neuromata* consist of tumors of various kinds, but in which there is no formation of nerve fibres, these fibres being indeed injured or destroyed by compression. The following false neuromata are met with: *fibroma*, often forming small knots named *tubercula dolorosa;* *myxoma*, frequently met with in nerves, and sometimes containing cysts, when they are called *neuroma cysticum;* *glioma*, found in the auditory nerve; *sarcoma* occurs in nerves, and transitional varieties between it and fibroma and myxoma are not unfrequently observed; *carcinoma* occurs occasionally as a primary, but much more frequently as a secondary growth; *syphilitic gumma*, most frequently found in the cerebral nerves; and *lepra nervorum* appears as a diffused, more or less fusiform swelling of the nerves.

The size of neuromata is extremely variable, being sometimes not larger than a mustard-seed, and at other times as large as a man's head; the majority range between the size of a bean and that of a hen's egg. The number of the tumors is as variable as their size. In some cases there is only a solitary tumor, while at other times a large number may

be present, either at a circumscribed spot or distributed over the body, the number in some cases being as high as from eight hundred to several thousands.

The nerve sometimes passes on one side of the tumor; at other times the tumor occupies the centre of the nerve; while in other cases the nerve runs directly into the tumor, the fibres breaking up into a kind of brush or pencil.

(*b*) *Gliomata* form tumors which vary in size from a cherry-stone to that of the closed fist; they may be localized in any part of the brain or spinal cord, but are most frequently found in the hemispheres of the brain. Gliomata consist of a matrix and an abundant admixture of round, oval, or stellate cells with granular contents and one or two nuclei, the structure of the tumor being like that of the neuroglia. When the cells are abundant the tumor is *soft*, vascular, of a grayish-red color, and infiltrates into the nervous tissues; but when the cells are relatively few, and the matrix, which is formed of fine fibrillæ or a dense reticulum, is abundant, the tumor is *hard*, not very vascular, white in color, and it is more or less circumscribed, although never encapsulated. The hard gliomata are allied in general characters to the fibromata, and intermediate forms are met with which are termed *fibro-gliomata*. Transitional forms are also observed between gliomata and sarcomata, named glio-sarcomata. At other times gliomatous tumors undergo a mucoid degeneration, and they then resemble *myxomata;* while some of them are so richly supplied by bloodvessels that they have been named teleangiectatic *gliomata*, these being of great importance from their liability to hemorrhage.

(*c*) *Hyperplasia of the pineal gland* is very similar to glioma. It forms a solid, grayish-red, slightly lobulated tumor, which may grow to the size of a walnut, or larger. In old persons the tumor generally contains a large number of sand-like bodies.

(*d*) *Myxomata* take their origin, like gliomata, from an overgrowth of the neuroglia, but are rarer in the brain than in the spinal cord and peripheral nerves.

(*e*) *Solitary tubercle* forms a hard, rounded nodule, which varies from the size of a pea to that of a pigeon's egg, and, on section, the interior is seen to be yellowish and cheesy; while the outer cortex, which is only about a line in thickness, is of a reddish-gray color, and very vascular. These tumors are met with in all parts of the brain, but their favorite seat is the cortical substance of the cerebrum and cerebellum, close upon the cortico-medullary boundary. It is also one of the most frequent forms of tumor met with in the spinal cord. This

tumor is often multiple, but when it is solitary it may attain a considerable size.

(*f*) *Carcinomata* may grow on the outer surface of the dura mater, and, ultimately perforating the bones of the skull, form *fungus hæmatodes* of the dura mater. It may also grow from the under surface of the pia mater, and the growth may then be primary or secondary; but primary cancer in this situation is the more common of the two. Cancerous tumors of the brain may be single or multiple. They destroy the neighboring tissues by pressure and infiltration, and are usually surrounded by a zone of softened tissue, of about a line in breadth, in which active growth proceeds.

(*g*) *Cholesteatoma*, or pearl cancer, appears to be derived from the pia mater, and is usually situated in some hollow at the base of the brain. The tumor, on section, is hard, pearly, non-vascular, and composed of epidermic cells, arranged in concentric layers, which have undergone partly horny and partly fatty degeneration. The tumor is enclosed in a delicate fibrous capsule, and its surface presents a beautiful mother-of-pearl lustre. These tumors grow very slowly, and may remain for a long time without giving rise to symptoms.

(*h*) *Papilloma* of the cerebral pia mater is occasionally met with.

(*i*) *Syphilomata* may reach the size of a walnut or of a hen's egg, and are usually found near the surface of the brain, developing from the perivascular sheaths. They are only met with in the cord on rare occasions.

(*k*) *Sarcomata* appear as hard, slightly vascular, round, somewhat nodulated tumors. Every variety of sarcoma is found in the brain, and transitional forms between sarcoma and other tumors are named *glio-sarcoma*, *myxo-sarcoma*, etc. In some of the spindle-celled sarcomas the cells are arranged in concentric layers or nests, and consequently this form has been named "nested sarcoma."

(*l*) *Melanoma* is a pigmented sarcomatous tumor which springs from the pigment cells of the pia mater.

(*m*) *Lipoma* has occasionally been met with on the inner surface of the dura mater, and the raphé of the corpus callosum, and the fornix.

(*n*) *Psammomum* is a tumor with a basis of connective tissue, or sometimes of mucoid tissue, which is distinguished by containing calcareous concretions. It appears to be a calcareous deposit in tumors of widely different structure, the most frequent of these being nested sarcoma.

(*o*) *Osteomata* are the rarest of all intracranial growths.

(*p*) *Cystic growths* are caused by a portion of the posterior cornu of the lateral ventricle being cut off from the general cavity by dropsy

of the septum lucidum, and cystic degeneration of the pineal gland and pituitary body, and gliomatous tumors.

(*r*) *Angiomata* generally occur in the brain as a complication of other tumors such as glioma. Pachymengitis hæmorrhagica belongs to this class.

(6) Aneurism.

Aneurisms of the larger cerebral vessels are not very rare, and when they attain to a considerable size they give rise to the usual symptoms of an intracranial growth.

(7) Parasites of the Brain.

(*a*) *Cysticercus cellulosa* is met with in the parts of the brain which are richly supplied with blood, and the parasite is sometimes found in other parts of the body as well as in the brain. Cerebral cysticerci are usually enclosed in a soft capsule, in which the animal may be seen with the naked eye as a small tubercle, and its neck, with the characteristic hooklets, may be discovered on microscopic examination.

(*b*) *Echinococcus hominis* form cysts which often attain to a large size in the brain. In a case reported by Dr. Morgan the cyst weighed eighteen and a half ounces, and contained eighteen ounces of serum. They reach their greatest size in the hemispheres and the ventricles, especially in children before the fontanelles are closed. The cyst is composed of an external fibrous membrane, which encloses the parasites; its internal surface is lined by small buds, each about the size of a millet-seed, which are provided with the characteristic ring of hooklets.

3. Dissolution of the Nervous System (*continued*).

a. Massive Hemorrhages.

Massive hemorrhages generally destroy a considerable portion of the nervous system. In the recent condition the apoplectic focus forms a dark red soft clot, which is frequently mixed with the débris of the brain substance. The internal surface of the cavity is irregular and consists of torn shreds of cerebral tissue, and it is surrounded by a zone of variable thickness in which the tissue is softened by the inhibition of serum, and in which numerous punctiform hemorrhages are observed. If the patient survives, the tissues surrounding the blood-clot become softened partly by the imbibition of serum and partly from a retrograde fatty metamorphosis of the torn fragments of brain tissue, and the softened tissues, when mixed up with the blood clot, form a

dark chocolate-colored mass of the consistence of gruel. The *hæmatin* now becomes gradually absorbed, and the substance filling the cavity changes to a brighter red or saffron color. After a time a fibrous capsule forms round the clot, and the solid constituent becoming absorbed, a cyst is formed which contains at first a turbid, and subsequently a clear, limpid, or straw-colored fluid, having frequently suspended in it a film of loose spongy connective tissue. When the cyst is small, and the fluid is absorbed, the opposite walls may come in contact and adhere by a connective tissue, which usually contains a considerable amount of pigment, and gives rise to the appearance known as the apoplectic or hemorrhagic cicatrix.

b. Morbid Changes caused by Occlusion of Bloodvessels.

When a terminal artery becomes occluded the arterioles and vessels of the part are imperfectly nourished, and consequently their walls dilate and frequently rupture, the former causing œdema and the latter hemorrhage. The softened tissues are named red, yellow, or white softening according as a considerable amount, a moderate amount, or no blood is extravasated from the vessels.

c. Morbid Changes caused by Thickening of the Walls of the Arterioles.

The walls of the bloodvessels of the nerve centres become thickened in Bright's disease, and the nervous tissues surrounding the altered vessels undergo, in some cases, morbid changes more or less similar to those of chronic inflammation, and consisting of an increase of connective tissue and destruction of nerve cells. This condition may be named *vascular sclerosis*.

d. Inflammation of the Nervous System.

(1) Acute inflammation of nervous tissues is characterized in its first stage by congestion, capillary extravasations, and some œdema of the tissues. If the process proceeds further, it causes *softening* of the inflamed part. Inflammatory softening may be divided into (*a*) red, (*b*) yellow, (*c*) white, (*d*) gray, and (*e*) green or purulent softening.

(*a*) *Red Softening.*—The spot affected with red softening is soft, and swells up above the level of the surrounding surface on section. The affected tissue may be washed away by a gentle stream of water, or it may be diffluent. Numerous capillary hemorrhages may be observed, and the affected part assumes a tint which varies from rosy to deep red, reddish-brown, or chocolate.

(*b*) *Yellow Softening.*—As the disease progresses the affected parts become paler and softer, and the color changes to yellow, partly from diffusion and alteration of the coloring matter of the blood, and partly from degeneration of the medullary sheath.

(*c*) *White Softening.*—Owing to the continued process of fatty degeneration the color becomes progressively whiter, and the diseased portions assume a creamy or milky appearance, and on section the medulla swells up above the surface of the surrounding tissues.

(*d*) *Gray Softening.*—In consequence of the absorption of fat granules and nerve substance, the affected part gradually assumes a grayish color, and finally becomes smaller and more depressed than normal.

(*e*) *Green Softening.*—The white blood-corpuscles may migrate in such large numbers that the affected part is converted into a cavity containing a greenish purulent fluid, and constituting an *abscess.*

Microscopical Changes.—In the early stage of inflammation the arterioles and capillaries are dilated and distended with blood, while they may be enveloped in layers of migrated white and red blood-corpuscles. As the disease advances the walls of the vessels become thickened and studded with fat granules and granule cells, while the lymph sheaths are filled with granular or cellular exudation. The reticulum of the neuroglia is swollen and thickened, and filled with nuclei, lymphoid cells, and granule cells, while Deiter's cells are increased in number. The nerve fibres present irregular contractions and enlargements, the medullary sheath becomes broken down into globules and is finally absorbed, and the axis-cylinders are greatly swollen, while in the second stage the medullary sheaths are in a state of fatty degeneration, and the axis-cylinders are altered or destroyed. The ganglion cells are first swollen, the nucleus and nucleolus may be observed in process of division, while at other times they undergo vacuolation, the cell processes are swollen, cloudy, irregular in shape, and partly destroyed, and at a later period the cells lose their processes, and shrivel so as to become reduced to small angular masses without structure. When a *cicatrix* has formed, the affected spot is occupied by a dense connective tissue containing many nuclei and neuroglia cells, and numerous Deiter's cells. When *cysts* are formed they are surrounded by a more or less dense layer of connective tissue, and are generally traversed by a loose connective-tissue network.

The morbid appearances just described apply more particularly to those occurring in the spinal cord and termed *acute myelitis*, and in the brain and named *acute encephalitis*, but essentially the same changes are met with in acute inflammation of nerves. When the inflammation begins in the nerve fibres the process is named *neuritis*,

and when in the sheath of the nerve it is named *perineuritis*. In acute neuritis or perineuritis the vessels become enlarged and distended, and the nerve-trunk is swollen from serous, gelatinous, or fibrinous exudation. If the inflammation subside at an early date the effusion is absorbed before there is any destruction of the nerve fibres, and the healthy condition is reëstablished. If the inflammatory action is very acute and severe, both white and red corpuscles escape from the vessels, the color of the nerve becomes yellow or brownish-red, its tissues are infiltrated with sanguineous pus, abscesses may form around its trunk, and the entire structure may become completely disintegrated.

(2) *Chronic inflammation* causes the affected part, as a rule, to be atrophied, and unusually dense or in a state of *sclerosis*. When the white substance is the subject of chronic inflammation it assumes a gray color, and consequently the condition is sometimes called *gray* degeneration. When the inflammation spreads over a considerable area of tissue it is named *diffused* sclerosis; when it is limited to the embryological tracts of the cord it is named an *ascending* or a *descending* sclerosis, according as the process extends from below upwards or from above downwards; when it is limited to certain spots or foci it is named *circumscribed* or *insular* sclerosis; and when these spots are numerous and distributed in different parts of the nervous system, it is named *multiple* or *disseminated* sclerosis. The terms insular, multiple, and disseminated are applied indifferently to the same disease, because, when the sclerosis is limited to circumscribed spots, the spots are also multiple and widely distributed over the nervous system. When the sclerosis is more or less restricted to the embryological tracts the morbid process is supposed to begin in the nervous elements themselves, and consequently some anatomists have called this form of chronic inflammation *parenchymatous sclerosis*. This distinction is admissible theoretically, but it is not always easy to maintain it practically. A microscopical examination of a part in a state of sclerosis shows that the connective-tissue septa are thickened, and that the cells of the neuroglia are swollen and their nuclei multiplied. Deiter's cells are also increased in size and number, and in long-standing cases the neuroglia becomes converted into a dense fibrillated connective tissue in which a large number of nuclei are observed. The nerve fibres undergo changes more or less similar to those which occur in secondary degeneration of the fibres of the peripheral nerves. The medullary sheath undergoes granular and fatty degeneration, and is finally absorbed; but the axis-cylinder persists a long time, although it becomes swollen at certain points, and, on being viewed longitudinally, presents spindle-shaped enlargements. After a time the axis-cylinders also waste and disappear,

and nothing remains but a dense fibrillated connective tissue. The walls of the small arteries and veins become thickened, and their calibre is diminished in size. The lymph spaces are destroyed, or contain fat and pigment granules, while granule cells and corpora amylacea are found scattered through the diseased tissue.

The morbid appearances just described are met with in the nervous centres, but essentially similar alterations occur in nerves. In chronic *neuritis* and *perineuritis* the trunk of the nerve becomes irregularly vascular, and is enlarged in some places and atrophied in others. The sheath of the nerve is thickened, fibrous, and resisting, while it is frequently adherent to the adjacent tissues. In cases of perineuritis the nerve fibres are compressed by the exudation and disappear after a time, so that the structure of the nerve is supplanted by a band of connective tissue.

Segmental periaxillary neuritis, induced by Gombault in the peripheral nerves of guinea-pigs, is a parenchymatous neuritis in which only a part of the nerve fibre is implicated. A segment lying between two of the nodes of Ranvier is diseased, while those on each side of it may remain healthy. Several segments may, however, be affected in the course of one fibre. The medullary sheath and the protoplasm of the fibre are at first alone implicated in the morbid change, while the axis-cylinder remains for a long time uninterrupted. After a time the diseased segment may either be restored by the growth of a new medullary sheath, or the axis-cylinder becomes ruptured, and the peripheral end of the nerve then undergoes the Wallerian degeneration.

e. Degenerations of the Nervous System.

(1) Primary Degeneration.

The gray substance of the nervous system may apparently undergo a primary chronic degeneration in addition to the degeneration which is consecutive to inflammation. In the primary degeneration the nerve cells undergo atrophy and various forms of pigmentary degeneration. This form of degeneration occurs most probably in the gray substance of the anterior horns of the spinal cord in progressive muscular atrophy, and in the cortex of the brain in the chronic forms of insanity. It is possible that the white substance may also undergo a primary chronic degeneration, and the disease known as *progressive multiple neuritis* appears to be a primary degeneration of the peripheral nerves. The morbid changes which have been found in the nerves in this disease consist of partial or complete destruction of many of the nerve fibres. The perineurium of the individual bundles has also been found thick-

ened, and Leyden has observed an accumulation of fat cells between the bundles, and a deposit of pigment around the bloodvessels. Leyden regards this pigment as a hemorrhagic inflammation of the tissues between the nerve fibres, and he believes that the absence of any sign of multiplication of nuclei proves that the nerve fibres had become atrophied through the compression caused by the congestion of the tissues surrounding them.

(2) Secondary Degenerations.

The Wallerian degeneration of peripheral nerves has already been described, but we have now to point out that essentially the same process occurs in the conducting paths of the nerve centres. It has already been stated that Waller observed that when a mixed nerve is divided the peripheral portion degenerates throughout its whole course in a few weeks, while the portion attached to the cord does not degenerate. This statement, however, is only a portion of the truth. He also found that while the efferent fibres degenerate their whole length on division of the anterior root, that the peripheral portion of the afferent fibres do not degenerate on division of the posterior roots. On division of the posterior roots what takes place is that the peripheral portions which are attached to the spinal ganglion remain healthy, but the small central portion which is attached to the cord soon wastes. From these facts Waller concluded that the efferent fibres receive their nutritive influence from the caudate cells of the anterior horns, and the afferent fibres from the ganglia of the posterior roots, and he then formulated the general law that nerve fibres degenerate when they are separated from their trophic centres.

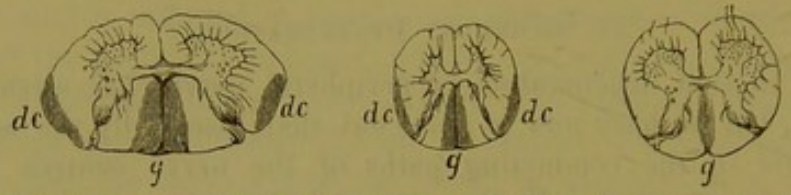
The central conducting paths also undergo degeneration in one direction after division or injury of them, and it may be confidently asserted that the degeneration occurs along the line of conduction of the fibres, some paths undergoing an *ascending* and others a *descending* degeneration.

(*a*) ascending degenerations.

The trophic centres of the columns of Goll and of the direct cerebellar tracts are situated at their inferior extremities, the ganglia of the posterior roots or the gray substance of the posterior horns forming the trophic centres of the former, and the cells of the vesicular columns of Clarke probably those of the latter. The lower limit of the degeneration of these columns will depend upon the position of the lesion of the cord which interrupts the continuity of the fibres, and the degeneration of the columns of Goll may extend from the lumbar

region of the cord up to the termination of the fibres in the cuneate nucleus, while the degeneration of the direct cerebellar tracts may extend from the inferior part of the dorsal region up to the external surface of the restiform bodies. In both of these degenerations the diseased area increases progressively in size from below upwards (Figs. 50, 51, 52). A case is reported by Dr. Gowers in which the lower

Fig. 50. Fig. 51. Fig. 52.



Transverse Sections of the Spinal Cord, from the middle of the Cervical Enlargement, middle of the Dorsal Region, and middle of the Lumbar Region, respectively, showing Ascending Degeneration of the Columns of Goll (*g*), and of the direct Cerebellar Tract (*dc*).

extremity of the spinal cord was crushed by a fracture of the spine, and in which, in addition to the ascending degeneration of the columns of Goll, a small area of degeneration was observed in each anterior root-zone in front of the lateral pyramidal tract, the latter being healthy on each side. Dr. Gowers suggests that these areas constitute a part of the sensory conducting paths. In lesions of the cauda equina, and sometimes after severe traumatic injuries of the sciatic nerve, the posterior root-zones as well as the columns of Goll undergo ascending degeneration in the lumbar and greater portion of the dorsal regions, but the degeneration becomes limited to the columns of Goll in the upper dorsal and cervical regions. In transverse lesions of the cord the posterior root-zones may also be found degenerated for a short distance above the lesion, but the evidence of degeneration soon ceases, probably because the fibres of these columns soon terminate in gray matter.

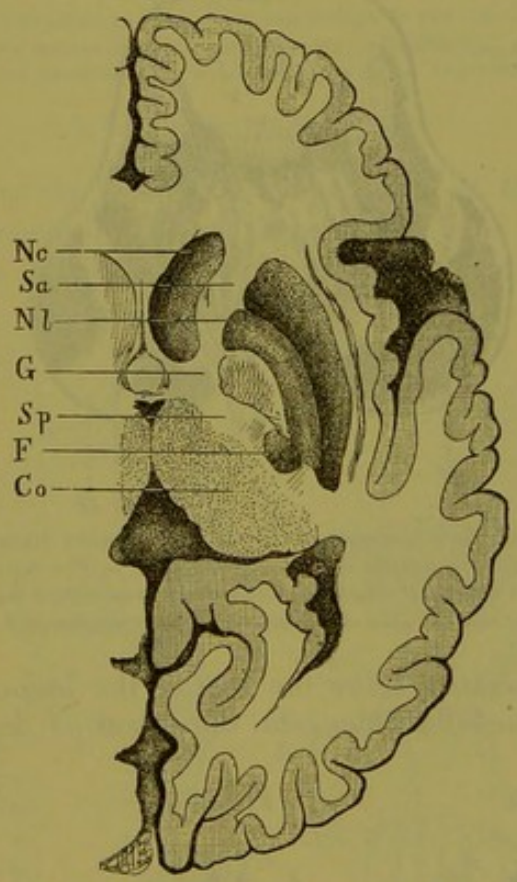
(*b*) DESCENDING DEGENERATION.

(j) *Sclerosis of the Pyramidal Tract.*

Lesions of the cortex of the brain, provided the whole thickness of the gray substance is implicated, are followed by descending degeneration of the fibres of the pyramidal tract which may be followed through the internal capsule, crusta, longitudinal fibres of the pons, anterior pyramids of the medulla oblongata on the anterior column of the same side as the lesion, and in the lateral columns of the cord on the side opposite the lesion. A descending degeneration of this kind has also been found in animals after extirpation of portions of the motor area of the cortex of the brain.

A focal lesion limited to the middle third of the internal capsule (Fig. 53, F) is followed by descending degeneration of the fibres of the middle third of the crusta (Fig. 54, D), and of a portion of the longitudinal fibres of the pons and the anterior pyramid of the medulla on the same side. The greater part of the degenerated fibres cross over at the lower end of the medulla oblongata to reach the lateral column of the opposite side of the cord (Figs. 55, 56, 57), while a few of them pass down the column of Türck on the same side.

FIG. 53.



HORIZONTAL SECTION OF THE RIGHT HEMISPHERE PARALLEL WITH THE FISSURE OF SYLVIUS. (CHARCOT.)
Nc, Caudate nucleus; Sa, Anterior segment of the internal capsule; Nl, Lenticular nucleus; G, Knee of the internal capsule; Sp, Posterior segment of the internal capsule; Co, Optic thalamus; F, A focal lesion in the middle third of the posterior part of the internal capsule.

In diseases of the spinal cord the degeneration is generally bilateral and symmetrical, and the positions occupied by the diseased areas at different levels of the cord are represented in Figs. 58, 59, 60. When the disease reaches to the medulla oblongata, the areas of degeneration are limited to the anterior pyramids.

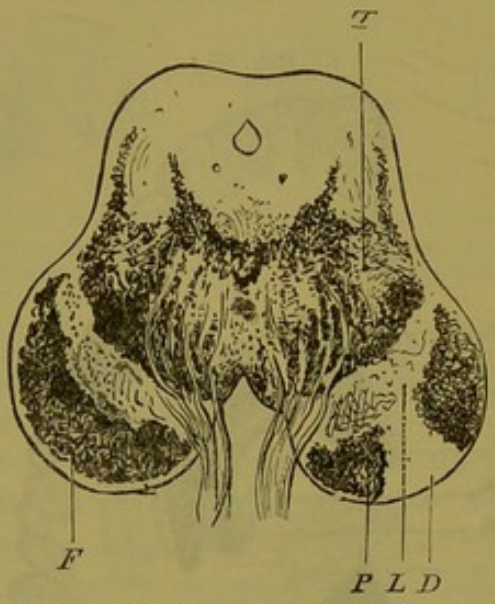
The portions of the pyramidal tracts which belong to the nuclei of

the medulla oblongata and pons are sometimes diseased in the absence of any affection of the spinal pyramidal tracts.

(jj) *Degeneration of the Fillet and Superior Peduncle of the Cerebellum.*

In a case reported by Homén, a focus of softening was found in the lateral half of the upper part of the pons, and the pyramidal tract of

FIG. 54.



HORIZONTAL SECTION OF THE CRURA CEREBRI IN A CASE OF SECONDARY DEGENERATION. (CHARCOT.)
T, Tegmentum; *F*, Crusta of the healthy side; *L*, Locus niger; *D*, The degenerated fibres, occupying about the middle third of the crusta; *P*, The fibres which undergo secondary degeneration only when the fibres of the anterior segment and the knee of the internal capsule are diseased

that side was degenerated below the seat of the lesion as far as to the lower end of the medulla oblongata, the streak of degeneration being

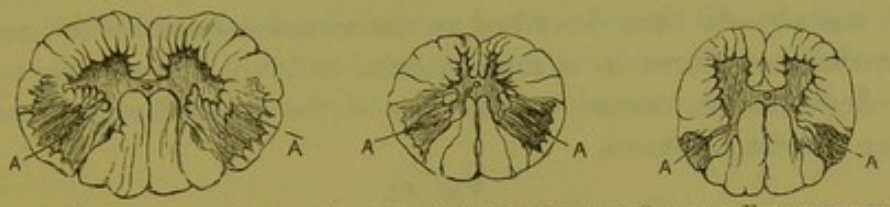
FIG. 55. FIG. 56. FIG. 57.

TRANSVERSE SECTIONS OF THE SPINAL CORD, FROM THE MIDDLE OF THE CERVICAL ENLARGEMENT, MIDDLE OF THE DORSAL REGION, AND MIDDLE OF THE LUMBAR REGION, RESPECTIVELY, SHOWING DESCENDING SCLEROSIS OF THE PYRAMIDAL TRACT IN THE LATERAL COLUMN SECONDARY TO A CEREBRAL LESION. (CHARCOT.)
A, A, A, Degenerated pyramidal tracts.

found in the part which lies to the inner side of the olivary body. In a case reported by Meyer, a focus of softening was found in the lateral half of the lower part of the pons, which embraced the nucleus of the

sixth nerve, the formatio reticularis, and the superior olivary body. Below the level of the lesion the internal division of the fillet and the olivary body were degenerated. The degeneration of the fillet could

FIG. 58. FIG. 59. FIG. 60.

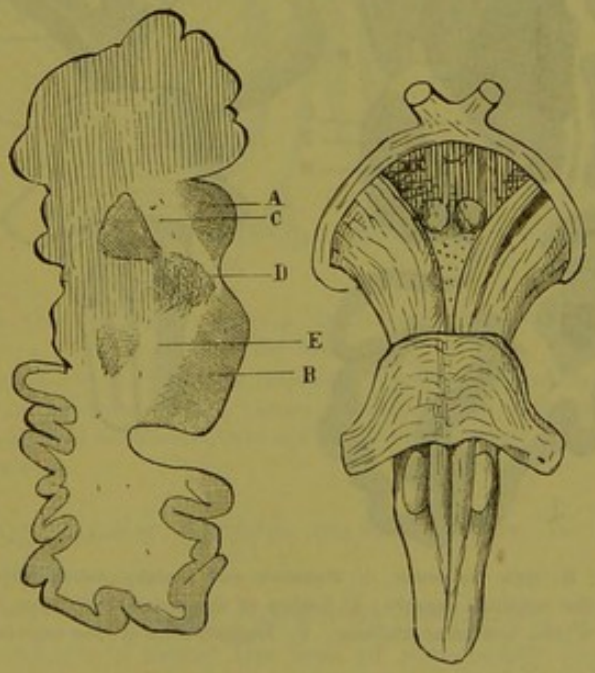


TRANSVERSE SECTIONS OF THE SPINAL CORD, FROM THE MIDDLE OF THE CERVICAL ENLARGEMENT, MIDDLE OF THE DORSAL REGION, AND MIDDLE OF THE LUMBAR REGION, RESPECTIVELY, SHOWING PRIMARY LATERAL SCLEROSIS OF THE CORD, OR SECONDARY TO A LESION HIGH UP IN THE CORD OR MEDULLA OBLONGATA. (CHARCOT.)

A, A, A, Degenerated pyramidal tracts.

be traced as far as the level of the decussation of the pyramidal tract, and it was represented immediately below this level by a small spot of degeneration near the periphery of the external division of the anterior

FIG. 61.



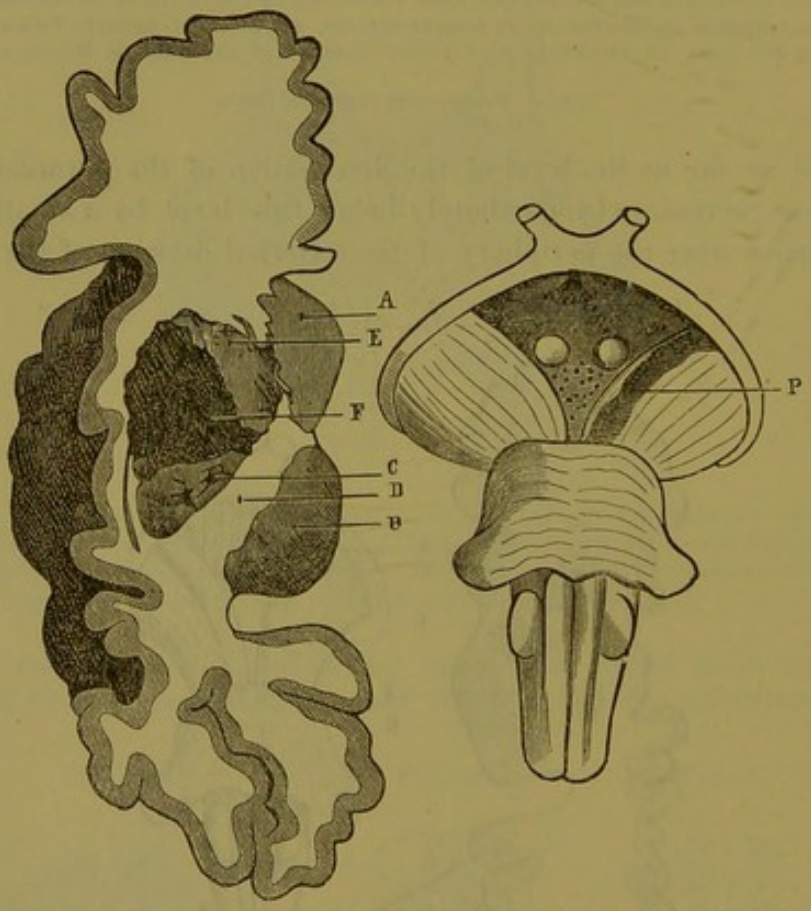
RECENT SOFTENING OF THE FRONTAL LOBE, THE ISLAND OF REIL, AND MIDDLE THIRD OF THE LENTICULAR NUCLEUS. (BRISSAUD)

D, Old focus of softening occupying the knee of the internal capsule; A, Caudate nucleus; B, Optic thalamus; C, Anterior, and E, Posterior division of the internal capsule.

root-zone, immediately behind the anterior nerve roots. Above the level of the lesion a secondary degeneration of the fillet could be traced as far as the posterior tubercle of the corpora quadrigemina.

A case has been described by Brissaud in which, along with extensive recent softening of one hemisphere, an old focus of softening was observed limited exactly to the knee of the internal capsule (Fig. 61, D). A streak of degeneration was observed lying between the internal and middle thirds of the crusta, being the anterior portion of the area which has already been described as the mixed area of medullated and non-medullated fibres in a nine months' embryo. According to Brissaud, degeneration occurs in the knee of the internal capsule in cases of long-standing aphasia.

FIG. 62.



A, Caudate nucleus; B, Optic thalamus; C, Posterior and healthy part of the lenticular nucleus; D, Posterior segment of the internal capsule; E, Lesion of the anterior segment of the capsule; F, Cyst presenting the form of the lenticular nucleus; P, Degeneration of the internal fibres of the crusta. (BRISSAUD.)

Another important case has been observed by Brissaud, in which an old focus of softening was found in the anterior half of the lenticular nucleus, destroying also the anterior segment of the internal capsule (Fig. 62, F). A streak of degeneration was observed in the internal third of the crusta (Fig. 62, P), but all the fibres of this area were not implicated in the degeneration, a small bundle of the innermost fibres

remaining normal. The degenerated fibres in this case corresponded very nearly to those which we have described as the accessory fibres of the tract. Degeneration of the internal tract of the crusta, according to Brissaud, appears to be always connected with intellectual disorders. It is important to remember that a few cases have been reported of destructive lesions localized in the motor area of the cerebral hemispheres, but in which no secondary degenerations were discovered in the internal capsule, crura, pons, medulla oblongata, or spinal cord.

FIG. 63.

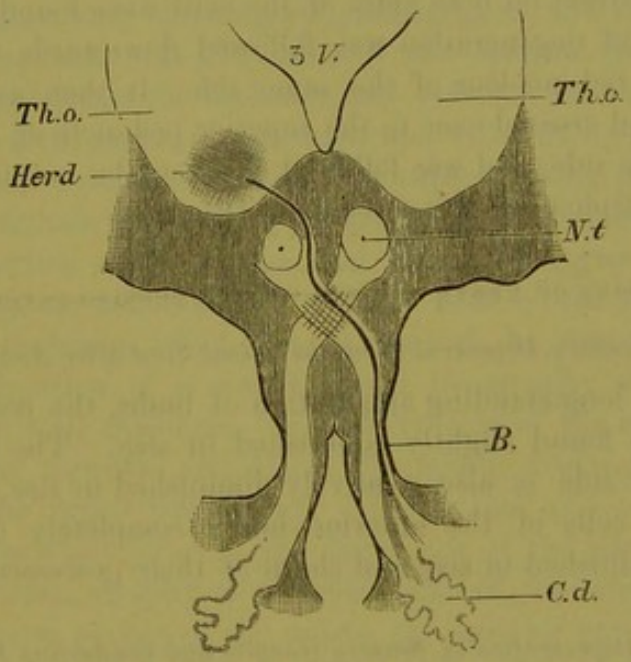


DIAGRAM OF THE OPTIC THALAMI, TEGMENTUM, AND SUPERIOR PEDUNCLES OF THE CEREBELLUM. (After MENDEL.)

3V., The third ventricle; Th.o., The optic thalamus; N.t, The red nucleus of the tegmentum; B., Superior peduncle of the cerebellum; C.d., Corpus dentatum of the cerebellum; Herd, The diseased focus. The dark line between the diseased focus and the corpus dentatum of the opposite side represents the streak of degeneration.

The following bundles of fibres may, therefore, be distinguished in the internal capsule:

(1) A *posterior or sensory fasciculus* (occupying the external third of the crusta), which is never the seat of secondary degeneration.

(2) A *middle fasciculus* (occupying the middle third of the crusta), which is the usual seat of secondary degeneration.

(3) A *geniculate fasciculus* (occupying the point of union of the middle and internal thirds of the crusta), which has erroneously been regarded as incapable of degeneration. This fasciculus contains fibres which are distributed to the bulbar centres, and are concerned in the production of the voluntary movements of the face and tongue.

(4) An *anterior fasciculus* (occupying the internal third of the crusta), degeneration of which appears only to be associated with intellectual disorders.

A descending degeneration of a few of the fibres of the superior peduncle of the cerebellum has been observed. In a case reported by Mendel, in which the patient had suffered, after a slight apoplectic attack, from right-sided hemiparesis and transitory right-sided hemianæsthesia, with left-sided hemiopia, a hemorrhagic focus about the size of a pea was found in the pulvinar of the left optic thalamus, close to the internal capsule. Slight evidences of descending degeneration of the pyramidal tract on both sides of the cord were found, and, in addition, a streak of degeneration was followed downwards to the internal aspect of the red nucleus of the same side; it then ascended in the tegmentum and crossed over to the superior peduncle of the cerebellum of the opposite side, and was followed as far as the corpus dentatum of the right hemisphere of the cerebellum (Fig. 63).

(c) DEGENERATIONS OF NERVE CENTRES AND CONDUCTING PATHS FROM DISEASE.

(j) *Secondary Degeneration of the Spinal Cord after Amputation.*

In cases of long-standing amputation of limbs, the posterior roots of the nerves are found slightly diminished in size. The posterior gray horn on that side is also relatively diminished in size, while some of the ganglion cells of the anterior horns completely disappear, and others are diminished in size and shorn of their processes.

(jj) *Degeneration of Sensory Ganglia and Conducting Paths.*

The facts so far known with regard to degeneration of sensory conducting paths in human pathology are very scanty. When unilateral blindness has existed for a long time the optic nerve on the blind side has been found atrophied, but the tract of the opposite side is affected to a greater degree than that of the same side. The external geniculate body and the anterior tubercle of the corpora quadrigemina on the side opposite the blind eye have also been found smaller than the corresponding parts on the same side. Similar phenomena have been found by Gudden after extirpation of one eyeball in young animals. The parieto-occipital convolutions of the left hemisphere of the brain were extirpated by Gudden in a young dog, and he afterwards found that the external geniculate body and the optic tract of the same side were atrophied. Experiments of this kind have recently been carried out systematically by Monakow, and he finds that extirpation of circumscribed portions of the cortex is followed by atrophy of strictly defined

parts of the optic thalamus and adjoining ganglia. The author distinguishes the following areas of the cortex: (1) *The zone of the corpus geniculatum externum*, so named because extirpation of it caused atrophy of this ganglion. This area is situated in the posterior superior part of the hemisphere; it forms about one-third of the hemisphere and corresponds to Munk's visual sphere. In addition to the external geniculate body, the posterior third of the internal capsule with the part of the corona radiata which lies between it and the injured cortex, the lateral layer of the optic thalamus, the stratum zonale in part, the optic tract, and in a less degree the tractus pedunculus transversalis, the anterior tubercle of the corpora quadrigemina, and the optic nerves were also atrophied. (2) *The zone of the internal geniculate body*, which is situated below the first area and corresponds to Munk's auditory sphere. Extirpation of this area was followed by atrophy of the posterior inferior portion of the internal capsule, along with the portion of the corona radiata which joins the atrophied part of the capsule with the injured portion of the cortex, and in a less degree the posterior part of the stratum reticulatum. (3) *The zone of the lateral nucleus* of the optic thalamus, which lies in front of the first zone or visual sphere. Extirpation of it was followed by atrophy of the external nucleus of the optic thalamus, the outer portion of the crusta, and to a less extent the formatio reticularis, the middle peduncle of the cerebellum, and the corpus trapezium. (4) *The zone of the stratum reticulatum* (Gitterschicht) lies to the outer side of the last zone. Extirpation of it causes atrophy of the third fifth of the internal capsule, a part of the lateral portion of the crusta, the anterior part of the stratum reticulatum, and to a less degree of the portion of the tegmentum which lies between the external geniculate body and the posterior tubercle of the corpora quadrigemina. (5) *The zone of the anterior tubercle* of the optic thalamus is situated in front of the third zone. Extirpation of it causes atrophy of the anterior tubercle of the optic thalamus, the anterior part of the internal capsule, a part of the pyramidal tract, and to a less degree of the laminæ medullaris of the anterior tubercle, and the bundle of Vicq d'Azyr. (6) *The zone of the internal nucleus* of the optic thalamus occupies the anterior area of the cerebral cortex. Extirpation of it causes atrophy of the most anterior portion of the internal capsule, and partial atrophy of the pyramidal tract and the internal nucleus of the optic thalamus. (7) External to the zone of the anterior tubercle lies another zone, extirpation of which causes atrophy of the anterior portion of the internal capsule, and of a tract which runs direct to the crus cerebri, which is supposed by Monakow to contain fibres of the facial and hypoglossal nerves.

II. GENERAL MORBID ANATOMY AND PHYSIOLOGY.

The problem of morbid physiology is to interpret morbid functions by morbid structures, and the law which underlies all our conclusions is that every disordered function is the correlative of a diseased structure; and, inversely, that every diseased structure has for its counterpart a derangement of function. It may be laid down as a general law that, when the irritability of a nervous structure is increased, it will manifest increased functional activity.

It may be supposed that a free arterial supply to a part, or a flushed condition of the arterioles, is the necessary correlative of increased irritability; and, conversely, that a diminished arterial supply, with an empty and contracted condition of the arterioles, is the necessary correlative of diminished irritability. This statement, however, can only be accepted as true within certain limits and with numerous qualifications. When the brain, for instance, is very freely supplied with blood so that its substance becomes congested, the irritability of the tissues is no doubt at first increased. It must, however, be remembered that the cranium is unyielding, and its contents practically incompressible, so that no additional quantity of blood can enter into the intracranial vessels except by displacing a corresponding quantity of some other fluid. When, therefore, the vessels become dilated beyond certain narrow limits the nervous tissue becomes compressed, the material exchanges within the cranium become less than when the circulation passes in normal quantity and under normal pressure, and the functional activity of the organ is diminished or abolished. A similar process no doubt occurs in the spinal cord and nerve trunks. Congestion in them, when carried beyond certain limits, is also attended with diminution of function, due, no doubt, to compression of the nerve tissues by the dilated vessels. The *irritative lesion* is attended with increased nutritive activity, and consequently with free arterial supply; but this lesion is exceedingly apt to terminate in the opposite condition of diminished nutrition and functional activity. The first stage of inflammation, for instance, is an *irritative* lesion, and it is attended by excess of functional activity, manifesting itself by symptoms of hyperæsthesia and hyperkinesis; but when the nervous tissues become partly compressed by effused products and partly disorganized by internal changes, the lesion becomes *depressive*, and the symptoms of excess give place to those of diminution of function; in other words, the symptoms of hyperæsthesia and hyperkinesis give place to those of anæsthesia and akinesis.

But if excess of nutrient activity is not always accompanied by increased functional activity, neither is diminished nutrient activity always accompanied by diminution of functional activity. When the nutrition of a nerve fibre is gradually lessened its irritability becomes, indeed, increased instead of diminished. The stock of irritable matter which the nerve fibre possesses is no doubt less under these circumstances, but an increased readiness to discharge the energy is manifested; and it is notorious that feeble and anæmic persons manifest an undue readiness to respond to the action of stimuli of all kinds, a condition which is correctly designated *nervous irritability*. One other important consideration must be taken into account before the amount of nourishment supplied to an organ or a tissue can be accepted as in any way a measure of the functional activity of the latter. When a strong faradic current is sent through the sciatic nerve of a frog, the gastrocnemius muscle contracts strongly; but a subsequent current passed through the nerve is followed by no reaction until the irritability of the nerve is restored by the absorption of more nourishment. A similar process doubtless occurs in disease of the nervous system. When a part is supplied with an excessive amount of nourishment, the tissues become so irritable that they discharge readily, either spontaneously, or in response to stimuli which would not affect them under normal conditions. Under these circumstances, excessive discharges of nervous energy readily take place, and these are followed by temporary loss of irritability, and the tissue becomes incapable for a time of performing its normal functions. The excessive liberations of energy from the cortex of the brain, which occasion epileptic attacks, for instance, are accompanied by loss of consciousness, which lasts for a considerable time, and the convulsive phenomena are not unfrequently followed by temporary motor paralysis. When the energy of the *discharging lesion* is once liberated, the part affected becomes incapable of performing its functions, until its irritability is restored by the absorption of a fresh stock of irritable matter. The primary effect of almost all chemical agents on the nervous system is to stimulate it, and to increase its functional activity, while their secondary effect is to depress or abolish its functional activity. The stimulant action of alcohol on the brain, for instance, is followed by a stage of depression, which may amount to complete abolition of the cerebral functions or coma. Strychnine increases the irritability of the gray substance of the spinal cord; but the reflex actions, which are at first greatly exaggerated, become ultimately abolished, and the animal poisoned by strychnine often dies from paralysis. Curara, which may be taken as the type of nervous sedatives, paralyzes the terminations of the motor

nerves, yet Bernard proved that it first increases the irritability of the fibres.

The direct tendency of all destroying lesions is to abolish function. It must, however, be remembered that these lesions are frequently surrounded by a zone of nervous tissue which is in a state of irritation, and the prominent symptoms of the affection are often produced by this zone, consequently the symptoms may be indicative of excess of functional activity. A gummatous tumor, for instance, in the cortex of the brain is generally declared by epileptoid convulsions; yet the direct tendency of the tumor, in so far as it has destroyed and replaced nervous tissue, is to abolish function. In such cases, both the direct and indirect effects of the tumor are often manifested: the former by paralytic and the latter by convulsive symptoms. Even the ischæmic softening, caused by plugging of vessels, is often surrounded by a congestive zone of tissue, and the latter may give rise to symptoms of irritation.

CHAPTER IV.

GENERAL SYMPTOMATOLOGY.

Disease of the nervous system gives rise to disorders (A) in the feelings of the patient—the *æsthesioneuroses*, (B) in the nutrition of the various tissues of the body—the *trophoneuroses*, and (C) in the movements of the body as a whole and of its various parts—the *kinesioneuroses*.

A. THE ÆSTHESIONEUROSES.

The æsthesioneuroses may be divided into disorders (I) of the primary or elementary feelings—the *primary* æsthesioneuroses, and (II) of the *secondary* or compound feelings—the *secondary* æsthesioneuroses, while each of these divisions may be subdivided into (1) disorders of the common subjective or emotional feelings (pleasures and pains), and (2) the special, objective, or intellectual sensations.

I. The Primary Æsthesioneuroses.

The general disorders of the feelings consist, *first*, of excess of the normal sensation caused by a stimulus, which is named *hyperæsthesia* when the objective feelings are increased in acuteness, and *hyperalgesia* when the common sensation of pain is increased; *secondly*, of diminution of the normal sensation evoked by a stimulus, which is named *anæsthesia* when the objective feelings are diminished, and *analgesia* when the subjective feelings are diminished in acuteness; *thirdly*, of paroxysms of pain in the region of distribution of a sensory nerve in the absence of external stimulus or active inflammation of the nerve, named *neuralgia*; *fourthly*, of abnormal sensations, such as numbness, tickling, crawling, itching, and feelings of heat and cold, also felt in the absence of external stimulus, and grouped together under the name of *paræsthesiæ*; and *fifthly*, a diminution or increase of the interval which elapses between the instant at which a stimulus is applied to a sensory surface and the moment at which the subject makes a voluntary effort to indicate that the sensation has been perceived, named acceleration or retardation of sensory perception respectively.

Disorders of the primary or elementary feelings may be divided

according to the tissues and organs implicated into sensory affections: 1, of the skin and exposed parts of the mucous membranes; 2, the voluntary muscles; 3, the bones and joints; 4, the viscera; 5, the special senses. The anatomical division is traversed at right angles by a physiological division into sensory disorders of (1) the common or subjective feelings, and (2) the special or objective feelings.

1. *Cutaneous Æsthesioneuroses.*

Sensory disorders are known through the statements and gestures of the patient, and the information obtained by this means must be checked by a systematic examination.

The following tests of the various forms of cutaneous sensory disorders may be used at the bedside:

(*a*) *Common cutaneous sensations* may be tested by the prick of a pin, application of heat and cold, pinching, and firm pressure, or the application of the faradic current.

(*b*) The *rapidity of sensory conduction* may be tested by getting the patient, with closed eyes, to give a signal by voice or by a tap on a table immediately on feeling the prick of a pin, and noting the interval which elapses between the prick and the signal.

(*c*) The *sense of pressure* may be tested by Weber's method, which consists of the superimposition of weights to determine the smallest difference which can be perceived. To exclude the muscular sense the part to be tested should be at rest on a table, and the sensation of temperature may be excluded by the interposition of a bad conductor like a wooden disk.

(*d*) The *sense of locality* may be tested by touching some part of the surface with the finger or point of a needle whilst the patient's eyes are closed, and asking him to indicate the point touched. A better plan is to ascertain to what distance the points of a compass must be separated from one another before they are felt as two. The distance to which the points of the compass must be separated varies for different regions of the body, so that the observer must know the normal scale of difference before drawing any conclusions from the results obtained, but in many cases valuable information may be obtained by comparing the anæsthetic area with the corresponding part on the opposite side, which may be normal.

(*e*) The *sense of temperature* is tested by applying hot and cold bodies to the surface when the patient's eyes are closed, and asking him to indicate each time whether the temperature of the touching body was hot or cold. Two test-tubes, one filled with cold and the

other with hot water, answer the purpose of testing very well, or two silver spoons, one dipped in hot and the other in cold water, are equally efficient and often more convenient.

(*f*) The sense of touch is a compound sensation, but it may be roughly tested by means of a light touch with a feather, and asking the patient to indicate with closed eyes each time he is touched.

a. Cutaneous Hyperæsthesiæ.

(1) *Hyperpselaphesia*, or abnormal acuteness of tactile sensibility, declares itself by excessive reaction to the various tests for the senses of pressure and locality. A smaller difference than usual in the increase of pressure is perceptible, and the diameters of the areas of sensibility are unusually small.

(2) *Polyæsthesia* is the condition in which one point of the compass on being placed on the skin is felt as two, three, or five points.

(3) *Allochiria* is a condition in which the patient is not sure, and is often in error when the eyes are closed, as to which side of the body is touched, even though the cutaneous sensibility is more or less normal in other respects.

(4) *Girdle sensations* consist of a subjective perception which produces the impression of having a girdle or a broad bandage tied about the trunk or limbs.

(5) *Thermo-hyperæsthesia* consists of an abnormal acuteness of the sense of temperature, so that differences in temperature so slight as to be inappreciable in health are recognized.

(6) *Causalgia* is a name given by Dr. S. Weir Mitchell to a distressing pain which appears to belong to the thermo-hyperæsthesiæ. It is described by patients as an intensely burning sensation, and is compared to that caused by a mustard plaster, or by "a red-hot file rasping the skin." It is generally associated with "glossy skin," but often precedes the trophic changes of the skin.

(7) *Dysæsthenia* is a term introduced by Charcot to indicate a sensation of a peculiarly distressing and vibratory character, which ascends towards the central end of a limb and descends towards its extremity. The sensation is excited by the slightest touch or the application of a cold body to the surface, and it persists from several minutes to a quarter of an hour after the exciting cause has ceased to act. After a short time an analogous sensation may be felt at a corresponding point of the limb, opposite to the one primarily excited.

(8) *Hyperæsthetic spots* consist of circumscribed patches of skin which are exquisitely painful to touch, and which are subject to attacks of spontaneous pains of a burning character.

(9) *Cutaneous hyperalgesia* consists of an increased sensibility of the common sensations, and is much more frequent than increase of tactile sensibility. In this condition, stimuli, which in health give rise to touch, or even to pleasant sensations like the minor degrees of tickling, now become painful. *Thermo-hyperalgesia* may be applied to designate the condition in which contact with a hot or cold body gives rise, not to a feeling of temperature, but to a painful sensation.

b. Cutaneous Anæsthesiæ.

(1) *Distribution of Anæsthesia.*—Anæsthesia or sensory paralysis is sometimes distributed in limited patches or in the area of distribution of particular nerves, and it is then termed *circumscribed anæsthesia.* At other times it appears in the form of a zone of variable width surrounding the body on one or both sides, and it is then named *anæsthesia in the form of a girdle.* When it is distributed over the lower half of the body and the lower extremities it is termed *paranæsthesia;* and when over the lateral half of the body, including half the face and the extremities, it is named *hemianæsthesia.*

(2) *Total anæsthesia* is a diminution or loss of every form of cutaneous sensibility, and *partial anæsthesia* a diminution or loss of certain forms of sensibility while others are preserved.

(3) *Apselaphesia* or *tactile anæsthesia* denotes a diminution or loss of the acuteness of tactile sensibility, a condition which is frequently associated with hyperalgesia. Severe pain is sometimes felt in anæsthetic parts, and the condition is then called *anæsthesia dolorosa.*

(4) *Thermo-anæsthesia* means insensibility to heat or cold, a condition which sometimes occurs as an isolated affection.

(5) *Analgesia* denotes a diminution or loss of the sensation of pain. Analgesia, with preservation of the tactile sensibility, is the most frequent form of partial sensory paralysis.

(6) *Retardation of sensory conduction* may be employed as a test of any form of anæsthesia, and gives rise in the partial varieties to the following anomalous phenomena:

(*a*) *Separation of Tactile and Painful Impressions.*—In locomotor ataxia the prick of a needle causes a prompt feeling of touch, which is often followed in two or three seconds by a feeling of pain. In cases of thermo-anæsthesia, a test-tube holding hot water may at first give rise to an immediate feeling of touch, to be followed in two or three seconds by a sensation of temperature.

(*b*) *Double Painful Sensations.*—In some cases of locomotor ataxia, pricking the skin on the back of the foot with a needle is sometimes

followed by a first painful sensation, and, when this subsides, by a second painful sensation, which is usually of greater intensity and more prolonged than the first. The first is felt after a lapse of two and a half to three seconds subsequent to the prick, but the second is not felt until after another interval of from two to five seconds.

(*c*) *Persistent After-sensations.*—In some cases of partial anæsthesia, pinching the skin or pricking with a needle gives rise to a sensation which begins slowly, but gradually increases in intensity, and is much more severe than the pain which occurs in health.

(*d*) *Inability to Count Successive Impressions.*—Closely connected with these persistent after-sensations is the inability of the patient to count correctly several impressions made in quick succession. Enumeration of successive impressions presupposes an interval to elapse between the sensation caused by each; but, when the conduction is retarded, each sensation is unusually prolonged so that the first does not fade before the second begins, and so counting becomes impossible.

c. Cutaneous Paræsthesiæ and Paralgesiæ.

(1) *Pruritus* is a sensation caused by abnormal irritation of the nerve-ends of the papillæ of the skin, or by a state of undue irritability of these nerve terminations themselves. Pruritus is related to such sensations as tickling and to burning and stinging pains, but an irresistible tendency to scratch is its characteristic symptom.

(2) *Formication* does not amount to pain, but is described as a feeling of creeping or pricking, or is compared to the crawling of ants. Formication occurs as a transitory symptom in minor mechanical injuries of nerve-trunks, and is felt in the foot as a sensation of "pins and needles," numbness, or "sleepy sensation," when the sciatic nerve is compressed for some time.

d. Neuralgia and Neuralgiform Pains.

(1) *Neuralgia* consists of periodic attacks of severe pain, occurring suddenly and spontaneously in the course of one of the larger nerve-trunks, and ramifying in a few or all of its sensory branches.

The character of the pain in cutaneous neuralgia will be described when the special forms of it are under consideration, but we shall here refer to some of the more prominent phenomena of the affection. During the height of the neuralgic paroxysm there is an *irradiation of the pain* to other sensory nerves, generally to branches of the same trunk or to neighboring nerves, but occasionally to more or less distant nerves, and the neuralgic pain is accompanied by various paræsthesiæ, hyperæsthesia, or sometimes anæsthesia.

Painful Points.—These points were first described by Valleix under the name of *points douloureux*. An examination of the part during an attack of superficial neuralgia will reveal one or more points which are extremely sensitive to the pressure of the tip of the finger. The sensitiveness of these points stands almost in a direct relation with the severity of the paroxysms, but they may be present occasionally during the period of remission, and in some cases pressure upon them induces an attack. These tender spots are found at various points in the course of the affected nerves, when their trunks pass from a deeper to a more superficial level, and especially where they emerge from bony canals or pierce fibrous fasciæ, or even where a nerve lies on a hard bed so that it may be easily compressed.

Point Apophysaire.—Trousseau believed that in all forms of neuralgia the spinous processes of the vertebræ corresponding to the origin of the painful nerve, and which he calls *points apophysaire* or *spinous points*, are painful on pressure, but these points are also present in spinal irritation and in myalgia. The concomitant symptoms of neuralgia will be described along with the special forms of neuralgia.

(2) *Lightning-like Pains.*—In locomotor ataxia patients often suffer from spontaneously occurring paroxysms of distressing pains, which are compared by them to forked lightning darting through the body, and have been described under the name of general neuralgia, or neuralgic rheumatism.

2. *Sensory Affections of the Voluntary Muscles (Muscular Æsthesioneuroses).*

a. Muscular Hyperæsthesia and Hyperalgesia.

The feeling of unrest and desire for constant change of position, called the "fidgets," appears to be due to *muscular hyperæsthesia*. In spasmodic wry-neck, and in "cramps" the hyperæsthetic condition is often very great, and causes intense pain, The excessive feeling of fatigue and prostration which occurs on slight exertion in the prodromal stage of acute diseases, is probably due to an increasing muscular sensibility, and consequently this condition may be regarded as a *muscular hyperalgesia*. Painful conditions of the muscles are called *myalgia* or *myodynia*. Pain is most frequently met with in the muscles of the neck and of the lumbar region, and inasmuch as it corresponds to cutaneous neuralgia and arthralgia, it may be called *muscular neuralgia* or *myoneuralgia*.

b. Muscular Anæsthesia.

Muscular anæsthesia consists of diminution or loss of the common sensibility and of the sense of muscular effort. When muscular sensibility is lost, as tested by the faradic current, while the muscular sense is retained, the condition is termed *muscular analgia*, or *muscular analgesia*. When there is a diminution or loss of the capacity of recognizing small weights, or of perceiving small differences by means of muscular effort with closed eyes, the condition is called *anæsthesia of the muscular sense*.

(1) *Tests of Muscular Sensibility.*—The state of muscular sensibility is best tested by the faradic current. When a healthy muscle is made to act, a dull feeling accompanies the contraction, this feeling being much increased in muscular hyperalgesia and diminished or lost in muscular analgesia.

(2) *Tests for Muscular Sense.*—To test the muscular sense the patient should be made to lift various weights, and to form an estimate, with closed eyes, of the weights and of the differences of successive weights. In order to eliminate the cutaneous sense of pressure, the weights should be placed in a cloth and suspended from the limb to be tested. The patient may also be made to move the limb into certain prescribed positions, with closed eyes; he may be asked to touch a particular part of the body, such as the tip of the nose, with the index finger, to take hold of a ticking watch held before him, or to describe an imaginary circle on the floor with the big toe. The patient fails to accomplish these actions with precision when there is *muscular anæsthesia*.

3. *Sensory Affections of the Joints and Bones (Articular and Osseous Æsthesioneuroses).*

a. Osteoneuralgia.

The bones are sometimes the seat of severe pain, and when the pain is not caused by recognizable anatomical changes the condition is regarded as a neuralgia, and named *osteoneuralgia*.

Neuralgia of the bones differs from cutaneous neuralgia in not radiating along the course of the principal branches of the affected nerves. It is probable that the sensory nerves of the bones reach them along with the sympathetic plexus which surrounds the vessels.

b. Arthroneuralgia.

Arthroneuralgia consists of severe neuralgiform pains in a joint in the absence of any recognizable anatomical alteration of it. The pain occurs in paroxysms which come on spontaneously, and which are separated by intervals of complete or comparative freedom from pain. The pain is sometimes described as tearing, or shooting through the joint like lightning, and at other times as a boring or stabbing pain.

The patient may also complain of numbness and formication about the joint, or of sensations of heat and cold, and the skin over the joint is often hyperæsthetic in the early stages of the affection, and anæsthetic when it is of long standing. The pain is much increased when the patient's attention is directed to it, while it is diminished under the influence of general fatigue, and does not prevent the patient from sleeping. Pressure increases the pain, but, as occurs in cutaneous neuralgia, slight and superficial pressure may produce intense pain, while deep, continuous, and uniform compression produces no effect, or even relieves the pain. Painful points may be obtained on pressure, but their position is somewhat indefinite. Neighboring nerve-trunks may also be found painful on pressure, and the spines of some of the vertebra may likewise be tender to pressure. In some cases spastic contraction of the muscles surrounding the joint occurs, while in other cases the limb is feeble and helpless, owing, perhaps, to the fear of inducing a paroxysm of pain by movement at the joint rather than to any muscular paralysis.

The vaso-motor disturbances consist of redness, heat, and increased secretion of sweat in the neighborhood of the affected joint, and a circumscribed dough or fluctuating swelling is sometimes observed in the skin over the joint, which was compared by Brodie to an unusually large urticaria wheal. Swelling of the joint from serous effusion within the capsule may occasionally take place, and is apt to be regarded as of inflammatory origin, and when effusion takes place in the tissues surrounding the joint as the result of irritating applications, the diagnosis is still further obscured.

4. *Sensory Affections of the Internal Organs (Visceral Æsthesioneuroses).*

The visceral sensations belong to the common or subjective sensations, and consequently excess of a normal visceral feeling may be regarded as a *hyperalgesia*, while an altogether abnormal visceral feeling belongs to the *paræsthesiæ;* but it is by no means easy to draw a distinction between these two kinds. Diminution or loss of the visceral

feelings constitutes a visceral *anæsthesia*, or, more correctly, a visceral *analgesia*.

a. Visceral Hyperalgesiæ and Paralgesiæ.

(1) *Titillation* is a sensation induced by irritation of the sensory branches of the vagus, especially the superior laryngeal branch, or by undue irritability of the ends of the nerve, and corresponds with pruritus of the external skin; and as pruritus leads to an irresistible tendency to scratching, so titillation leads to the reflex respiratory movements which produce coughing.

(2) *Globus* is a sensation which gives the feeling of a ball ascending from the epigastric region to the throat. It is a frequent symptom of hysteria, and occasionally forms the aura of an epileptic attack. Globus is supposed by some to be caused by spasm of the œsophagus and pharynx, and it has consequently been called *œsophagismus*, but the explanation is not a satisfactory one.

(3) *Pyrosis or water-brash* is a painful sensation in the epigastrium, consisting of a sense of burning, which is generally attended with the rising of a quantity of clear watery and strongly alkaline fluid into the mouth. An attack of pyrosis may last from a few minutes to many hours, with alternating remissions and exacerbations, and is most probably caused by a spasm of the cardiac end of the stomach when its contents are very acid, thus arresting the secretion of saliva in the œsophagus.

(4) *Bulimia* is a feeling of hunger, which is abnormal in its period of occurrence or in its intensity, and which is appeased only for a short time by taking food.

(5) *Polydipsia* is an excessive feeling of thirst, and is a constant symptom of polyuria and diabetes, and is an occasional symptom in hysteria.

(6) *Excessive voluptuous feelings* arise without any exciting cause in both sexes, and are associated with the erections and ejaculations which accompany normal coitus. These sensations may be caused by a local irritation of the genitals, or by a central disease like tabes dorsalis.

(7) *Feeling of oppression* arises, when general, from overwork or deficient nourishment of the nervous system. It is described by the patient as a feeling of heaviness, dulness, and depression of spirits. A more specific form of oppression occurs in connection with cardiac affections, which will be subsequently described as angina pectoris.

b. Visceral Anæsthesiæ and Analgesiæ.

Very little is known with regard to diminution or loss of visceral feeling. The normal functions of the viscera are performed without

definite sensibility, although a diffused visceral sensibility contributes greatly to our feeling of comfort and well-being, and it is probable that diminution of visceral sensations contributes in a corresponding degree to our general feeling of bodily discomfort. Visceral anæsthesia is best defined in the organs which are most in relation with external forces, such as the larynx, stomach, sexual organs, and rectum.

(1) *Anæsthesia of the laryngeal and bronchial branches of the vagus* renders titillation and the consequent reflex act of coughing impossible. In this condition catarrhal secretions fail to be expelled, and may by their accumulation cause suffocation.

(2) *Anæsthesia in the territory of the gastric branches of the vagus* gives rise to *polyphagia*, a condition in which an unusual quantity of food must be taken before the feeling of hunger is appeased, or in which the feeling of repletion is never obtained however much food is taken. The experiments of Brachet, Arnold, and others have proved that, on section of the vagi, animals continue to eat until the œsophagus is filled with food.

(3) *Anæsthesia of the sexual feelings* is most frequently observed in the female sex. Complete absence of voluptuous feelings in hysterical females, along with diffused or circumscribed cutaneous anæsthesia, is most probably due to anæsthesia of the mucous membrane of the vagina. Anæsthesia of the genitals is observed in the male sex as a result of sexual excesses or onanism, or as a symptom of chronic affections of the spinal cord, such as spinal meningitis and tabes dorsalis, or in the absence of any appreciable cause. In such cases the electrical sensibility of the glans penis and of the external genitals is diminished, and the power of erection is lost, constituting *impotency*; or the loss of reflex irritability arrests the secretion of semen and abolishes the power of ejaculation, a condition named *aspermatism*.

(4) *Anæsthesia* of the mucous membrane of the rectum, which is a frequent symptom of grave organic diseases of the spinal cord, permits the stools to pass unconsciously.

(5) *Anæsthesia of the mucous membrane* is probably present in the early stages of many cases of locomotor ataxia, and causes great tolerance of the bladder to its contents. The bladder may become greatly distended without giving rise to the desire to micturate. Anæsthesia of the mucous membrane of the urethra produces a condition in which the urine may pass in a full stream without the knowledge of the patient.

5. *Sensory Disorders of the Special Senses.*

The consideration of this part of the æsthesioneuroses is reserved for special mention in a later part of this work.

II. The Secondary or Compound Æsthesioneuroses.

The sensory conducting paths unite with each other for the first time on reaching the common centre of sensory connections—the *sensorium commune;* and molecular change in this centre is the correlative of the compound feelings and cognitions, while an abnormal molecular discharge from this centre is the correlative of the disorders of the compound feelings comprised under the name of the secondary æsthesioneuroses. But we have seen that the cortex of the brain forms also a common centre of motor connections, a *motorium commune*, and that there is no clear dividing line between the common sensory and common motor centre; and it may, therefore, be expected that the disorders of the compound feelings will not be separated by a sharp line of demarcation from the disorders of the compound movements of the body. As a matter of fact, the disorders of the elementary feelings, of the compound feelings, of the compound movements, and of the elementary movements merge into one another by insensible gradations, and it is impossible to draw anything like a clear-cut division between them. We shall, therefore, describe briefly in this section a few of the disorders of the compound feelings and movements under the name of psychical disorders, and without attempting to differentiate clearly between the two sets of phenomena:

(1) *Pseudo-æsthesia* is a generic name given to sensations and perceptions experienced in the absence of any adequate irritation of the peripheral end-organs or of the conducting paths, the condition of their production being a molecular discharge from the common sensory centre. When a sensation of this kind is experienced, and gives rise to an erroneous perception of external objects and relations, it is named an *illusion;* when it gives rise to an objective sensation in the entire absence of an external cause, it is termed a *hallucination;* and when the patient forms such a distorted conception of the properties and relations of things actually existing that he is led to a false conclusion with regard to them, the condition is called a *delusion.*

(2) *Unconsciousness.* — The molecular activity of the sensorium commune is intermittent, and under normal conditions it is suspended for several hours every night, and during this time consciousness is abolished. This constitutes *sleep.* If the subject cannot be aroused to consciousness by the application of ordinary stimuli, the condition is called *somnolence* or *stupor;* and if the unconsciousness become so profound that the subject cannot be aroused by the strongest external stimuli, the condition is called *coma.*

(3) *Subconscious and Semiconscious Psychical Actions.*—Between the active consciousness of a healthy person after being restored by sleep and complete insensibility there are all degrees of diminution of sensibility. It is well known that a person whose attention is strongly directed to a particular subject is insensible to ordinary stimuli. A more advanced degree of diminution of the activity of consciousness is manifested by persons suffering from great fatigue and loss of sleep. Under such circumstances a person may walk about in a half-conscious state, and a similar *dazed* condition is frequently observed after epileptic seizures. The patient may perform complicated motor actions while in a totally unconscious condition, as is seen in somnambulism, the mesmeric state, or the narcosis caused by chloroform and similar agents.

Unconscious conditions are, indeed, frequently associated with motor disturbances. When the inhibitory action of the highest coördinating centre is removed, the functional activity of the lower centres may be increased. Many atrocious murders are committed during the period of semiconsciousness which sometimes follows an epileptic seizure, and it is well known how fierce and brutal many men become during the semiconscious condition induced by alcoholic excess. In *delirium*, again, the highest form of consciousness is in abeyance, while the lower, earlier organized forms are abnormally active. The patient, for instance, is unable to sleep, and yet he is only partially conscious of surrounding objects and events; he is subject to illusions, hallucinations, and delusions, and motor disturbances are manifested by great restlessness and incoherent speech. It is probable that in delirium the stock of irritable matter in the gray substance of the cortex is much exhausted, and that what remains manifests an undue degree of irritability, so that the protoplasm gives out energy either spontaneously or on the application of slight stimuli, while functionally there is a dissolution from the later to the earlier acquired feelings and experiences.

But although the patient is only semiconscious in delirium, yet his mental experiences during that time may be subsequently remembered with painful intensity. This also occurs in dreams. The patient is wholly unconscious of external impressions at the time, but he is partially conscious of a succession of feelings and images, either of a joyful or a painful kind, which may be subsequently revived in memory with greater vividness than almost any of his mental experiences during waking hours. Dreams are frequently accompanied by motor disturbances, such as vocalization and articulate sounds. The most prominent feature of the night-terrors of children is the outward expression of extreme terror by which the attack is manifested. The partial unconsciousness which precedes or follows an epileptic seizure is often asso-

ciated with the outward manifestations of one of the emotions, and sometimes by a corresponding inward feeling. The aura of an epileptic attack may be a guilty expression, and the patient may subsequently be able to remember that immediately before the attack he experienced a feeling as if he had been guilty of an infamous action. What appears to be an increase of consciousness often results from a diminished activity of the higher sentient centres. Increase of the normal desires and appetites sometimes results from a peripheral irritation, but a person is liable to an illegitimate indulgence of the passions when the moral feelings are weakened, and temptation is apt to be strongest during states of mental enfeeblement from cerebral exhaustion. It is then also that remorse for previous indulgence is liable to become quite disproportionate in its intensity to the degree of guilt, and may be experienced in the absence of any guilt to atone for. The depressing emotions, such as fear and anger, are also liable to become excessive during states of nervous exhaustion, and it is a matter of common observation that a person who is in feeble health is often very irascible, while others are easily excited to laughter or tears. Experiments on animals have shown that a nerve whose nutrition is lowered discharges its energy more readily than one whose nutrition is perfect, and similarly when the nutrition of the sensorium commune is defective it responds to stimuli of less intensity than when its nutrition is normal.

(4) *Double Consciousness; Periodic Amnesia.*—Closely allied to somnambulism and the mesmeric sleep is the curious condition which has been called double consciousness, or periodic amnesia. In this condition the subject is liable to periodic seizures, which may last from some hours to as many days, and during which there is complete forgetfulness of the feelings and events of ordinary existence, although rational thought and action still remain.

(5) *Abnormal States of Consciousness.*—There are some abnormal elementary feelings which must be regarded as qualitative rather than quantitative alterations of consciousness. As examples of these, may be mentioned (*a*) headache, (*b*) vertigo, and (*c*) fainting. To the consideration of these may be added a few remarks on (*d*) abnormal appetites and emotions.

(*a*) *Headache.*—Although headache is an excess of painful feeling, yet it cannot as a rule be regarded as an excess of any normal feeling, or of any feeling which can be excited by the application of external stimuli. From this statement neuralgic, rheumatic, and probably some other forms of headache must be excepted, but what is generally known as a nervous headache is a truly abnormal feeling, and must be experienced by a person before he can form an adequate idea of it.

Such headaches are caused by changes of circulation in the brain, the circulation of poisons in the blood, or they may arise spontaneously at recurring intervals. It is probable that in all of them there is an alteration of the irritability of the cells and fibres of the sensorium. Recurring headaches are generally associated with vaso-motor phenomena in the regions of distribution of the cervical sympathetic nerves, but it is probable that these are the results and not the cause of the sensorial disturbance. Such headaches are regarded by Dr. Hughlings Jackson as a sensory epilepsy and as being dependent upon a discharge from the sensory portion of the cortex of the brain.

(*b*) *Vertigo* is a sensation of swimming in the head, during which surrounding objects appear to oscillate before the eyes, or rotate in a definite direction, and which is also accompanied by a sense of staggering or of rotation of the body. Vertigo appears to be the subjective correlation of want of coördination between the various muscular contractions necessary for adjusting the body to the different objects which surround it in space. It is a prominent symptom of those diseases in which the automatic mechanism for maintaining the erect posture is deranged, such as affections of the cerebellum and Ménière's disease. The position of the body in space is largely determined by the association of objects seen with the appreciation of the position of the eyes and head. Displacement of the position of the eyes, such as occurs in paralysis of one or more of the recti muscles, or of the position of the head, such as occurs in rotation of the head with conjugate deviation of the eyes, and in the compulsory movements to be subsequently described, is also accompanied by severe vertigo. This symptom frequently attends visceral disease, as dyspepsia, and it is then probably caused by vaso-motor changes influencing the cerebral circulation. This opinion is rendered all the more probable from the fact that vertigo is a troublesome symptom both of anæmia and congestion of the brain. Vertigo is usually accompanied by a motor phenomena in the region of distribution of the pneumogastric, such as feeble and irregular pulse, irregular respiration, and vomiting.

(*c*) *Fainting* is a deadly feeling caused by sudden anæmia of the brain occasioned by severe loss of blood or cardiac failure. It also is attended by gasping respiration, and frequently accompanied by vomiting.

(*d*) *Abnormal Appetites, Emotions, and Impulses.*—Drunkenness may be regarded as an abnormal appetite, especially when it assumes the aggravated form of *dipsomania;* but the most remarkable example of an abnormal appetite is afforded by the condition described by Westphal and others as *perverted sexual instinct* (conträre sexualemp-

findung). This condition is defined by Westphal as "a congenital perversion of the sexual instinct with retained consciousness of the morbid nature of the condition," and the recorded cases show that some persons are attracted in their sexual desires exclusively by individuals of their own sex. The most remarkable of the abnormal emotions consist of a morbid dread experienced in the absence of any circumstance or event which could be thought in the remotest degree capable of inducing such a feeling. Some persons experience an unconquerable feeling of dread when they are alone in an open space (*angoraphobia*); others have the same feeling when in a narrow lane between two walls (*claustrophobia*). Some people have a morbid dread of society (*anthrophobia*), while there are some men who only experience an aversion to the society of women (*gynephobia*). Some persons are totally unable to sign their names in the presence of a witness. A gentleman in business once told me that while able to keep accounts and attend to his business as well as ever, he found himself totally unable to sign a check if his clerk made a sudden demand for one. Suicidal, homicidal, and other morbid impulses are liable to become uncontrollable during periods of great nervous exhaustion, and when the highest manifestations of consciousness are in abeyance. Atrocious crimes are usually committed by persons during the period of depression which follows a prolonged carouse, or when the individual is in a state of semi-stupefaction, either from alcohol or as a sequel to an epileptic seizure.

CHAPTER V.

GENERAL SYMPTOMATOLOGY (*continued*).

B. TROPHONEUROSES.

THE degenerations of the nervous system having already been considered, we shall now proceed to describe the degeneration of muscles which result from nervous disease.

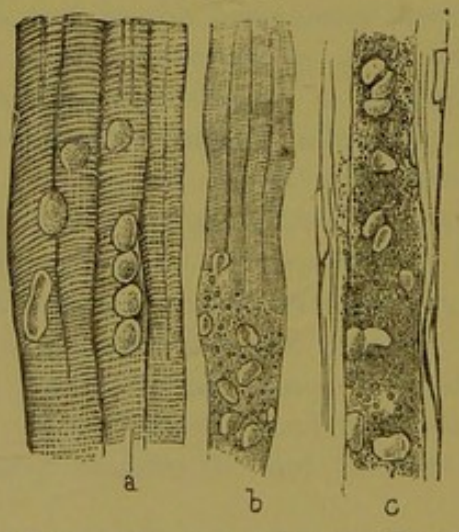
I. NEUROTIC ATROPHY OF MUSCLE.

Neurotic atrophy of muscle may be divided into three stages, namely: 1, Simple atrophy; 1, Atrophy with nuclear proliferation; 3, Cirrhosis of muscle.

1. *Simple Atrophy.*

In simple atrophy and the early stage of the severer forms of atrophy, the muscular fibres undergo a simple diminution in size,

FIG. 64.



ATROPHY OF MUSCULAR FIBRES FROM A CASE OF INFANTILE PARALYSIS. (After HAYEM.)
a, Fibres of normal size, showing multiplication of nuclei; *b*, simple atrophy, with granular degeneration; *c*, advanced granular degeneration, with atrophy.

without presenting any degenerative changes. The longitudinal and transverse striations are at times as well preserved as in health (Fig.

64, *a*), and not a trace of fatty degeneration can be discovered. It would appear that the fibrillæ of which the fibre consists are diminished in number, but those which remain do not seem to be sensibly diminished in size. At other times the striation becomes less marked and more delicate than in health, probably owing to a diminution in the length of the sarcous elements of the contractile disks (Fig. 64, *b*). The substance of the contractile disks may also present a finely granular aspect, which appears to be the first indication of the profound chemical change which this substance subsequently undergoes.

2. *Atrophy with Nuclear Proliferation.*

When the muscle is examined from three to five weeks after the injury the contents of the fibre are now seen to have undergone a finely granular degeneration (Fig. 64, *c*). The granules at first consist of altered protein, but they soon become distinctly fatty. The primitive fibrils now disappear, and only small fragments of the fibre present, here and there, either transverse or longitudinal striation. In addition to these changes the nuclei or muscle corpuscles are multiplied, and in the later stages of atrophy the sarcolemma may become almost filled with masses of nuclei which are surrounded by granular and fatty detritus, while the contents of the fibres are completely disintegrated (Fig. 65, *b b*). The nuclei of the endomysium also seem to be more numerous than in health.

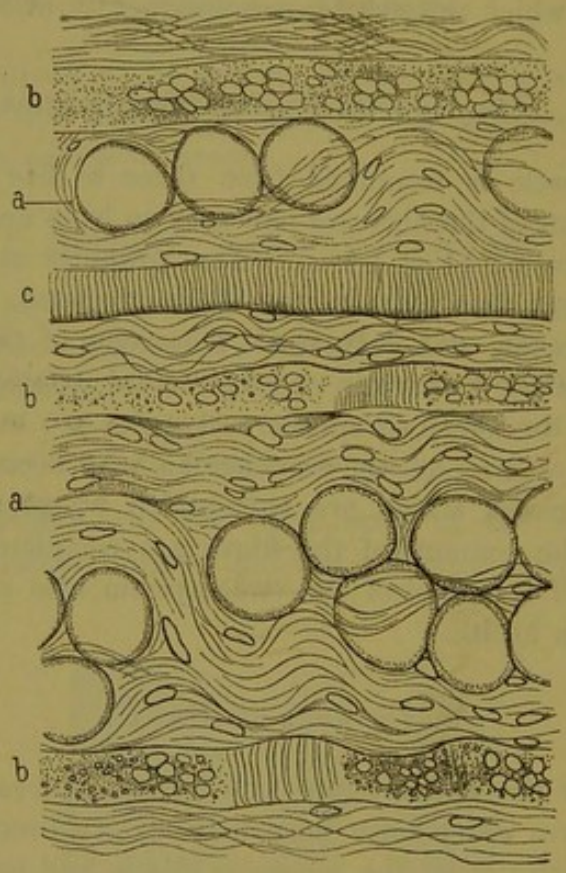
3. *Cirrhosis of Muscle.*

The nuclei of the endomysium, now greatly increased in number, elongate into fibres which form narrow bands of fibrous tissue running parallel to the direction of the muscular fibres, and cicatricial contraction of this tissue completely destroys the remnants of muscular fibre and gives rise to organic shortening of the band of fibrous tissue which now represents the muscle. These bands of fibrous tissue contain many oat-shaped nuclei and connective-tissue cells (Fig. 65, *a a*) which may become distended with fat, and these fat cells may become so abundant that the original volume of the muscle may be maintained or even exceeded.

It is now fully ascertained that active neurotic atrophy of muscle never occurs except when the spino-neural mechanism is injured, either by disease of the ganglion cells of the anterior horns of the cord and the corresponding cells in the medulla oblongata and pons, or of the efferent fibres which connect these with the muscles. A moderate

degree of muscular atrophy may also be caused by irritation of the afferent fibre of the reflex arc. Whether the same cell exercises both trophic and motor functions, or there exist separate cells for each function, is as yet undetermined. It would appear that a destructive lesion of the ganglion cells, or of the efferent fibres which connect these with the muscle, causes both motor paralysis and active atrophy, while in

FIG. 65.



INFANTILE PARALYSIS. (After HAYEM.)

a a, Excess of connective tissue, containing a large number of connective-tissue and fat cells; *b b*, atrophied muscular fibres, containing a large number of nuclei; *c*, simple atrophy of muscular fibre

severe cases there is also rapid loss of the faradic contractility. A certain degree of wasting may occur in paralyzed muscles when the lesion is restricted to the cerebral mechanism, but this form of atrophy arises simply from disease of the muscle, and differs entirely from the active atrophy which has just been described.

II. Cutaneous Trophic Affections.

1. *Cutaneous Eruptions in Lesions of Peripheral Nerves.*

Erythematous patches are often observed on the extremities after traumatic lesions of the peripheral nerves, on the root of the nose and forehead in trigeminal neuralgia, and on the hand in cases of brachial neuralgia. Erythema often arises in the course of digestive disorders, and it is then most probably of reflex origin. The urticaria which is caused by the stings of insects and nettles, and that which arises in the neighborhood of the puncture in subcutaneous injections, seems to indicate that this eruption is often, if not always, of nervous origin. Vesicular eruptions are often observed after injury to nerves. Eczematous eruptions are often associated with neuralgic pains in the area of distribution of an injured nerve, and eczema of the whole side of the face has been observed to accompany a severe attack of trigeminal neuralgia. Herpes zoster often accompanies a severe attack of neuralgia. Its favorite seat is the skin covering one or more of the intercostal spaces, and the neuralgia which accompanies it generally begins and terminates with the eruption; but, in aged people, the pain, which is very intractable, continues long after the eruption has disappeared. Herpes zoster of the face occurs in trigeminal neuralgia. It may appear on any part of the face, but is most frequent on the forehead. When the palpebral nerves are affected the eruption spreads over the upper eyelid, and the conjunctiva is inflamed; and when the frontal and its nasal branches are likewise involved, the iris and other structures within the eyeball are inflamed, and the disease may then cause serious damage to the eye. Herpes zoster has also been observed in the area of distribution of various spinal sensory nerves when the nerve is compressed by aneurism, cancer of the vertebra, or other tumors. Evidences of neuritis of the affected intercostal nerves have been found in cases that died whilst the patients were suffering from intercostal herpes zoster. Pemphigus bullæ sometimes develop with great rapidity over various parts of the surface supplied by the cutaneous branches of an injured nerve. After section or injury of nerves, the skin frequently becomes dry, harsh, and scaly; and a case is reported by Eulenburg in which several branches of the brachial plexus were injured and compressed in consequence of a dislocation of the humerus, and in which ichthyosis of the skin of the affected extremity had supervened.

After traumatic injuries of nerve-trunks the skin often undergoes atrophy, loses its wrinkles, and becomes smooth and glossy, and conse-

quently this condition has been named "glossy skin" by Paget. "In well-marked cases," this author says, "the fingers which are affected (for this appearance may be confined to one or two of them) are usually tapering, smooth, hairless, almost void of wrinkles, glossy, pink or ruddy, or blotched as with permanent chilblains. They are commonly also very painful, especially on motion, and pain often extends from them up the arm." Glossy skin is often met with in injuries of the ulnar nerve of the brachial plexus and in the upper extremity; the palm of the hand is the part which usually suffers, while in injuries of the nerves of the lower extremity the dorsum of the foot appears to be the part most liable to be attacked. Patches of leucoderma, with anæsthesia of the affected skin, have been observed on the face in trigeminal neuralgia, and over other parts of the body after injuries to large nerve trunks. Patches of highly pigmented skin, with anæsthesia, have also been found scattered irregularly, but symmetrically, all over the body; these also being doubtless of nervous origin.

The early occurrence and the severe degree of anæsthesia in the tubercular variety of leprosy, as well as the manner in which it progresses from the periphery towards the central parts of the affected limbs, would alone indicate that disease of some part of the nervous system is a prominent part of the affection, and characteristic anatomical changes have been found in the nerve-trunks. Morbid changes have also been discovered in the spinal cord and brain, but it is probable that disease of the nerve-trunks takes the most active part in causing the local symptoms.

2. *Cutaneous Eruptions in Diseases of the Spinal Cord.*

Cutaneous eruptions of various kinds occur in cases of chronic myelitis, but they are most frequently met with during attacks of lightning pains in tabes dorsalis. These eruptions consist of patches of urticaria, vesicular eruptions of which herpes is the most common, and papular eruptions like lichen planus, and they are always limited to nerve territories affected with neuralgic and lightning pains. Several cases of pemphigus in connection with spinal disease have been recorded by Chovstek and others. A case is recorded by Balmer in which an attack of pemphigus occurred in the course of progressive muscular atrophy.

3. *Cutaneous Eruptions in Diseases of the Brain.*

Herpes is not infrequently associated with hemiplegia, but it is doubtful whether there is anything more than an accidental connection between them. Two cases are reported by Dr. Duncan, in each of

which an eruption of herpes appeared on the affected side simultaneously with the motor paralysis. Several cases are reported in which a pemphigus eruption appeared on one of the paralyzed extremities in hemiplegia of central origin.

III. Trophic Disorders of the Nails and Hair.

1. *Trophic Disorders of the Nails and Hair in Lesions of the Nerve-trunks.*

Traumatic lesions of nerve-trunks in which the nerve is not completely divided, are followed by various deformities of the nerves. The nails become greatly curved—both laterally and longitudinally—furrowed, dry, and cracked at their extremities, and of a yellowish-brown color. These deformities may also occur in connection with neuralgia and idiopathic neuritis of sensory nerve-trunks. Local affections of the hair often occur in man after traumatic lesion of nerve-trunks, or in connection with idiopathic neuritis and neuralgia. The hairs over the region of distribution of a nerve affected with neuralgia have sometimes been observed to be hypertrophied and even increased in number; but, as a rule, the effect of neuralgia upon the hair is to make it brittle, and to cause it to fall out in considerable quantities. Localized grayness of the hair is often associated with ophthalmic neuralgia, and it may also involve the eyebrow of the affected side. This grayness sometimes assumes an intermittent character, increasing during and sometimes after an acute attack of pain, the color becoming partially restored in the interval between the paroxysms. In glossy skin the surface becomes hairless.

2. *Trophic Disorders of the Nails and Hair in Spinal Disease.*

The nails may become deformed in cases of acute and chronic myelitis, but the most remarkable change to which they are liable in spinal diseases occurs in locomotor ataxia. In the course of this disease the nails of the great toes sometimes fall off spontaneously, and are then rapidly replaced by a new and perfectly normal nail, which may in its turn fall off a few months later, and this process may occur in the same nail several times in succession. The falling off of the nail may be preceded for some weeks by a dull pain, or by a feeling of uneasiness in the toe, and then the nail falls off without being accompanied either by suppuration or by apparent ulceration of the matrix. In other cases the patient observes that the nail of one great toe, and a few days later that of the other, suddenly become of a dark blue color

from subungual effusion of blood, and a few days afterwards the nail falls off, without being preceded or accompanied by pain or other warning. The nails have also been known to fall off in sclerosis in patches. An increased growth of hair has occasionally been observed in cases of chronic myelitis.

3. *Trophic Disorders of the Nails and Hair in Cerebral Diseases.*

In cases of hemiplegia the nails become curved and cracked, but the trophic disorders to which they and the hair are liable in cerebral disease are by no means so well marked as in spinal affections and lesions of nerve-trunks.

IV. Bedsores and other Destructive Processes.

1. *Decubitus Acutus.*—Some days or even hours after the occurrence of a severe spinal or cerebral lesion, or after a sudden exacerbation of these affections, one or several erythematous patches appear over the sacrum and gluteal regions, the trochanters of the femur, ankles, or other parts subjected to pressure. In these patches, which are of variable extent and irregular form, the skin assumes a rosy hue, or becomes dark red or violet, but the color disappears momentarily on pressure with the finger. After a period which varies from twenty-four to forty-eight hours, the central part of the erythematous patch is covered with vesicles or bullæ, the contents of which, at first colorless and transparent, become more or less opaque, reddish, or brown colored. Under favorable circumstances the vesicle may wither, dry up, and disappear, and the part recover without further change; but in most cases the vesicles burst, and leave ill-looking ulcers, the bases of which are composed of the true skin in a state of phlegmonous inflammation, and infiltrated with blood. The base of the ulcer soon perishes by gangrene, the neighboring skin becomes inflamed to a greater and greater extent, and the gangrenous destruction extends deeper and deeper, laying bare and including in its destructive operation muscles, tendons, fasciæ, ligaments, and even the subjacent bones. One of the most remarkable characteristics of the affection is the extreme rapidity of its development, the entire cycle of changes being completed in a few days. Cystitis and hæmaturia are not infrequent complications of this condition, and the muscles of the lower extremities become the subjects of rapid atrophy. Metastatic abscesses now occur in the kidney, lungs, and other viscera, the accompanying fever assumes a remittent type, and the patient dies of septic fever. In some cases the gangrenous process extends to the sacral bones, and with the destruction of the

sacro-coccygeal ligament the vertebral canal is opened, and thus the ichorous discharges find access to the fatty cellular tissue which surrounds the dura mater, and by penetrating this membrane they may even make their way to the arachnoid cavity. This grave accident is followed by a *simple purulent* or an *ichorous* ascending meningitis, which rapidly reaches the base of the brain, and is soon fatal.

2. *Chronic Decubitus.*—In chronic diseases of the spinal cord the portions of skin subjected to pressure in the recumbent posture assume a dark red color, and at times become covered with superficial ulcerations. After a time a black spot appears on the reddened portion of skin, and, if pressure is continued, it enlarges rapidly, and the affected skin dries up into a hard leathery mass. In a short time a boundary line of inflammation forms around the gangrenous portion of skin, and the latter may, under proper treatment, be thrown off, leaving a more or less healthy granulating surface, which may sometimes cicatrize. But if the pressure be continued, or if the primary disease of the nervous system undergo a fresh exacerbation, the ulcerated surface assumes a dark violet color, the gangrene spreads rapidly, and all the destructive changes characteristic of acute bedsore make their appearance and soon lead to a fatal result.

3. *Symmetrical Gangrene and Local Asphyxia.*—This disease, which was first described by Raynaud, generally involves the fingers, and less frequently the toes, tips of the nose, and external ear. The affected parts become suddenly white, cold, bloodless, and insensible, and motor power is diminished. The skin is wrinkled and shrunken, the ends of the fingers appear thin and conical, and, when the whole extremity is affected, the pulse is feeble or imperceptible. After some months reaction sets in: the parts then become congested, of a violet or livid color, intensely painful, and the seat of troublesome itching, while vesicles form which are filled with a sero-purulent fluid, and, on bursting, leave the cutis excoriated. Even at this stage recovery may take place, but the attack usually recurs, and ultimately the parts undergo a true mummification and the last phalanges of the fingers drop off. The disease occurs usually in chlorotic and nervous persons, and is seldom met with in children and old people. It appears to be caused by a spastic ischæmia of the arterioles.

4. *Perforating Ulcer of the Foot.*—This affection, as seen in the foot, is less like an ulcer than a sinus. It usually presents itself as a small aperture, which leads directly by a narrow channel to exposed and diseased bone. From this opening there is little or no discharge; the skin surrounding the orifice is greatly thickened by superimposed layers of epidermis, and, indeed, the formation of a large corn appears

always to precede the destructive process. The ulcer is, as a rule, insensible to ordinary stimuli, and there is no pain when the patient is at rest; but considerable pain may be caused by pressure on the sole during locomotion, and the patient often suffers from severe lightning pains in the lower extremities. Not only are the tissues surrounding the wound insensible, but there is also, as a rule, more or less complete cutaneous anæsthesia and analgesia of the whole of the sole of the foot, and sensation may be diminished in the region of distribution of one or more of the cutaneous nerves as far up as the calf or knee. The surface is usually cold in the anæsthetic area, and the extremity is likewise apt to become livid on slight exposure, while it is prone to attacks of inflammation or of eczema. These inflammatory attacks sometimes implicate the subcutaneous tissues; the limb then becomes greatly swollen and œdematous, and the attack occasionally terminates in suppuration. Lesions of the articulations of the foot frequently accompany this affection, and not only is the joint in direct relation with the wound diseased, but it is also not uncommon to meet with more or less complete ankylosis of all the phalangeal, metatarso-phalangeal, and tarso-metatarsal articulations, while subluxations of these joints may take place in other cases. It is, however, probable that extensive disease of the bones and joints of the foot only occurs when perforating ulcer is a symptom of locomotor ataxia. The nails assume a brownish color; they become greatly thickened, curved longitudinally and laterally, furrowed, dry, and cracked. The skin of the leg becomes at times pigmented, and there is an increase in the growth of the hair, while the foot is bathed in sweat which has a permanently fetid odor. The ulcer is generally situated over the metatarso-phalangeal articulations, most frequently over those of the big and little toes. There may be as many as three ulcers on one foot, and when both feet are affected the disease is generally symmetrical. Perforating ulcer has on rare occasions been met with in the hands. The disease is essentially chronic; the ulcers may, under favorable circumstances, remain stationary for a long time, and may even heal under prolonged rest, but a relapse readily occurs when the patient begins to walk.

The most recent observations on perforating ulcer of the foot show that it is very frequently, if not always, associated with other symptoms of locomotor ataxia. It was known a long time ago that this form of ulcer was often accompanied by shooting pains and anæsthesia in the lower extremities, while strabismus and other ocular troubles were also mentioned as being present in such cases. It is now found that patients with perforating ulcer also suffer from gastric crises, arthropathies, swaying movements on closing the eyes, tottering or unsteady gait, and

absence of the patellar-tendon reactions. Perforating ulcer is associated with the neuralgic form of locomotor ataxia, and motor disorders do not form prominent features of such cases. It may be one of the earliest symptoms of the disease, and may even precede the anæsthesia of the lower extremities, so that its association with locomotor ataxia is not always readily made out. In a case which was under the care of my colleague, Mr. Hardie, there was some degree of anæsthesia of the lower extremities, but the patellar-tendon reactions were exaggerated, and I was about to conclude that the case was not one of locomotor ataxia. A closer examination, however, showed that the patient had suffered from diplopia, and that the pupils failed to react to light, but reacted to accommodation. The presence of these symptoms rendered it very probable that the patient was in the early stage of locomotor ataxia, notwithstanding the patellar-tendon reactions were exaggerated. Dengenerative lesions of the nerves have been found, on microscopical examination, in several cases of perforating ulcer.

5. *Unilateral Progressive Atrophy of the Face* (*Hemiatrophia Facialis Progressiva*).—This disease has been observed about twice as often in women as in men, and it generally begins between 10 and 15 years of age, although it has occasionally been observed as early as 2 and as late as 30 years of age. The characteristic phenomena of this disease may be preceded for some time by such symptoms as a local herpetic eruption, toothache, tearing pains in the head and superior maxillary region, epileptiform attacks in which the spasms are sometimes more or less limited to the side of the face, an attack of hemiplegia, spasms of the masticatory muscles, and hyperæsthesia with paræsthesia in the side of the face, which is afterwards the subject of atrophy.

The first definite symptom to attract notice is a peculiar discoloration of circumscribed areas of the skin. Small spots of a white color and slightly depressed appear on the side of the face, and these gradually spread so as to coalesce into a patch of considerable size. The affected area may now assume a yellowish or brownish tint like that often observed in cicatrices after burns, the skin over them becomes thin and emaciated, the subcutaneous fat disappears, and the side of the face becomes deformed by pits of greater or less size and depth. On the affected side the eyeball often sinks back into the orbit from disappearance of the orbital fat, the palpebral fissure is narrowed, the beard, eyelashes, and hair of the head become gray and undergo other structural changes, and the secretions of the sebaceous follicles is arrested, but the functions of the sweat glands appear to be normally

performed. In advanced cases the affected skin feels irregular and atrophied, and it may assume the form of a cicatrix, but does not become adherent to the underlying structures. Cutaneous sensibility is not, as a rule, much affected, but patients sometimes complain of various paræsthesiæ in the atrophied portions of skin, and others have suffered from neuralgiform attacks, while in one case partial anæsthesia, and in a few others hyperæsthesia was present.

The muscles are not usually implicated in the atrophy, but in a case observed by Eulenburg and Guttmann the masticatory muscles on the affected side were relatively feeble and emaciated, in a few cases atrophy of one half of the orbicular of the mouth was observed, and in other cases one half of the tongue, the veil of the palate, and the uvular were found atrophied, but the muscles always gave normal electrical reactions. Romberg reports a case occurring in an unmarried woman, aged twenty-eight years. The left side of her face had gradually atrophied as the result of extensive suppuration on the left side of the neck, which had burst through the tonsil. Every feature, including the brow, eye, nostril, lips, cheek, and chin, as well as the left half of the tongue and left arch of the palate, was smaller than those on the opposite side.

The *large arteries* of the face are generally unaltered in size, and the tone of the small arteries is retained or increased. The atrophied parts are generally capable of blushing, and also redden under local electrical excitation. The temperature is the same on both sides.

The *bones* of the face have been found decidedly diminished in volume. The upper and lower maxillary bones and the cartilages of the nose are often atrophied, especially when the disease begins at an early age. Romberg was the first to give an accurate description of this affection, and he classified it amongst the trophoneuroses. The trophic fibres are not likely to run in the motor branch of the fifth nerve, inasmuch as paralysis and atrophy of the masticatory muscles, although occasionally present, are never prominent symptoms. Cases have been described by Seeligmüller and Brunner, in which the symptoms appeared to have been caused by disease of the cervical sympathetic, but it is probable that, as suggested by Müller, the lesion is situated in the medulla oblongata. This disease is, with occasional long pauses and recommencements, a progressive one, and no treatment has hitherto been of any avail.

6. *Neuroparalytic Ophthalmia.*—This affection begins with congestion of the conjunctiva, which is followed by profuse secretion of mucus or pus, insensibility and opacity of the cornea, and a pseudo-membranous exudation of the iris. In a few days ulceration and perfora-

tion of the cornea may occur, which is followed by escape of the humors and collapse of the eye.

Several hypotheses have been advanced to account for this destructive inflammation of the eyeball, but the most probable is that it is due to irritation of the trophic fibres which descend from the casserian ganglion to the eyeball.

7. *Simple Glaucoma.*—Experiments on animals have shown that irritation of the nucleus of the trigeminus in the medulla, or of the nerve itself, is followed by increase of the intraocular pressure, caused by augmented secretion of the aqueous humor. As a result of the high tension the iris and lens are pushed forwards, and the internal membranes are stretched. It is supposed by many pathologists that glaucoma is produced by a similar mechanism, although this opinion is not accepted by all.

v. Nutritive Affections of the Joints, Bones, and Teeth (Articular and Osseous Trophoneuroses).

1. *Affections of Peripheral Origin.*—Traumatic injuries of nerves, in which the nerves are not completely divided, are often followed, any time after the first few days, by disease of the joints, which consists of a painful swelling like that of subacute articular rheumatism. This swelling may attack any or all of the articulations of a limb, and often begins in the joints remote from the injury, so that it cannot be caused by direct extension of inflammation from the wound. After the acute stage is over, the tissues about the affected articulations become thickened, and partial ankylosis results, which may ultimately destroy the mobility of the joint. The bones also may become swollen and thickened after injuries of nerve-trunks, and in young people the same bones may, at a later period, be arrested in their development. In progressive unilateral atrophy of the face the bones participate, to some extent, in the wasting.

2. *Affections of Spinal Origin.*—Attention has been directed by Charcot and his scholars to the great frequency with which nutritive changes occur in joints in spinal diseases. These joint affections may be divided into acute or subacute, and chronic arthritis.

The *acute or subacute* form is accompanied by more or less severe pain, tumefaction, and redness, just as occurs in acute rheumatism. This form occurs in Pott's curvature, traumatic lesions of the cord, idiopathic myelitis, progressive muscular atrophy, acute and chronic poliomyelitis, and disseminated sclerosis.

Chronic arthritis is generally observed in association with locomotor

ataxia. The knee, hip, shoulder, and elbow-joints are most frequently attacked, although the fingers and toes are also liable to be affected. When the tarsal bones are affected a characteristic deformity is produced which is named the *tabetic* foot. The joint disease usually begins about the same time as the locomotor incoördination, and its onset is accompanied or preceded by severe paroxysms of lancinating pains. The symptoms begin suddenly in the absence of any appreciable external cause, generally without pain or febrile reaction, and the joint may be enormously swollen within twenty-four hours from the commencement. The general tumefaction disappears after a few days, but a more or less considerable local swelling remains, caused by the accumulation of serous fluid in the joint and the periarticular serous bursæ. The fluid, however, disappears from the joint in a few weeks from the onset. In the *benign* form of the affection the joint may recover completely, but in the *malignant* form the articular surfaces become greatly altered and so roughened that cracking sounds are heard on movement. After a time the heads of the bones become atrophied and worn, the ligaments become relaxed, and the surrounding muscles are so much atrophied and enfeebled that spontaneous laxations may occur.

Fractures.—Spontaneous fractures occur not infrequently in the course of locomotor ataxia. The period of fracture is generally preceded by two or three severe paroxysms of lancinating pains, the limb is then found swollen and presenting all the symptoms of osteo-periostitis, and it then becomes fractured on the slightest movement, or in the absence of any movement or external cause. The femur is more frequently fractured than any other bone, the seat of fracture being generally the neck, but the bones of the leg, arm, forearm, and indeed almost every bone in the limbs and trunk, including those of the vertebral column, have been found fractured. The spontaneous fractures of ataxics often reunite very readily and rapidly, with an enormous formation of callus. The earthy phosphates are diminished in the bones which undergo spontaneous fractures, forming sometimes only sixty instead of eighty per cent. of the bone as in health, while the fatty constituents are enormously increased in amount.

3. *Osseous Affections of Cerebral Origin.*—In the spastic hemiplegia of infancy the bones on the paralyzed side are arrested in their development, being smaller and shorter than the corresponding bones on the opposite side. In hemiplegic patients arthropathies of the joints of the hand and foot are met with which are like the acute arthropathies of spinal origin. The affection begins with a slight swelling and local increase of temperature, either with or without pain in the joint, and at times tumefaction and redness are so marked as to resemble the

articular affections of acute rheumatism. The sheaths of the tendons are sometimes implicated along with the joints. These arthropathies occur, as a rule, simultaneously with late rigidity, although it may begin a few days after the attack or at a much later period.

4. *Osseous Lesions in the Insane.*—In insane patients the bones may either become so soft that they yield readily to pressure, and thus produce various deformities, or so fragile that they are liable to undergo spontaneous fracture, and may be found to crumble readily under the finger and thumb after death. Out of 100 post-mortem inspections of the insane made by Gudden, evidences of fracture were found in 16 cases, and chiefly in men who had suffered from general paralysis. In three-fourths of these cases there were multiple fractures; in one case as many as 14, in another 23, and in another 36 fractures. The morbid changes which occur in the bones of the insane are closely related to those observed in the spontaneous fractures of locomotor ataxia.

5. *Trophic Affections of the Teeth.*—The teeth are not very liable to undergo changes in diseases of the nervous system. They have been known to fall out after an attack of herpes affecting the maxillary branches of the fifth nerve, probably more from necrosis of the bone than from disease of the teeth themselves. Attention has, in recent years, been directed to the fact that the teeth sometimes fall out suddenly in the course of locomotor ataxia, and in the absence of pain or caries of the bone. They have also been observed to fall out in a case of sclerosis in patches.

VI. Nutritive and Secretory Affections of the Glandular Apparatus.

1. *Cutaneous Secretory Disorders.*

Various pathological facts appear to prove the existence of cutaneous secretory nerve fibres independently of the vaso-motor nerves. Diminution or absence of the secretion of sweat may at times exist side by side with local increase of temperature and redness, indicating vaso-motor paralysis; and, conversely, the secretion may be increased in amount along with local diminution of temperature and pallor of the surface, indicating vaso-motor spasm. Recent experiments show that peripheral irritation of a divided sciatic nerve in animals induces an increased secretion of sweat in the paralyzed part.

The cutaneous secretory neuroses consist of excessive sweating or *hyperidrosis*, diminution or absence of secretion or *anidrosis*, and

qualitative changes which may be grouped under the name of *paridrosis*. The profuse sweating of acute disease, that which results from the action of various toxic agents, and the partial sweats which occur during hysterical and epileptoid attacks, are doubtless of nervous origin. Still more striking examples are to be found in the unilateral perspirations which have been described under the name of *hyperidrosis unilateralis*. This affection is sometimes limited to one-half of the head, and at other times extends to the arm of the same side, or even extends over one-half of the body, and is usually associated with severe nervous affections, such as hemicrania, Graves's disease, diabetes mellitus, tabes dorsalis, and general paralysis of the insane. It is probably caused by lesion of the sympathetic or of the cerebro-spinal centres, with which it is united.

Anidrosis is a symptom of fever, diabetes mellitus, chronic Bright's disease, and of certain skin diseases, and is often associated with grave nervous diseases like general paralysis of the insane. The diminution of perspiration caused by various toxic agents like atropine is evidently due to action on the nervous system. A good example of local dryness of the skin occurs in unilateral atrophy of the face, and a similar local condition may also be found on the extremities in the course of most of the different cerebral, spinal, and peripheral chronic nervous affections.

Paridroses of various kinds have been observed in diseases of the nervous system. In some nervous affections the secretion emits a peculiar, generally very offensive, odor—*osmidrosis*. S. Weir Mitchell observed excessive sweating, with strong odor of vinegar, after severe contusion of peripheral nerves, and in one case the smell resembled that of a bad drain. The secretion at other times becomes changed to a black, blue, red, or green color, *chromidrosis*. Colored perspiration generally occurs in hypochondriacs, in women with uterine disorders of various kinds, or as the result of severe emotional disturbance. In some few cases extravasation of blood takes place into the sweat glands, giving rise to bloody sweating or *hæmatidrosis*. This condition appears to be occasionally vicarious menstruation, but it is usually associated with hysteria and other central nervous affections. It is probable that in most of the cases described as hæmatidrosis the coloring matter of the blood alone escapes.

2. *Secretory Disorders of the Salivary Glands.*

Secretory disorders of the salivary glands may occur in connection with lesions of the peripheral fibres of the trigeminus, the facial nerve, or the cervical sympathetic. An increased secretion of saliva is not

an unusual symptom of trigeminal neuralgia, caused by irritation of the lingual branch of the fifth as the afferent, and the chorda tympani as the efferent channel. Stimulation of the glosso-pharyngeal nerve also causes an increased flow of saliva. In peripheral paralysis of the facial nerve, caused by lesion of the nerve in the Fallopian canal, where it is accompanied by the chorda tympani, the secretion of saliva is deficient on the paralyzed side, because the secretory fibres of the submaxillary and sublingual glands pass through the chorda tympani. In paralysis of the cervical sympathetic the salivary secretion is diminished in quantity because the parotid gland receives a portion of its secretory fibres through the cervical sympathetic. Irritation of the sensory nerves of the stomach, especially great acidity of the gastric secretions, causes an increased flow of saliva, which appears to be an automatic action, by means of which the gastric acidity becomes neutralized. Certain poisons, like atropine, paralyze the secretory fibres of the chorda tympani, and lead to a diminution or arrest of the salivary secretion; whilst others, like digitalis, physostigmin, nicotin, and jaborandi, irritate the secretory fibres and cause an increased flow of saliva. The secretion of saliva may be influenced by direct or reflex action on the intracerebral secretory paths. Bernard found that an increased flow of saliva is produced by puncture of the floor of the fourth ventricle behind the origin of the trigeminus, and it is probable that the increased flow of saliva observed in bulbar paralysis may at times be due to irritation of this point. An enormously increased flow of saliva has been observed by Eulenburg in dogs, after destruction by the actual cautery of portions of the cortex of the brain lying in front of the cruciate sulcus. The saliva flowed out in a constant stream from the angle of the mouth on the opposite side to the injured hemisphere, and was of the same thin watery character which is observed after irritation of the chorda tympani.

3. *Secretory Disorders of the Lachrymal Glands.*

Trigeminal neuralgia gives rise to an increase in the amount of lachrymal secretion both by reflex and direct action, the secretory fibres of this gland being contained partly in the lachrymal and partly in the subcutaneous malar nerve. After traumatic injury of the cervical sympathetic there is an increased flow of tears on the affected side, probably from vaso-motor paralysis, and the free flow of tears which often takes place towards the termination of an attack of hemicrania is probably caused by a similar mechanism. Many anomalies in the flow of the lachrymal secretion are doubtless of central origin, such as the

copious flow which occurs in hysterical attacks and in connection with emotional disturbances.

4. *Secretory Disorders of the Glands of the Digestive Tract.*

An increase in the flow of the secretions of the stomach and intestines appears to be caused by affections of the vagus, or of the sympathetic plexuses and ganglia. Diarrhœa and vomiting are often caused by emotional disturbance, and they may also accompany hysterical attacks, while apepsia and constipation are frequent accompaniments of numerous cerebral diseases. Paroxysms of vomiting, and less frequently of diarrhœa, are also prominent symptoms of locomotor ataxia.

5. *Secretory Disorders of the Glands of the Genito-urinary Apparatus.*

The influence of the nervous system on the secretion of urine is very great, but the various channels by means of which it is conveyed are not accurately ascertained. The most notable examples are polyuria and diabetes mellitus. Various anomalies may occur in the secretions of the vagina, uterus, and mammæ in hysterical females, although it is doubtful whether they are due to affections of vaso-motor or secretory nerves. In the condition known as irritable uterus there are often, in addition to the sensory disturbance, numerous anomalies of circulation and secretion, which are probably of reflex origin. Similar phenomena may be associated with ilio-lumbar neuralgia. Attacks of hysteria are often followed by an abundant secretion of mucus from the vagina, and erotic thoughts may give rise to an obstinate discharge in the absence of any organic lesion. In "irritable testis" the organ and the spermatic cord are often swollen, and many cases of spermatorrhœa, pollutions, and aspermatism may probably be caused by functional disturbances of secretory or motor nerve fibres issuing from the lumbar portion of the spinal cord. Pollutions may be caused by reflex irritation, or by an increased excitability of the centre of ejaculation in the spinal cord in such diseases as tabes dorsalis, while spermatorrhœa is caused most probably by debility of the vesiculæ seminales.

VII. Nutritive Affections of the Viscera (Visceral Trophoneuroses).

It has not been found possible to separate the vaso-motor and trophic fibres of the viscera, either with regard to their anatomical distribution or their functions. It is very likely that the congestion, ecchymoses,

and extravasations which occur in various central nervous diseases are caused by implication of vaso-motor nerves. Cerebral hemorrhage is frequently accompanied by pulmonary apoplexy or pneumonia of the lung on the side opposite to the lesion; while lesions of the nuclei of the origin of the pneumogastric, or of the nerve itself, may give rise to pneumonia and fatty degeneration of the heart. The most frequent consequences of extirpation of the cœliac and mesenteric plexuses in animals are congestion of the liver, congestion and extravasations in the stomach and intestines, and diabetes—all of them symptoms which are likely to be caused by vaso-motor paralysis.

CHAPTER VI.

GENERAL SYMPTOMATOLOGY (*continued*).

C. THE ELEMENTARY KINESIONEUROSES.

THE movements of the body and of its different parts are caused by muscular contractions, the muscle being stimulated to act by impulses received through the nervous system, when a nervous stimulus gives rise to a muscular contraction. Elementary motor disorders admit of a double classification—the one anatomical, the other physiological, the two traversing each other at right angles.

Anatomical Classification.—Elementary motor disorders may be divided into those which affect (I) the muscles of external relation—the *external kinesioneuroses*, (II) the muscles of the internal organs—*internal* or *visceral kinesioneuroses*, and (III) the muscular fibres of the vascular system—the *vascular kinesioneuroses*, or the *angioneuroses*.

Physiological Classification.—Motor disorders may be divided into (1) the conditions in which there are excessive muscular contractions—*spasms* or *hyperkineses*, (2) those conditions in which there is a diminution or loss of the power of exciting the muscles to contraction—*paralyses* or *akineses*, and (3) various anomalous muscular contractions which may be comprised under the name of the *synkineses*. The hyperkineses and akineses may be subdivided, according to the function of the nervous apparatus which is the seat of the lesion, into three classes, namely: *a*, the voluntary, *b*, the reflex, and *c*, the automatic kinesioneuroses.

I. THE EXTERNAL KINESIONEUROSES.

Methods of Examining the Motor Apparatus.

As an introduction to this part of the subject it will be useful to describe briefly the various methods employed for discovering the presence or absence of motor disorders of the external muscular apparatus. The striped muscles are, for the most part, connected with the skeleton, and by their contractions they move the bones to which they are attached, and thus constitute the active agents in maintaining the various attitudes of the body. The graceful form of the body is also

in great measure due to the rounded bellies of the muscles which help to fill up the space between the bones and the skin and the subcutaneous tissues. In subjecting the neuro-muscular mechanism of external relation, therefore, to a methodical examination we must attend (1) to the form of the body during repose, and (2) to the attitudes of the body during actual or attempted movements.

1. Examination of the Body during Repose.

The bulk and consistence of a muscle as felt through the skin may, as a rule, be accepted as a more or less adequate sign of the degree of its nutrition and motor power, and consequently an examination of the exposed body by inspection and palpation affords much valuable information with regard to the motor mechanism. If, for instance, the outer surface of the shoulder is seen to be flattened and the head of the humerus can be felt immediately underlying the skin, it is known that the deltoid is wasted, and if the opposite shoulder is at the same time plump and rounded, it is inferred that the deltoid of the affected shoulder is wasted from a special and not from a general cause. But if the deltoid, instead of being wasted, is unduly prominent and tense, and the elbow is held permanently removed from the trunk, it is at once apparent that the muscle is the subject of spasm. Distortion of the limbs often shows at once that certain groups of muscles must be paralyzed, or that their antagonists are in a state of spasm, and performance of passive movements will generally decide this question, showing directly whether a particular group of muscles is relaxed or tense. In the case of spasm valuable information may be obtained by producing a corresponding deformity on the healthy side by means of the faradic current, and in the case of paralysis, by exciting the antagonists of the affected muscles to contraction by means of faradization.

2. Examination of the Body during Movement.

The muscles of external relation may be excited to contraction by (*a*) voluntary, (*b*) reflex, (*c*) automatic, (*d*) mechanical, and (*e*) electrical stimuli, and the response of the muscles to one or more of these stimuli may be wanting, excessive, or otherwise disordered.

(*a*) *Voluntary Movements.*—The student should make a careful study of the different forms of disordered locomotion, and be able to distinguish at a glance the hemiplegic walk, the spastic gait, and the loose and dangling limbs of the various forms of atrophic paralysis. The patient should be asked to perform special movements, such as standing on one leg, standing with feet touching along their inner

borders (the eyes being closed), ascending a stair, writing, speaking, and mimetic facial movements, in order that any deviation from the normal may be carefully observed. Attention should be directed to ascertain whether incapacity to perform a particular movement is caused by paralysis of a group of muscles or spasm of their antagonists. An approximate estimate can be formed of the degree of paralysis present by comparative testing of the resistance which can be opposed by passive movements, while a more accurate test of motor power which can be increased by certain groups of muscles is afforded by the various forms of *dynamometers*. It is also important to observe whether the muscular contraction can be maintained for some time without inducing exhaustion, and whether the intended movement is executed with precision and steadiness, or is interrupted by tremors and antagonistic secondary movements. Slowness in executing certain movements is a sign of diminished motor power, and feebleness of the muscles of the hand, for instance, may sometimes be more readily detected by asking the patient to write his name or to perform the "devil's tattoo" on the table, than by estimating the strength of the grasp.

(*b*) *Reflex movements* may be tested by tickling, pricking, pinching, and faradic excitation of the skin and accessible mucous membranes. The reflexes of the special senses must be tested by their special excitants, while it is most important to examine the deep reflexes by tapping tendons, fasciæ, and the periosteum.

(*c*) *Automatic Movements.*—The movements of respiration and of the iris, and the action of the sphincters, may be mentioned as examples of automatic actions which ought to be carefully observed. In ordinary locomotion, the observer ought to be able to distinguish at a glance the ataxic walk, the cerebellar reel, the staggering gait of Ménière's disease, and the uncertain, tremulous walk of sclerosis in patches.

(*d*) *Mechanical Stimuli.*—A moderately strong blow over almost any muscle induces a contraction of the fasciculus struck, especially if the blow fall near the point of entrance of the motor nerves. In exhausting diseases like phthisis, when the nutrition of the muscles is greatly diminished, a sharp blow causes the formation of a wheal which lasts several seconds, and from which small waves of contraction run in both directions towards the extremities of the muscle, this reaction being called idio-muscular contraction.

(*e*) *Electrical Stimuli.*—The electrical currents usually employed for clinical investigation are (1) the faradic and (2) the galvanic currents.

(1) *The faradic current* consists of a series of isolated currents, each of momentary duration, and of very rapid development and decline, following each other in quick succession and flowing alternately in

opposite directions. *Faradic excitability or irritability* is the term used to designate the kind and strength of the reactions exhibited by muscles and nerves under the influence of the faradic current. Muscular contraction may be induced by the *direct* application of the faradic current to the muscles themselves, or *indirectly* through excitation of the motor nerves. The cathode of the secondary induced current is usually employed as the exciting pole, while the anode may be placed upon some indifferent part of the body, as the sternum or patella. Direct excitation of accessible muscles is best performed when the poles are applied over the points at which the motor nerves enter the muscles, which may be ascertained by reference to Ziemssen's diagrams. The faradic excitability of both nerves and muscles in diseased conditions admits only of quantitative changes. It may remain normal or be slightly increased in cases of muscular spasm, in that form of muscular paralysis which is attended by tension, and in hysterical paralysis; while it is diminished or lost in that form of paralysis which is attended by wasting of the muscles, with the exception of the active stage of progressive muscular atrophy, in which it may be increased. The degree of the excitability runs a more or less parallel course in the nerves and muscles.

(2) The *galvanic current* is continuously produced and runs in the same direction, and with the same intensity, but by means of the commutator the current may be interrupted at pleasure, or even quickly reversed, a change which induces a very powerful contraction.

Galvanic excitability or irritability is a term used to express the reactions obtained in response to opening and closing the circuit, and to the continuous passage of the current. The law of contraction both of motor nerves and muscles rests upon the facts that the cathode produces contraction chiefly on closure of the current, the anode chiefly on opening the current, and that the stimulus of the cathode is stronger than that of the anode.

The law of normal contraction may be expressed by the following formulæ.

Let An = anode, Ca = cathode, C = medium contraction, c = feeble contraction, C′ = strong contraction, S = closure of current, O = opening of current, Te = tetanic contraction, thus:

Weak currents produce CaSc.

Medium currents produce CaSC′, AnSc, AnOc.

Strong currents produce CaSTe, AnSC, AnOC, CaOc.

In diseased conditions the law of contraction may remain (*a*) normal, or deviations from the normal law may occur by way of (*b*) increase or

(*c*) diminution of the excitability, or by changes in (*d*) the quality of the various reactions.

(*a*) *A normal degree of the galvanic excitability* is obtained in cases of muscular spasm, in hysterical paralysis, and in paralysis attended by muscular tension.

(*b*) *Simple increase of the galvanic excitability* occurs in certain forms of spasmodic paralysis, *tabes dorsalis*, and as a transient symptom in a few cases of peripheral paralysis. Increase of the excitability gives rise to the following deviations from the normal law:

Weak currents produce CaSC′, AnOC.

Medium currents produce CaSTe, AnOC′, CaOC.

Strong currents produce CaSTe, AnOTe, CaOC′.

(*c*) *Simple diminution of the galvanic excitability* occurs in all those cases in which paralyzed muscles undergo a minor degree of atrophy. The following formulæ express simple diminution of excitability:

First degree, strong currents produce CaSC, AnSc, AnOc.

Second degree, strong currents produce CaSc.

(*d*) *Qualitative as well as quantitative alterations of the excitability* are found in all those cases in which paralyzed muscles undergo a considerable or a profound degree of atrophy, and this kind of reaction has consequently been called the "atrophic test," or the "reaction of degeneration." The alterations in the reaction of the nerves and the muscles do not run a parallel course, so that the two must be separately described.

(j) *Reaction of the Affected Nerves.*—The reaction in the affected nerves begins on the second and third day after a severe attack of that form of paralysis which will be subsequently described as atrophic paralysis, and a continuous uniform diminution of the galvanic as of the faradic excitability is observable without any qualitative change, and in rare cases only is it preceded by slight increase. The diminution begins in the part nearest the lesion, and extends rapidly to the periphery. At the end of the first, or in the course of the second week (from the seventh to the twelfth day), the excitability wholly disappears. In incurable cases the loss of the excitability is permanent, but if repair of the diseased tissue takes place, the excitability, after being lost for a variable period, is restored. The reactions to both currents appear simultaneously, beginning first in the central segments of the nerve and spreading slowly to the periphery.

(jj) *Reaction of the Affected Muscles.*—The reactions obtained by the application of the *faradic* current directly to the muscles are quite similar to those obtained from the application of the current to the degenerated nerves. When the electrode is placed over the para-

lyzed muscle a diminution of the excitability is observed towards the end of the first week, and in severe cases there is complete extinction of it towards the end of the second week. When the case is incurable the faradic contractility of the muscle is permanently abolished, but in curable cases it reappears along with the restitution of voluntary power, although usually somewhat later than in the nerves. As recovery proceeds the faradic excitability increases gradually but slowly, and generally remains for a long time abnormally low, especially if the paralysis has been of long duration.

The *galvanic excitability* falls slightly during the first week, but in the course of the second week it becomes enormously increased and continues to increase until about the end of the fourth week. The affected muscles now respond to currents much too feeble to act upon healthy muscles. The character of the contractions is also changed, and instead of appearing suddenly and being of short duration, as in health, they now develop gradually and slowly, are protracted in duration, and readily pass into tetanus even when produced by feeble currents.

The law of muscular contraction also becomes qualitatively altered, so that anodal closing contraction soon equals or exceeds cathodal closing contraction (AnSC = or > CaSC), and cathodal opening contraction soon equals or exceeds anodal opening contraction (CaOC = or > AnOC). The following formulæ express the qualitative changes in the reaction of degeneration:

Weak currents produce during stage of increase CaSC, AnSTe, AnOC, CaOC′.

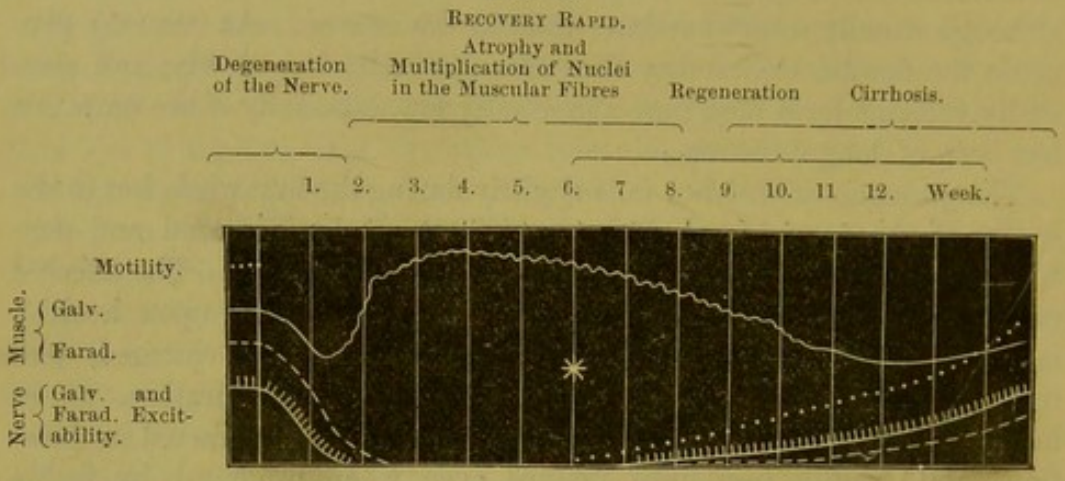
Medium currents produce during stage of gradual decrease CaSc, AnSTe.

Strong currents produce during final stage prior to abolition AnSc.

The following diagrams, borrowed from Erb, represent graphically the general relations of motor power, electrical excitability and structural changes of the nerves and muscles which are present in the different stages of paralysis. The first thick vertical line or ordinate indicates the sudden appearance of paralysis (▬), and the period of return of motor power is indicated by a (*), whilst the succeeding ordinates represent intervals of one or more weeks dating from the occurrence of the attack. The undulations in the line representing the galvanic excitability of the muscles indicate qualitative changes in the reactions. In the first degree of the reaction of degeneration (Fig. 66), the electrical excitability of both nerve and muscle falls during the first week, the nerves lose all their electrical reactions during the second week, but the muscles lose only their faradic con-

tractility during this period, whilst the galvanic excitability becomes greatly increased and manifests the qualitative changes already described. At the end of the sixth week there is a gradual return of

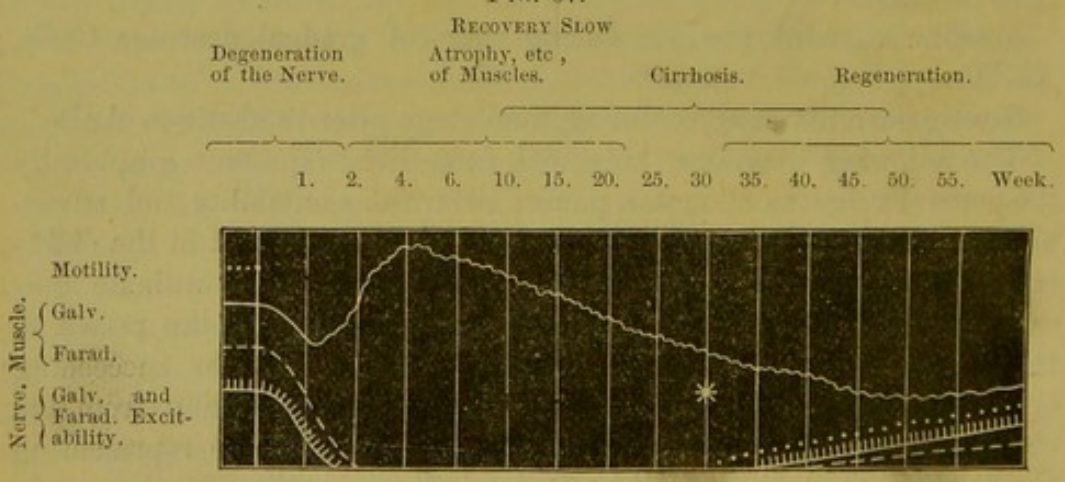
FIG. 66.



motor power, and at the end of the seventh week there is a gradual return of the electrical reactions of the nerve and of the faradic contractility of the muscle, while the galvanic reactions of the muscle gradually sink, and the qualitative changes disappear until gradually the normal reaction is established.

In the second degree of the reaction of degeneration the faradic

FIG. 67.

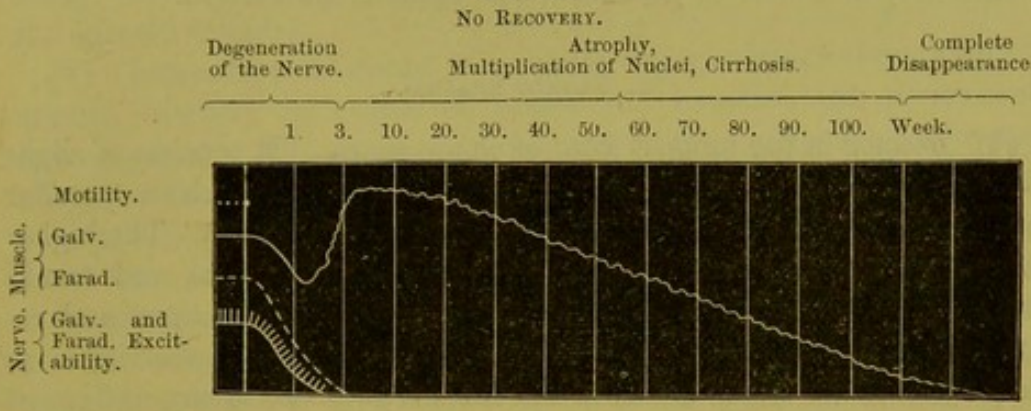


and galvanic excitability of the nerve does not appear until the thirtieth week (Fig. 67), while in the third degree (Fig. 68) the excitability of the nerve never returns, but the galvanic excitability of the muscle

only becomes finally lost after a prolonged period, in some cases extending over a period of two years.

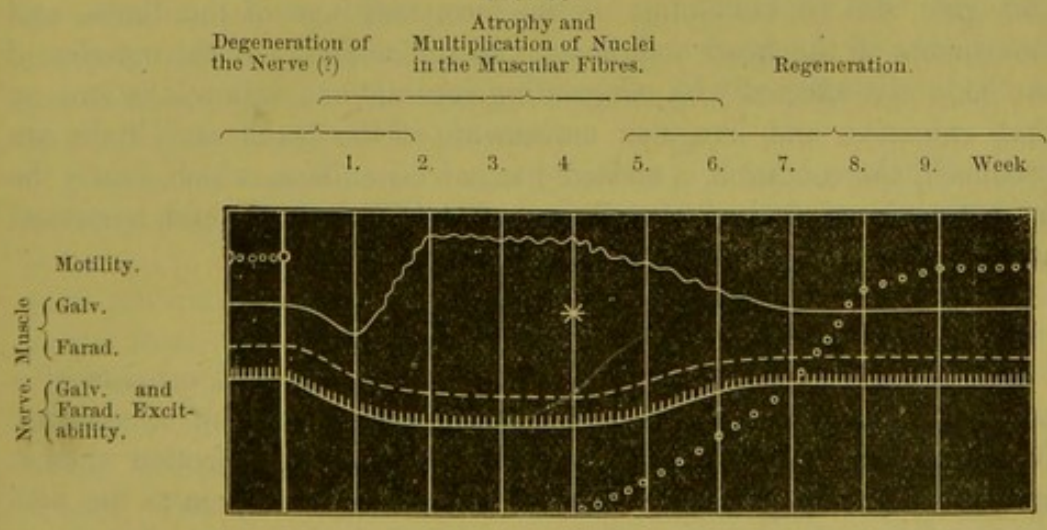
A "partial reaction of degeneration" (Fig. 69) has been described by Erb, in which the faradic and galvanic excitability of the affected

FIG. 68.



nerve is diminished, but not abolished, the diminution being sometimes only to a slight degree. The faradic excitability of the paralyzed muscle undergoes a diminution corresponding to that of the nerve, but the galvanic excitability of the muscles manifests the quantitative and qualitative changes which are so characteristic of the severer form of the reaction of degeneration.

FIG. 69.



1. *Hyperkinesis of the Muscles of External Relation.*

Hyperkinesis of the voluntary muscles consists of abnormal contractions called *spasms*. Muscular spasms consist of contractions which

are disproportionate to the degree of external stimulus, or which arise in the absence of external stimulation as the result of morbid irritation. Spasmodic affections may be divided into, *a*, *clonic spasms*, in which the muscles are in a state of rapidly alternating contractions and relaxations; and, *b*, *tonic spasms*, in which the affected muscles are maintained in a state of persistent and equable contraction.

a. Clonic Spasms.

(1) *Tremor* is the mildest form of clonic spasm. It consists of slight contractions of groups of muscles by means of which a peculiar rhythmical oscillation of the limbs and trunk is produced. The higher degrees of tremor cause manifest trembling of the limbs and trunk, while *fibrillary contractions* consist of alternate contractions and relaxations of individual bundles of muscular fibres which are visible as wavy oscillations under the skin, but do not give rise to any movement of the limb. There are two chief varieties of the severer form of tremor which moves the limbs: one of which persists during repose, and is met with in paralysis agitans; and the other which appears only when the patient makes a voluntary effort, and is observed in sclerosis in patches.

(2) *Convulsion* is the severest form of clonic spasm. It consists of energetic contractions and relaxations of particular muscles or groups of muscles which produce a rapid succession of vigorous movements, and give rise to twitchings of the face, startings of the limbs, and movements of the head and body. If the majority of the muscles of the body are affected with alternating contractions and relaxations, so that extensive and irregular movements of the trunk and limbs are produced, the condition is termed *general convulsion*, which forms the most prominent feature of epilepsy, uræmia, eclampsia, and hysterical attacks.

b. Tonic Spasms.

(1) *Cramp* is the simplest form of tonic spasm, and consists of a persistent painful contraction of a muscle or of a group of muscles. Cramp of the calf is the most common variety of this affection when it is limited to a single muscle, while tetanus may be taken as the best example when the majority of the muscles of the body are affected.

A peculiar modification of cramp is met with in catalepsy. The muscles are moderately contracted, but the resistance they offer to passive movements may be readily overcome, and the limbs may be made to assume constrained positions which they retain. From the

manner in which the limbs can be moulded into various positions this condition has been called *flexibilitas cerea*.

(2) *Muscular tension* is a state of moderate contraction of muscles which occurs either when they are stretched by passive movements or by a voluntary contraction of their antagonists. This condition is always associated with a certain degree of loss of voluntary power over the affected muscles.

(3) *Contracture* is meant to express any persistent shortening of a muscle, whereby its points of origin and insertion are permanently approximated. The varieties of contraction are *myopathic contracture*, when the shortening occurs as a result of disease in the muscle itself; *paralytic or secondary contracture*, when it occurs in healthy muscles which have their ends permanently approximated owing to paralysis of their antagonists; and *primary or neuropathic contracture*, when the muscles are persistently rigid and shortened from abnormal innervation, a condition which is always associated with a certain degree of paralysis. In the last variety the rigidity of the muscle usually disappears during sleep and gradually returns on awakening, and it is almost always increased by voluntary and passive movements.

Pressure points are frequently observed in spasmodic affections. Pressure upon certain points puts a stop at times to the convulsion when present, and consequently these points may be called *pressure-arresting points*. In other cases the convulsions are brought on by pressure on particular points, and these may, therefore, be called *pressure-exciting points*. Pressure points of the first kind have been particularly observed in facial spasm, and they correspond, like the painful points in neuralgia, to the various branches of the trigeminus, and are not unfrequently sensitive to pressure.

Spasms are caused by increased irritability of some part of the motor nervous mechanism. When the spasm, whether it be clonic or tonic, is limited to the area of distribution of particular nerves, the lesion is most probably situated in some part of the spino-neural system, either in the centre or efferent fibre, or in some afferent fibres, the last constituting *reflex spasms*. Paroxysmal clonic convulsions are caused by irritation of the cortex of the brain, and certain forms of tonic and clonic spasms are caused by irritation of the fibres of the pyramidal tracts. Paroxysmal tonic contractions are most probably caused by irritation of the cortex of the cerebellum. Persistent muscular tension and contracture are caused by disease of the pyramidal tracts, and are associated with paralysis. Dr. Hughlings Jackson explains the presence of tension under such circumstances by supposing that the withdrawal of the cerebral influence allows the tonic action of the cere-

bellum to become predominant. Against this theory it is urged that tension is present in cases of transverse myelitis, in which both the cerebral and cerebellar influences are withdrawn. It is therefore probable that muscular tension is caused by the removal of the inhibitory action of the cerebrum, owing to disease of the pyramidal tracts, permission being thus given for the predominant action of the reflex nervous mechanisms of the spinal cord.

2. *Akinesis of the Muscles of External Relation.*

By akinesis or paralysis of the muscles of external relation is understood the diminution or abolition of the power to contract the affected muscles by voluntary effort. The term *paresis* is used to denote the diminution of motor power. Some authors have endeavored to restrict *paralysis* to its complete abolition, but this term will be employed here in a generic sense as embracing both conditions.

Classification.

Paralyses of the muscles of external relation are susceptible of being classified according to the nature, cause, and situation of the lesion, the distribution of the paralysis, and the functional disorders of the muscles which accompany paralysis of them. These divisions constitute the—I, pathological, II, etiological, III, topographical, IV, clinical, and, V, physiological classification.

I. THE PATHOLOGICAL CLASSIFICATION.

The different varieties of paralysis are arranged, according to the *nature* of the lesion, into rheumatic, syphilitic, inflammatory, and other forms. They may also be divided into *organic* and *functional* lesions, according as the morbid changes which underlie the paralysis are or are not capable of being recognized by our present means of research.

II. THE ETIOLOGICAL CLASSIFICATION.

The *functional* paralyses are not susceptible of being arranged according to the situation of the lesion, and they are therefore usually classified according to the cause of the affection. They are usually divided into (1) toxic, (2) febrile and post-febrile, (3) reflex, (4) post-epileptic, and (5) hysterical paralysis.

III. THE TOPOGRAPHICAL CLASSIFICATION.

The organic paralyses lend themselves readily to a classification according to the situation of the lesion. They may be divided into (1)

myopathic paralyses, or those in which the primary disease is situated in the muscles themselves, and (II) *neuropathic* paralyses, or those in which the primary disease is localized in some part of the nervous system.

The *neuropathic* paralyses, with which we have to do chiefly here, may be subdivided into (1) cerebral, (2) spinal, and (3) neural or peripheral paralyses, according as the lesion is situated in the brain, spinal cord, or peripheral nerves respectively. But although this division is very convenient, a much more important distinction is that which divides them into (1) *cerebro-spinal* and (2) *spino-neural* paralyses.

In the *cerebro-spinal* variety the lesion is situated either in the motor centres of the cortex of the brain or in the pyramidal tracts.

In the *spino-neural* variety the lesion is situated in the anterior gray horns of the spinal cord and their upward continuations in the medulla oblongata, pons, and crura cerebri, or in the fibres of the peripheral nerves which connect the ganglion cells of these horns with the muscles. It will immediately be found that this topographical division corresponds more or less closely with the divisions of the physiological classification, and this constitutes its chief advantage.

It may here be noticed that when the lesion is restricted to one of the physiological tracts of the spinal cord the affection is called a *system-disease*, and when several of them are simultaneously implicated the affection is called a *mixed or indiscriminate* disease. Amongst the *mixed* diseases we shall also include complicated cases of cerebral paralysis.

IV. THE CLINICAL CLASSIFICATION.

Various names have been given to a paralysis according to its extent and distribution. The paralysis is sometimes limited to a single muscle or group of muscles, or all the muscles supplied by a single nerve or plexus of nerves may be implicated; when all or almost all the muscles of a single extremity are paralyzed, the condition is called a *monoplegia*. In other cases the paralysis affects both halves of the body symmetrically, and then it generally begins in the lower extremities, and spreads to the trunk and upper extremities. This is the usual form of paralysis which results from disease of the spinal cord, and is termed *paraplegia*. In other cases the paralysis affects the lateral half of the body, implicating the face, arm, and leg of the same side, and it is then termed *hemiplegia*. The lesion which causes this form of paralysis is usually situated in the opposite hemisphere of the brain; hemiplegia of spinal origin is named *hemiparaplegia*. When the upper and lower extremities on both sides are paralyzed, the condition, when due to spinal dis-

case, has been named *paraplegia cervicalis*, and when caused by cerebral disease, *bilateral hemiplegia*, or *pamplegia*. When the ocular or facial muscles on one side, and the limbs on the opposite side, are paralyzed, the condition is called *crossed* or *alternate hemiplegia*.

V. THE PHYSIOLOGICAL CLASSIFICATION.

A much more important distinction than those depending upon the extent and distribution of the affection is that which divides the various forms into (1) *atrophic*, and (2) *spastic or spasmodic paralysis*. This division does not embrace every form of paralysis, inasmuch as in some paralytic affections the muscles neither undergo active wasting, nor are affected by spasm. This distinction is, nevertheless, a very important one, and ought to be kept in view in the clinical examination of every case of paralysis. Both atrophic and spasmodic paralyses embrace some forms of myopathic paralysis, but, when these are due to an organic disease of some part of the nervous system, they correspond respectively to the spino-neural and cerebro-spinal paralyses of the topographical classification.

The different methods of classification are combined into one scheme in the following tables:

TABLE I.—*Neuropathic Paralyses.*

A. ORGANIC PARALYSES.

I. SPINO-NEURAL OR ATROPHIC PARALYSES.

i. Neural or Peripheral Paralysis.

ii. Reflex Atrophic Paralysis.

iii. Spinal Atrophic Paralyses.
1. Acute Atrophic Spinal Paralysis of Infants.
2. Acute Atrophic Spinal Paralysis of Adults.
3. Paralysis Ascendens Acuta.
4. Chronic Atrophic Spinal Paralysis.
5. Peri-ependymal Myelitis—Syringomyelia.
6. Progressive Muscular Atrophy.
7. Primary Labio-glosso-laryngeal Paralysis.
8. Ophthalmoplegia Externa.
9. (Pseudo-hypertrophic Paralysis.)

II. CEREBRO-SPINAL OR SPASMODIC PARALYSES.

i. Spinal Spasmodic Paralyses. (PARAPLEGIÆ.)
1. Primary Lateral Sclerosis.
2. Compound Lateral Sclerosis.
   a. Amyotrophic Lateral Sclerosis.
   b. Combined Posterior & Lateral Sclerosis.
3. Secondary Lateral Sclerosis.
   a. Compression Myelitis.
   b. Transverse Myelitis.

ii. Cerebral Paralyses. (HEMIPLEGIÆ.)
1. Tonic Spasm.
   a. Early Rigidity.
   b. Late Rigidity.
2. Tonic & Clonic Spasm.
   a. Intermittent Tremor.
   b. Choreiform Movements.
      i. Pre-hemiplegic Chorea.
      ii. Post-hemiplegic Chorea.
      iii. Spastic Hemiplegia of Infancy.
3. Clonic Spasm.
   a. Continuous or Remittent Tremors.
   b. Athetosis.
   c. Post-hemiplegic Hemiataxia.

III. MIXED PARALYSIS.

B. FUNCTIONAL PARALYSES.

(1) Toxic Paralysis.
(2) Febrile and Post-febrile Paralysis.
(3) Reflex Paralysis.
(4) Post-epileptic Paralysis.
(5) Hysterical Paralysis.

TABLE II.—*Paralyses from Organic Disease of the Nervous System.*

CLINICAL DIAGNOSIS.	TOPOGRAPHICAL DIAGNOSIS.
I. ATROPHIC PARALYSES.	I. SPINO-NEURAL LESIONS.
I. NEURAL OR PERIPHERAL PARALYSES.	I. LESIONS OF EFFERENT NERVE FIBRES.
II. REFLEX ATROPHIC PARALYSIS.	II. LESIONS OF AFFERENT NERVE FIBRES.
III. SPINAL ATROPHIC PARALYSES.	III. LESIONS OF THE ANTERIOR GRAY HORNS. (POLIOMYELOPATHIES.)
1. Acute Atrophic Spinal Paralysis of Infants.	Poliomyelitis Anterior Acuta Infantium.
2. Acute Atrophic Spinal Paralysis of Adults.	Poliomyelitis Anterior Acuta Adultorum.
3. Acute Ascending Paralysis.	Poliomyelitis Acuta.
4. Chronic Atrophic Spinal Paralysis.	Poliomyelitis Anterior Chronica.
5. Peri-ependymal Myelitis. 6. Progressive Muscular Atrophy. 7. Primary Labio-glosso-laryngeal Paralysis. 8. Ophthalmoplegia Externa.	Degeneration of the Ganglion Cells of the Anterior Horns of the Spinal Cord and Motor Cells of the Medulla Oblongata.
9. (Pseudo-hypertrophic Paralysis.)	(Primary Muscular Disease.)
II. SPASMODIC PARALYSES.	II. CEREBRO-SPINAL LESIONS. (PYRAMIDAL TRACT.)
I. SPINAL SPASMODIC PARALYSES.	I. LESIONS OF THE LATERAL COLUMNS.
1. Primary Spinal Spasmodic Paralysis.	Primary Lateral Sclerosis.
2. Compound Spinal Spasmodic Paralysis.	Amyotrophic Lateral Sclerosis. Combined Posterior and Lateral Sclerosis.
3. Secondary Spinal Spasmodic Paralysis.	Compression Myelitis. Transverse Myelitis.
II. CEREBRAL PARALYSES.	II. LESIONS OF THE CEREBRAL PYRAMIDAL TRACT AND MOTOR AREA OF CORTEX.
1. Ordinary Hemiplegia.	Lesions of Lenticular Nucleus. Area of Lenticulo-striate Artery.
2. Alternate Hemiplegia.	Lesions of Crura and Pons.
3. Hemiplegia and Hemianæsthesia.	Lesions in Area of Opto-striate Artery.
4. Hemiplegia, Hemianæsthesia, and Hemianopsia. 5. Pre-hemiplegic Chorea. 6. Post-hemiplegic Chorea. 7. Athetosis. 8. Post-hemiplegic Continuous Tremor and Hemiataxia.	Lesions in the Area of the Posterior External Optic Artery.
9. Spastic Hemiplegia of Infancy.	Unilateral Atrophy of the Motor Area of Cortex.
10. Unilateral Convulsions and Hemiplegia.	Porencephalus. Lesions of Motor Area of Cortex.

3. *Reflex Central Kinesioneuroses.*

The disorders which occur in the reflex mechanisms which are situated in the different parts of the nervous system are almost infinitely numerous, but we shall here mention only a few of the more usual disorders of the spinal reflexes. The spinal reflexes may be divided into (1) the superficial, and (2) the deep reflexes, while disorders of the reflex mechanisms may declare themselves by way of an excessive reaction, constituting (*a*) *reflex hyperkinesis*, or by diminution or loss of reaction, constituting (*b*) *reflex akinesis.*

(1) The Superficial Reflexes.

The superficial reflexes are excited by stimulation of the skin and accessible mucous membranes. The tests employed for estimating the various degrees of these reflexes are tickling, pricking, pinching, or gently scratching the surface, or the application of the faradic current to the surface by means of dry electrodes or the faradic brush.

The following superficial reflexes may be distinguished (Fig. 70):

(*a*) The *plantar reflex*, obtained by tickling the sole of the foot and depending upon the integrity of the reflex loops through the lower end of the cord (conus medullaris).

(*b*) The *gluteal reflex*, consisting of contraction of the gluteal muscles caused by stimulating the skin over the buttock, and depending upon the integrity of the loops through the fourth and fifth lumbar nerves.

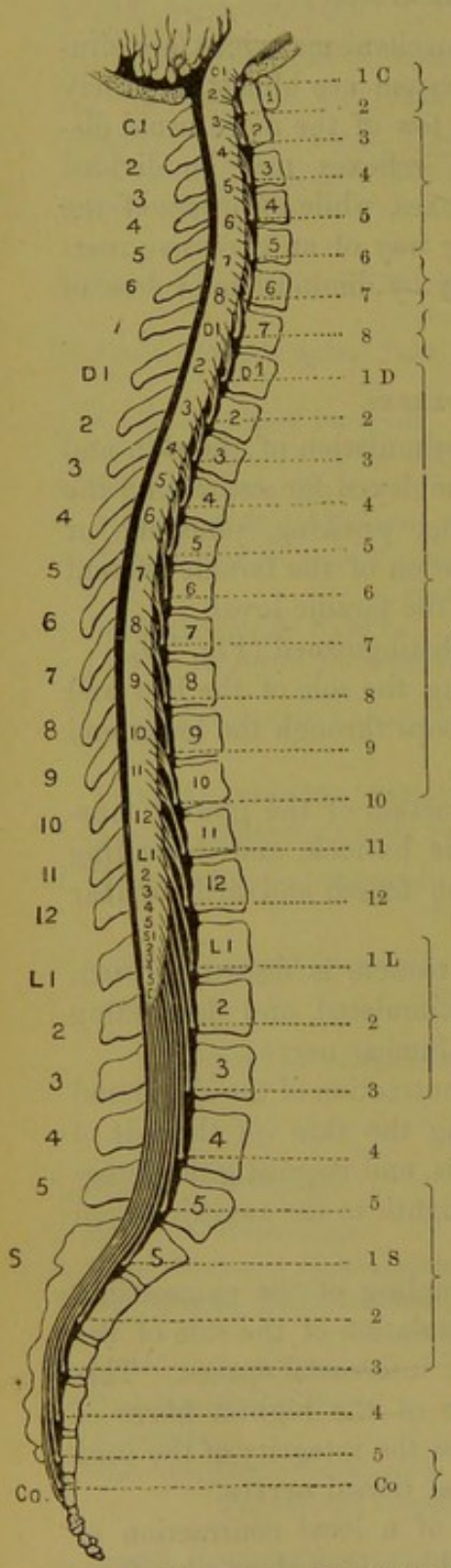
(*c*) The *cremasteric reflex*, by which the testicle is drawn up when the skin on the inner side of the thigh is stimulated, and demanding the integrity of the first and second pairs of lumbar nerves.

(*d*) The *abdominal reflex*, consisting of a contraction of the abdominal muscles, chiefly the rectus, caused by stroking the skin on the side of abdomen from the edge of the ribs downwards, and requiring the integrity of the arc through the nerves from the eighth to the twelfth dorsal nerves.

(*e*) The *epigastric reflex*, producing a dimpling of the epigastrium on the side stimulated. It is induced by stimulation of the side of the chest in the sixth, fifth, and sometimes fourth intercostal spaces. This dimpling probably depends upon contraction of the highest fibres of the rectus abdominis, and its presence requires the integrity of the cord from the fourth to the sixth or seventh pairs of dorsal nerves.

(*f*) The *erector spinal reflex*, consisting of a local contraction of these muscles, caused by stimulation of the skin along their edge from

FIG 70.



	Motor.	Sensory.	Reflex.
1 C	Sterno-mastoid	Neck and scalp.	
2			
3	Trapezius; Rhomboids and rotators of humerus; Diaphragm	Neck and shoulder.	
4			
5	Deltoid, biceps, brachialis, supinators. Serratus.	Outer aspect of arm and forearm.	Scapular.
6	Triceps, extensors of wrist and fingers, and pronators	Anterior and posterior aspects of arm, forearm, outer half of hand, and 2½ fingers.	
7			
8	Flexors of wrist and long flexors of fingers		
1 D	Intrinsic muscles of hand	Inner aspect of arm, forearm, inner half of hand, and 2½ fingers.	
2			
3			
4			
5	Intercostal muscles	Front of thorax; Ensiform cartilage	Epigastric.
6			
7			
8			
9			
10	Abdominal muscles	Abdomen; Umbilicus; Buttock, upper part	Abdominal.
11			
12			
1 L			
2	Flexors, hip	Groin and scrotum (front)	Cremasteric
3	Extensors, knee	Thigh: Outer side, Front side, Inner side	*Knee-jerk.*
4			
5	Adductors; Abductors; Extensors (?) — Hip	Leg, inner side; Buttock, lower part; Back of thigh	Gluteal.
1 S			
2	Flexors, knee (?)	Leg and foot, except inner part	*Foot-clonus.* Plantar.
3	Muscles of leg moving foot.		
4	Perineal and anal muscles	Perineum and anus.	
5			
Co		Skin from coccyx to anus	

DIAGRAM AND TABLE SHOWING THE APPROXIMATE RELATION TO THE SPINAL NERVES OF THE VARIOUS SENSORY AND REFLEX FUNCTIONS OF THE SPINAL CORD. (After GOWERS.)

the angle of the scapula to the iliac crest, and demanding the integrity of the reflex arcs in the dorsal region of the spinal cord.

(*g*) The *scapular reflex*, consisting of a contraction of some, or nearly all, of the scapular muscles according to its degree, and demanding the integrity of the cord at the level of the upper two or three dorsal and lower two or three cervical nerves.

(*h*) The *palmar reflex* consists of a contraction of the flexors of the fingers induced by tickling the palm of the hand. It requires the integrity of the reflex arcs through the greater part of the cervical enlargement. This reflex is not readily induced during waking hours and consequent cerebral activity, probably because the hand is much more under cerebral influence than the foot. During sleep, however, and in young infants, when the cerebral influence is suspended, or not yet fully established, this reflex is as readily induced as the reflex of the sole of the foot.

(*i*) *Cranial Reflexes.*—The chief reflexes of the cranial nerves are the contraction of the palatal muscles caused by irritation of the fauces; the facial contortions caused by irritation of the fifth nerve; the sneezing and lachrymation caused by irritation of the mucous membrane of the nose; the cough caused by irritation of the mucous membrane of the larynx; the closure of the eyelids caused by irritation of the conjunctiva; and the reflex contraction of the iris caused by light.

Some of these reflexes are absent in healthy individuals more especially the reflexes of the back and abdomen, so that the diminution or absence of these must not be taken as a sure sign of disease. Their presence, however, is a proof that the respective paths through the cord are not seriously interrupted.

a. HYPERKINESIS OF THE SUPERFICIAL REFLEXES.

Increase of the reactions obtained from reflex stimulation indicates that the irritability of the reflex arc is increased in some portion of its course, or that the inhibitory influence of the cerebrum is withdrawn. Exaggerated reflex reactions are obtained by increased irritability (j) of the afferent portion of the arc; (jj) of the efferent portion of the arc; (jjj) of the gray matter of the cord, as in tetanus and strychnia poisoning; (iv) by arrest of the functions of the pyramidal tracts (Fig. 71, 6, 7). The condition of the cutaneous reflexes in cerebral paralysis, however, appears to be an exception to the rule that withdrawal of the cerebral influences causes excess of the reflex actions, inasmuch as the cutaneous reflexes are diminished or lost on the paralyzed side in hemiplegia. The diminution of the cutaneous reflexes under such

circumstances is caused probably by a loss of tone of the muscular fibres distributed to the skin, which prevents the papillæ from being properly exposed to the irritation of tickling and other cutaneous stimulants.

b. AKINESIS OF THE SUPERFICIAL REFLEXES.

When stimulation of the surface produces either feeble or no reactions, it indicates that there is diminution or loss of the irritability (j) of the afferent portion of the arc—examples of which are met with in destructive lesions of the sensory branches of the fifth nerve; (jj) of the efferent fibres—examples being afforded by lesions of the facial nerve and the anterior roots of the spinal nerves; (jjj) of the gray matter of the anterior horns—examples of which are afforded by the spinal atrophic paralyses, and by the diminution of the reflexes which is observed after large doses of bromide of potassium; and (iv) by increased cerebral action conveyed through the pyramidal tracts—an example being afforded by the inhibition of reflex actions by means of voluntary control.

(2) THE DEEP REFLEXES.

a. TENDON REACTIONS.

If a man in health sits with one leg crossed upon the other, and the ligamentum patellæ be then smartly struck immediately below the knee-cap, the extensor muscles in front of the thigh become suddenly contracted, and the foot is jerked forwards to a variable extent according to the degree of contraction. The jerk is named the *patellar-tendon reaction, knee-phenomenon, or knee-jerk.* The blow is usually delivered by the inner edge of the hand; but an ordinary stethoscope, held loosely by the small end while the blow is struck by the edge of the ear-piece, is a convenient instrument for the purpose, and a Wunterich percussion hammer is a still more efficient instrument. It is also desirable to uncover the knee that the blow may be delivered on the bare skin. In stout people, who cannot cross one leg over the other in a sitting posture, the operator may pass his hand beneath the patient's thigh just above the knee-joint, and, grasping the opposite knee, support the extremity to be examined by his forearm. Another convenient position is to get the patient to sit on a table or on an elevated seat with the legs hanging freely. Similar reactions may be obtained by striking the tendons of the upper extremity, but these are not well marked except under certain diseased conditions. The reactions are best elicited by striking with the edge of the stethoscope the tendon of the triceps at the elbow, and those of the supinator longus and extensor muscles at the wrist.

b. VARIOUS FORMS OF CLONUS.

Ankle-clonus, or *Achilles-tendon reaction*, consists of a rhythmical clonic spasm, which can be obtained under certain circumstances at the ankle-joint. When the necessary conditions are present, the reaction is most readily elicited if the operator will support the lower extremity of the patient by placing his hand behind the knee-joint, the leg being slightly bent upon the thigh, while with the right hand he seizes hold of the toes lightly between the fingers and thumb and puts the tendo Achillis suddenly upon the stretch by producing a quick and energetic dorsal flexion of the foot, and then maintaining pressure against the toes for some time. When the tendon is first stretched, the gastrocnemius immediately contracts and the toes are depressed; the muscle now relaxes, and, the pressure of the hand being still continued, the toes are once more elevated, when the muscle again contracts and depresses the toes a second time; and thus the contractions and relaxations are continued in rhythmic sequence so long as the tension of the tendo Achillis is maintained. This series of rhythmic contractions constitutes ankle-clonus, and, so long as the movement continues, between eight and ten contractions of the gastrocnemius take place in a second of time.

Toe-clonus.—In cases of increased tension of the foot, rhythmical contractions of the big toe may be obtained by producing sudden passive extension of the first phalanx, flexion being produced by contraction of the abductor and flexor brevis pollicis.

Wrist-clonus.—Movements like those of ankle-clonus may be obtained in the hand, in cases of late rigidity of hemiplegia, by grasping the tips of the fingers and pressing the hand backwards so as to produce hyperextension at the wrist.

c. PERIOSTEAL AND FASCIAL REACTIONS.

The best known of these movements are the contraction of the quadriceps femoris muscle, induced by gently tapping the front of the tibia near its middle; that of the biceps on tapping the lower end of the radius with the edge of the stethoscope; and that of the triceps on tapping the lower end of the ulna, in cases of muscular rigidity with contraction. When the muscles of the shoulder and arm are implicated in the rigidity, contractions of the pectoralis major, deltoid, and biceps may be obtained by a gentle tap on the sternal end of the clavicle, and even a crossed reaction may be obtained by a tap on the clavicle of the opposite side. Under similar circumstances a considerable

number of the muscles of the scapula and shoulder contract on tapping the spine of the scapula. Contractions of the erector muscles of the spine may, under certain circumstances, be induced by tapping the lumbar fascia.

d. SPINAL EPILEPSY.

The paroxysms of violent tremors which occur in certain affections of the spinal cord, and which Brown-Séquard named spinal epilepsy from a fancied similarity to an epileptic convulsion, are of a compound nature. These movements appear, indeed, to be caused by contractions produced by simultaneous and successive stimulations of the superficial and deep reflexes. A painful cutaneous impression causes reflex contractions of both lower extremities, which predominate in the flexors; but when the anterior flexors of the legs contract the Achilles tendons are put upon the stretch, and ankle-clonus results. These actions and reactions, reverberating for a time through all the muscles, maintain the lower extremities in a state of tremor, which may be so violent as to shake the bed on which the patient reposes. When these tremulous movements are proceeding, if the toes of one foot be grasped by the hand and brought suddenly and powerfully into plantar flexion, the muscles immediately relax and the tremors cease for a time.

e. PARADOXICAL CONTRACTION.

Some muscles may, under certain circumstances, be made to contract by suddenly approximating their points of origin and insertion. The curious fact that a sudden relaxation of a muscle causes it to contract has led Westphal, who was the first to direct attention to this phenomenon, to name it *paradoxical contraction*. This symptom is best studied in the tibialis anticus, which may, in certain nervous diseases, be made to contract by producing a sudden, or sometimes a gradual, dorsal flexion of the foot. When the patient is laid on his back in bed, and the muscles are relaxed, the feet occupy the position of extensor or plantar flexion. If dorsal flexion of the foot be now produced, the tibialis anticus, under certain not well-ascertained circumstances, enters into contraction, its tendon becomes prominent, and the foot is maintained for some minutes in the position of dorsal flexion and adduction. Distinct resistance is also offered to the production of plantar flexion of the foot by passive movements. After a variable interval of time the muscle relaxes, either gradually and continuously or with several intermissions, and the foot falls by its own weight to the position of plantar flexion. The paradoxical contraction sometimes extends to the extensor

communis digitorum and extensor brevis pollicis. It is possible that this anomalous reaction is caused by an impression upon the sensory nerves of the joints, and not by the sudden relaxation of the muscle.

f. HYPERKINESES OF THE DEEP REFLEXES.

(j) It is probable that increased irritability of both the afferent and efferent portions of the reflex arcs will increase the activity of these reactions, but no crucial examples of this kind have as yet been described. When, however, the irritability of the muscular fibres themselves is increased in degree, as in phthisis and other exhausting diseases, the tendon reactions are much more readily induced than in health.

(jj) Irritation of the posterior roots, and of the efferent fibres as they pass through the inner radicular fasciculus of the posterior columns, probably also increases the activity of the tendon reactions, but direct evidence upon this point is as yet wanting.

(jjj) Increased irritability of the gray substance of the spinal cord causes increase of the activity of the tendon reactions. The patellar-tendon reaction is, for example, exaggerated by the administration of strychnia, and probably also by such diseases as tetanus and hydrophobia.

(iv) The tendon reactions are increased in activity when the cerebral influence is withdrawn from the spinal cord by disease of the pyramidal tracts, and consequently all these reactions are exaggerated in all forms of spasmodic paralysis, unless, indeed, the reflex arc is at the same time arrested by a local spinal disease like chronic spinal pachymeningitis.

g. AKINESES OF THE DEEP REFLEXES.

(j) The tendon reactions are diminished or lost in disease of the afferent portion of the reflex arc. The disease may be situated in the tendon itself; in such a case the movements of the joint are generally interfered with by an ankylosis, and the conditions are not favorable for applying the test. The reaction may be lost when the lesion is situated in the peripheral course of the nerve (Fig. 71, 1), but the motor and sensory fibres are then interfered with, and under such circumstances it is not possible to prove that disease of the afferent fibres takes any part in the arrest of the reaction. This reaction may, however, be lost in spinal meningitis affecting the posterior roots (Fig. 71, 2), when the absence of any atrophic paralysis of the corresponding muscle shows that the afferent portion of the reflex arc is intact. The patellar-tendon reaction is, for instance, often absent in

(jj) This reaction is lost when the afferent fibres are diseased in their passage through the inner radicular fasciculus (Fig. 71, 3) of the pachymeningitis affecting the lumbar region in the absence of any atrophic paralysis of the quadriceps femoris muscle.

Fig. 71.

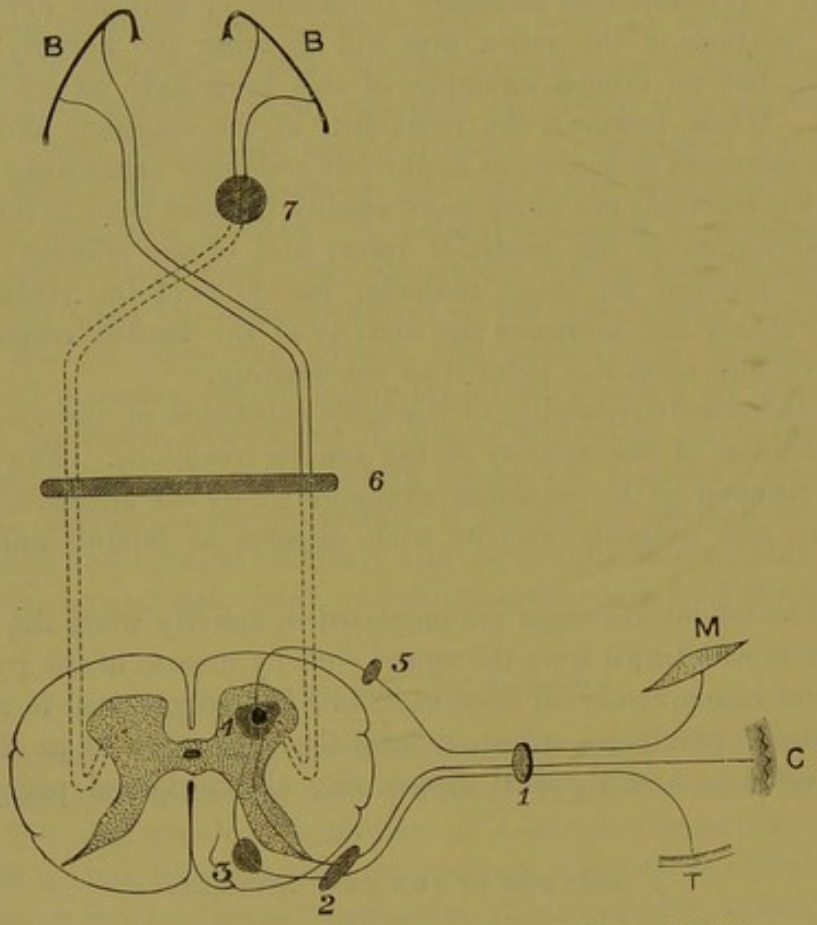


Diagram of the Reflex Functions of the Spinal Cord. (Young.)

C, skin and, T, tendon with their afferent nerve fibres, both terminating in the ganglion cell of the anterior gray horn, from which issues an efferent fibre which connects the cell with the muscle M. B B, the cortices of the cerebral hemispheres, with their attached pyramidal tract fibres connecting the cortical giant cells with the ganglion cells of the anterior gray horns. 1, lesion of the peripheral nerve, causing atrophic paralysis, loss of sensation, and abolition of both kinds of reflexes; 2, lesion of the posterior root (pachymeningitis), causing loss of sensation and abolition of both kinds of reflexes, but no paralysis; 3, lesion of the posterior root-zone (tabes dorsalis), causing loss of the tendon reflex only; 4, lesion of the anterior gray horns (poliomyelitis), causing atrophic paralysis and loss of both kinds of reflexes, but no sensory disorder; 5, lesion of the anterior root (pachymeningitis), causing the same symptoms as 4; 6, transverse lesion of the spinal cord, causing a spasmodic paralysis of the lower extremities, with excess of both kinds of reflexes; 7, focal lesion of the cerebral hemisphere, causing hemiplegia of the opposite side, with excess of the tendon reflexes on the paralyzed side, but with diminution of the cutaneous reflexes. The dotted lines indicate descending sclerosis of the pyramidal tracts

spinal cord, this being the essential anatomical change in locomotor ataxia. It will hereafter be found that loss of the patellar-tendon reaction is the earliest and most constant symptom of locomotor ataxia.

(iii) The tendon reactions are diminished or lost in depressive and destructive lesions of the gray anterior horns. Large doses of bromide of potassium and of opium diminish the activity of the tendon reaction, and it is very probable that a poison circulates in the blood which diminishes or abolishes these reactions in certain cases of diabetes. Destructive lesions (Fig. 71, 4) of the anterior gray horns abolish the tendon reactions, but the loss of the reactions is accompanied by paralysis with atrophy of the corresponding muscles.

(iv) The tendon reactions are likewise lost in destructive lesions of the efferent portion of the reflex arc (Fig. 71, 5), and in this case, also, the abolition of the reaction is accompanied by paralysis with atrophy of the muscles. Disease of the muscle itself abolishes the reflex, the early loss of the patellar-tendon reaction in pseudo-hypertrophic paralysis being a good example.

(v) Increase of cerebral influence on the gray substance of the spinal cord diminishes the activity of the tendon reactions. The patellar-tendon reaction can, for instance, be arrested by voluntary effort, and it is diminished or lost during the convulsive stage of an epileptic attack. The conditions under which paradoxical contraction is obtained are not well ascertained.

h. AUTOMATIC KINESIONEUROSES.

The groups of symptoms which may be included under automatic affections of the muscles of external relation, cannot be distinctly separated from the reflex and voluntary affections of these muscles. There are, however, disturbances of muscular adjustments in which the simple reflex actions of the spinal cord, the sensory mechanisms, and the voluntary mechanisms are normal, and yet in which complex muscular adjustments either fail to be effected in spite of all voluntary efforts to accomplish them, or are effected in spite of all voluntary efforts to prevent them, the last kind of adjustment being named *compulsory* or *forced movements*. It is such movements as these which are meant to be included under the name of automatic kinesioneuroses of the muscles of external relation. These movements are, in all probability, coördinated in the cerebellum and basal ganglia of the cerebrum.

(1) *Peripheral Automatic Disorders.*

Disorder of Labyrinthine Impressions.—Disease of the peripheral nerves generally involves either the sensory or the voluntary motor fibres, or both together, so that disorder of muscular coördination becomes thus obscured by the more prominent symptoms caused by

disorder of the voluntary mechanism. There is, however, a growing conviction that ataxia is frequently, if not always, caused by a peripheral lesion, but we shall at present adhere to the more classical view that it is caused by a spinal lesion. It would seem, however, that the seventh pair of nerves contain afferent fibres which are not subservient to the conduction of sensory impressions, and yet disease of which gives rise to the phenomena of motor incoördination. These fibres are involved in disease of the internal ear or of the semicircular canals, and in such cases the patient suffers from a staggering and uncertain gait, which will be subsequently described as Ménière's disease. The disorderly and incoördinate movements of animals after division of one or other of the semicircular canals are described in manuals of physiology.

(2) *Spinal Automatic Disorders.*

Ataxia is a very characteristic kind of motor incoördination observed in diseases of the spinal cord, and constitutes the most prominent feature of *tabes dorsalis*. It is characterized by inability to make combined or complicated movements with certainty and precision, and in advanced cases all movements requiring intricate and delicately balanced muscular adjustment become impossible. The motor incoördination usually presents itself in the most marked manner during locomotion and station, these being respectively named *dynamic* and *static ataxia*. These forms of muscular incoördination will be more fully described when locomotor ataxia is under consideration.

(3) *Encephalic Automatic Disorders.*

(*a*) *Reeling* is the well-known gait of a drunken man. It is caused by irregular swaying movements of the trunk, from side to side and from before backwards, requiring the legs to be moved irregularly in various directions in order to maintain their position vertically under the trunk. Staggering is a slighter degree of motor incoördination than reeling, and *vertigo* is the subjective correlation of this form of motor disorder.

(*b*) *Cerebellar rigidity* consists of rigidity of the muscles of the neck, which in aggravated cases extends to those of the back and extremities, so that complete opisthotonos is induced. This form of rigidity is frequently associated with tumor of the middle lobe of the cerebellum.

i. COMPULSORY OR FORCED MOVEMENTS—COÖRDINATE CRAMPS.

These movements are best seen in animals after experimental injury to various parts of the medulla, pons, and crura cerebri, but most of

them are probably caused by injury to one or other of the peduncles of the cerebellum. The usual forms of these movements are, that in which the animal rolls around the longitudinal axis of its own body, that in which it moves round and round in a circle, and that in which it rotates round the transverse axis of the body, tumbling head over heels in a series of somersaults. Movements of this kind are never so marked in man as in animals, but less degrees of these movements are sometimes observed in disease in the neighborhood of the peduncles of the cerebellum.

All the automatic disorders just described are caused by the overthrow of the delicate balance of the tonic muscular contractions, which is necessary for the maintenance of complicated adjustments in space. Irritative lesions of the cortex of the cerebellum, which is the organ for regulating these tonic contractions, gives rise to excessive tonic contractions of certain muscles, which destructive lesions cause paralysis of them, which, however, is not recognized as a paralysis, because the cerebral influence on the muscles is still intact. In other cases the cerebellum itself is healthy, but false intelligence is sent to it owing to disease of the cerebello-afferent conducting paths, and this leads to a loss of the balance of the tonic contractions of the body, or to loss of equilibration, as it is called.

k. SYNKINESIS.

Under this term are generally included certain involuntary movements of paralyzed parts, but we shall also include under it certain motor disorders which occur in muscles affected with spasm, as well as certain anomalous movements which occur in muscles that in health are associated in their actions with those primarily affected.

(1) *Associated Movements of Paralyzed Parts.*

In facial paralysis of cerebral origin the muscles of the paralyzed half of the face may occasionally perform the movements necessary to changes of expression in association with those of the opposite side, although in most cases the contrast between the actions of the two sides is rendered all the more evident under changes of expression. In cases of hemiplegia automatic movements may occur in a completely paralyzed arm. When the patient sneezes, and under the influence of emotional excitement, the paralyzed extremities may be strongly flexed, while the unaffected limbs remain passive. A voluntary movement of the healthy side is often accompanied by a contraction of the corresponding muscles on the paralyzed side.

(2) *Relative Immunity of some Muscles from Paralysis, and their Relative Liability to Convulsion.*

In ordinary cases of severe hemiplegia some muscles are completely paralyzed, while others are little if at all affected. The muscles which are most paralyzed are those of the extremities and the lower muscles of the face, and those which escape are the muscles of the trunk, and the upper muscles of the face. In unilateral convulsions, however, the spasm keeps limited to one-half of the body in the extremities and lower half of the face, while it often extends to both sides in the trunk and upper part of the face. Looking broadly at the muscles which are most liable to be completely paralyzed in hemiplegia, it may be said of them that they are the muscles which are most engaged in executing special movements, while those least liable to be paralyzed are engaged in effecting the most general movements. The muscles of the hand, for instance, which are so peculiarly liable to be paralyzed, effect the special movements of writing, while those of the trunk, which generally escape, are those which are engaged in carrying on respiration and other automatic actions. But the laryngeal muscles are engaged in effecting the very special movements of articulation, and yet they are by no means liable to be affected in hemiplegia, and consequently the rule just laid down is not of universal application, and we may, therefore, be certain that the muscles which remain comparatively free from paralysis have some other common characteristic. This characteristic is to be found in the fact, first pointed out by Dr. Broadbent, that the muscles which have a relative immunity from paralysis are those which are *bilaterally associated in their actions*. The thoracic, abdominal, and laryngeal muscles, and even those of the eyelids and eyebrows are brought into simultaneous action on both sides of the body, while those of the limbs, and, to a less extent, those of the lower part of the face, act more or less independently of one another on the two sides. This bilateral association of the functions of certain groups of muscles would lead us to expect that there will be a corresponding bilateral association of the nervous mechanisms by which their movements are regulated. Dr. Broadbent has suggested that in bilaterally associated actions the muscles of each side are connected with the cortical centres in both hemispheres. He believes that this connection is effected by means of spinal commissural fibres, and has happily enunciated the principle as the *bilateral association of the nerve nuclei of muscles bilaterally associated in their actions*. In Fig. 72, for instance, d', representing the spinal nuclei of the dorsal nuclei of the left side, is connected with the cortex of the opposite side by fibres (5 5) which ascend in the

pyramidal tract, and also with that of the same side through the commissural fibres which connect the two spinal nuclei (c'''), and the fibres ($5'$ $5'$) which connect the nuclei of the right side (d) with the left cortex (c'). Suppose now that the fibre 5 5 is ruptured and the nucleus d' is thus severed from the cortex of the opposite hemisphere, which usually controls its function, it can still obtain impulses from the cortex of the same side through $5'$ $5'$ and c'''. When, however, the muscles of the opposite sides act independently of each other, such as those of the right and left hands, commissural fibres are not established between their nerve nuclei. In the nucleus (a) of the right upper extremity, as represented in the figure, rupture of fibre $6'$ severs the connection with the cortex of the opposite side, and no channel is established by means of which it can obtain impulses from the cortex on the same side.

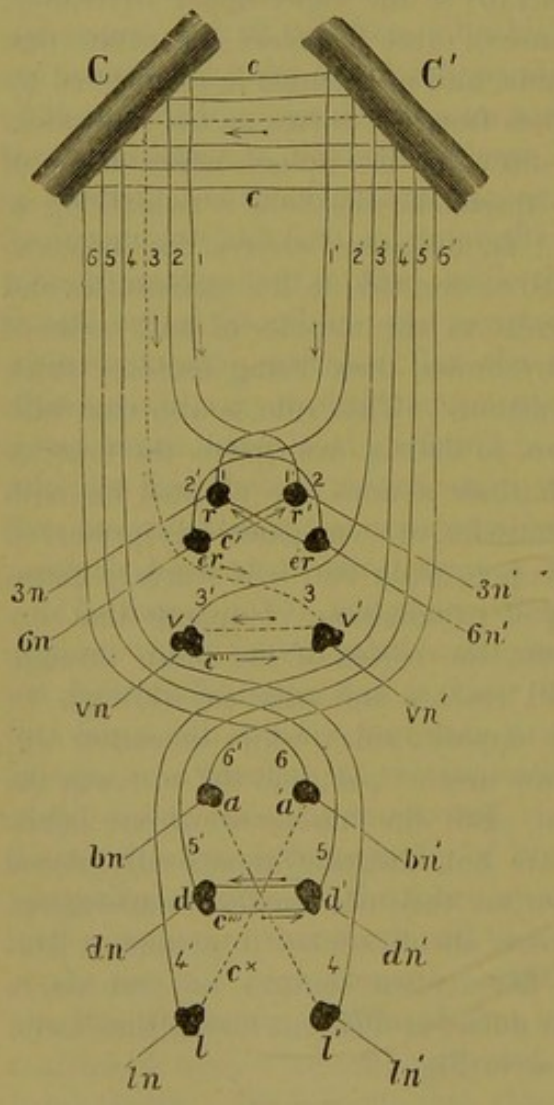
The effect which this bilateral fusion of the spinal nerve nuclei of various nerves produces in certain spasmodic affections is as striking as that produced by it in hemiplegia. In unilateral chorea, for instance, while the spasmodic action is limited to one side in the extremities and lower muscles of the face, it extends to the muscles of both sides of the trunk and of the eyelids and eyebrows, these being muscles which are habitually associated in their actions. This rule is also true with respect to other forms of convulsion, so that in *hemispasm* the muscles which are habitually associated in their actions are affected on both sides; while in *hemiplegia* these muscles are comparatively preserved from paralysis. The reason of this is so plain that it is scarcely necessary to add another word by way of explanation. Suppose that excessive impulses are sent down from the cortex of the brain through the fibres $6'$ and $5'$, the former will reach a and pass out through bn to the muscles of the arm on the opposite side; while the latter will reach d and pass both through dn and c''', d', and dn' to reach the muscles of the trunk on both sides. But the muscles which are habitually associated in their actions are not always symmetrically placed on the two sides of the organism, nor are their actions always analogous. It is only necessary that their actions should concur to produce a definite result; and the commissural fibres often connect, not two nerve nuclei on the same spinal level, but nuclei at different levels, thus forming an oblique crossed connection as in Fig. 72, $c^{\times}$.

(3) *Conjugate Deviation of the Eyes, and Rotation of the Head and Neck.*

The actions of the external rectus of one eye and of the internal of the other is a good example of muscles having quite opposite actions, and yet concurring to produce a harmonious result. It is evident that

if commissural connections exist anywhere they must exist between the nucleus of the sixth nerve of one side and the portion of the nucleus of the third nerve which presides over the action of the internal rectus of the opposite eye; and as these nuclei are placed at different levels in the pons and crura, the connection between them must be oblique and crossed. In Fig. 72, let r and r' represent respectively the nucleus of the portion of the third nerves ($3n$ and $3n'$) supplied to the right and left internal recti, and er and er' be respectively the nuclei of the

FIG. 72.



C, C', cortex of right and left cerebral hemispheres respectively; 1, 2, 3, 4, 5, 6, fibres of the pyramidal tract uniting C, the cortex of the right hemisphere, and r', $e'r$, v', a', d', l', the respective spinal nuclei of the internal rectus, and the external rectus muscles of the eye, the muscles of articulation and vocalization, those of the upper extremity, the dorsal muscles, and those of the lower extremity, all of the left side; 1', 2', 3', 4', 5', 6', fibres of the pyramidal tract, connecting the cortex of the left hemisphere with r, er, v, a, d, l, the spinal nuclei of the right side corresponding to those already enumerated on the left side; c, c, fibres of the corpus callosum uniting identical regions of the two hemispheres; c', commissural fibres connecting the spinal nucleus of the internal rectus muscle of one eye with that of the external rectus muscle of the opposite eye; c'', those connecting the spinal nuclei of the muscles of vocalization and articulation of the two sides; c''', those connecting the special nuclei of the muscles of the trunk; c^x, those connecting the spinal nuclei of the posterior extremity of one side with the anterior extremity of the opposite side. The arrows indicate the direction of the conduction.

right and left sixth nerves ($6n$ and $6n'$); while c' represents the crossing of the commissural fibres. The external rectus of one eye and the internal of the other eye acting simultaneously rotate both eyes so as to direct the axes of vision to lateral objects. When the object is placed to the right it is manifest that the right eye is in a better posi-

tion than the left to catch the first glimpse of it, hence the external rectus, which rotates the right eye outwards, takes the lead in the action. But the internal rectus of the opposite side rotates at the same time the left eye inwards; and to effect this movement it will be a clear gain of time, as well as economy of force, if it were to receive its impulses to action through the short commissural fibres which connect the two nuclei, and not from the cortex of the cerebrum of the opposite side. When, therefore, the eyes are directed by a voluntary effort to the right, the impulse to action may be supposed to come from the cortex of the brain (C′) on the opposite side, to pass out through the fibres (2′) of the pyramidal tract which connect the cortex with the nucleus of the sixth nerve (*er′*), and then to pass on through the commissural fibres (*c′*) to the part of the nucleus (*r*) of the opposite third nerve concerned in the action. According to this statement, therefore, in directing the eyes laterally, say to the right, both the right external rectus and the left internal rectus receive the impulse to action from the cortex of the left hemisphere, the impulses of the nucleus of the third nerve being received through the commissural fibres which connect it with the nucleus of the sixth nerve of the opposite side. So far, we have only spoken of the two recti muscles; but when these muscles are contracting so that the eyes are directed laterally, the muscles which rotate the head also become contracted in such a way that the head is turned in the same direction as the eyes, this movement being frequently observed when a man looks over his shoulder. Rotation of the head, say to the right, is produced mainly by contraction of the right inferior oblique muscle of the neck, although the left sterno-mastoid, and probably other muscles, coöperate in the movement, and these muscles also receive their voluntary impulses to action through commissural fibres which connect their nerve nuclei with the nucleus of the sixth nerve of the right side. According to this supposition, when a strong impulse is sent from the left cortex (C′) of the brain through the fibres (2′) which connect it with the nucleus (*er*) of the sixth nerve of the opposite side, these impulses will also pass through commissural fibres to the nuclei of the nerves which supply the internal rectus and sterno-cleido-mastoid muscles of the opposite side, and of the inferior oblique muscle of the neck of the same side; and the eyes and head will consequently be strongly rotated to the right, and away from the hemisphere from which the impulses originated. But this lateral deviation or conjugate deviation of the eyes, as it is called, occurs frequently in disease, and it is then associated with rotation of the head and neck to the same side as the eyes are directed. This position of the eyes and head is almost a constant accompaniment of convulsions of cerebral

origin, and, when the convulsions are unilateral and due to disease of the cortex of one hemisphere, the rotation always takes place towards the convulsed side and away from the seat of the lesion. Unilateral convulsions are often associated with a certain degree of hemiplegia, the convulsions being then limited to the paralyzed side; and when, under these circumstances, conjugate deviation of the eyes occurs, the rotation is always *towards* the paralyzed side. This, then, constitutes *spasmodic* lateral deviation of the head and eyes. But Graux has drawn attention to the fact that this lateral deviation is often of *paralytic* origin. Let us now suppose that the fibres (2′) which connect the left cortex (C′) and the right nucleus of the sixth (*er*) are suddenly interrupted, the cerebral impulses to the nucleus are arrested, the external rectus of the right eye becomes paralyzed, and that eye is rotated to the left. But the impulses through the commissural fibres which connect the nucleus of the right sixth, and those of the left internal rectus, and of the rotators of the head to the left must also be arrested, so that the latter muscles likewise become paralyzed; hence the left eye and the head become rotated to the left, the rotation now taking place *away* from the paralyzed side and *towards* the hemisphere of the brain in which the disease is situated. The rotation of the eyes in this direction has been facetiously described as an attempt on the part of the patient to inspect the cerebral lesion which is the cause of the paralysis. The rotation of the eyes, head, and neck is not now due to spasm of the muscles engaged in producing the action, but to paralysis of their antagonists. This symptom is usually associated with all sudden and severe attacks of hemiplegia; it is generally absent in the slighter forms of the attack, and in all cases in which the paralysis is more or less gradual in its onset. The phenomenon is also, as a rule, a very transitory symptom in hemiplegia, and usually disappears in from four days to a week. The rotation of the head generally disappears first, and then the deviation of the eyes improves; but it not unfrequently happens that a temporary squint may be observed during the progress of the rotation of the eyes towards recovery.

The reason of the temporary character of the paralytic form of conjugate deviation of the eyes and rotation of the head and neck—say towards the right—appears to be that although the nucleus of the left third (*r′*) usually receives its impulses to action through the commissural fibres which connect it with the nucleus of the right sixth nerve (*er*), and consequently from the cortex of the left hemisphere, yet channels of communication (1) still exist between the nucleus of the left third and the cortex of the right hemisphere. There is no congenital deficiency of the channels which connect the cortex of the right hemi-

sphere and the nucleus of the third nerve of the opposite side, nor indeed of the oblique commissural fibres which connect the latter with the nucleus of the right sixth nerve; and now that the more usual channels are interrupted by disease, impulses begin to pass through the less used channels. In a few days, then, the channel (1) between the right cortex and the nucleus of the left third nerve becomes patent, and some days later the commissural fibres (*c*) between the two nuclei become so far open as to convey impulses from the nucleus of the left third to that of the right sixth, so that the paralysis of the muscles supplied by these nerves disappears. A destroying lesion in the pons situated above the nucleus of origin of the sixth nerve, but below the upper crossing of the fibres of the pyramidal tract, causes a conjugate deviation, which is directed away from the side of the lesion and towards the paralyzed limbs. It has also been shown by Graux that whereas disease of one of the sixth nerves produces an internal squint of the eye on the side of the lesion, and no affection of the other eye, disease of the nucleus of origin of one of the sixth nerves produces a conjugate deviation of the eyes, the external rectus on the side of the lesion and the internal on the opposite side being thus more or less paralyzed. But the internal rectus is not completely paralyzed, although it does not act when the eye has to be directed to lateral objects, it contracts quite well in association with the internal rectus of the opposite eye when the eyes are converged on a near object in front. These facts prove that the internal rectus muscle is innervated by fibres issuing from, or at least passing near the nucleus of origin of the sixth nerve of the opposite side, as well as by fibres from the third nerve of the same side. As we have seen, conjugate deviation of the eyes is, as a rule, a transitory symptom in hemiplegia, but if a lesion in the pons interrupts the commissural fibres (*c*) so as to prevent impulses passing from one nucleus to another, a second lesion situated in any position which will interrupt the fibres of the pyramidal tract will then produce a paralytic conjugate deviation of the head and eyes which remains permanent.

(4) *Secondary Deviation of the Sound Eye.*

In paralysis of one of the ocular muscles, say of the external rectus of the right side, the eye is of course subject to internal squint. Now, if during recovery from this condition, when the conduction through the sixth nerve (6*n*) is still delayed, the eye of the sound side be closed and the patient be directed to look at an object with his right eye in such a way as to strain the external rectus muscle, this strain is

accompanied by a strong voluntary effort, but owing to the diminished conductivity of the nerve only a relatively small amount of the voluntary impulses will pass to the muscle. But the impulses generated by the strong voluntary effort will pass through the commissural fibres (c') to the nucleus of the left third nerve (r') in undiminished degree, so that the internal rectus of the left eye becomes strongly contracted. The energetic contraction of the internal rectus of the left eye induces a secondary squint in it, the extent of which is much in excess of that of the squint of the paralyzed side. But although this secondary deviation is more apparent in the case of paralysis of the ocular than in paralysis of other muscles, yet essentially the same phenomenon occurs in the extremities. If the common extensor muscle of the toes is partially paralyzed, a voluntary effort to extend the toes is followed by flexion of them. A simple movement like flexion at the elbow-joint is not caused by contraction of the flexors only, but by the predominance of their contractions over the contraction of the extensors simultaneously induced. During recovery from an attack of hemiplegia it often happens that when the patient makes an effort to flex the forearm the flexor muscles may be observed to contract, yet either no movement or movement in the opposite direction occurs, because the balance of the innervation to the antogonistic muscles is equal to, or the innervation to the extensors is in excess of, that to the flexors.

(5) *Disorders of the Associated Movements of the Extremities.*

We have seen that the movements of the limbs, and especially of the hand of one side, are largely independent of those of the other, and consequently that the spinal nuclei of the nerves which supply the limbs are not intimately connected by transverse commissural fibres. But in walking, the movement of the right leg is always associated with swinging of the left arm; and, conversely, that of the left leg with swinging of the right arm. It may be inferred, therefore, that the nuclei of the nerves of the upper (a a') and lower extremities (l l') are connected by oblique and crossed commissural fibres. In man the movements of the leg of one side are not very intimately associated with that of the arm of the opposite side, hence the commissural fibres, which connect their respective nerve nuclei, are represented by dotted lines (c^x). In quadrupeds, however, the crossed association between the movements of the anterior and posterior extremities of opposite sides is much more intimate than in man, and consequently the oblique commissural fibres are patent in a corresponding degree.

Let us now suppose that the fibres (4′ and 6′) which connect the cortex (C′) of the left hemisphere with the spinal nuclei (a, l) of the

right extremities are ruptured. Rupture of these fibres would produce hemiplegia in man; but in the dog only a certain amount of paresis results, inasmuch as the right hind limb receives impulses through the open commissural fibres which connect the spinal nuclei of its nerves with the nuclei of the nerves of the left anterior limb. The right anterior limb likewise becomes innervated through the commissural fibres which connect the nuclei of origin of its nerves with those of the nerves of the left posterior extremity. All the limbs of the dog, therefore, become innervated from one hemisphere when the other hemisphere is injured, so that, although disease of one hemisphere causes a certain amount of paresis, no true paralysis or hemiplegia results as in the case of man. This condition has often been induced by experimental lesions of one of the hemispheres in the dog, and it is always associated with conjugate deviation of the head and eyes, showing that both phenomena are induced by disease of the same mechanism. But although the dog does not manifest complete paralysis of the muscles of the side opposite the lesion—say the right side, the lesion being in the left hemisphere—yet, on standing, a slight degree of pressure on the left side pushes the animal over to the right, the vertebral column is arched with the convexity towards the right, showing a predominance of the action of the left erector-spinæ over their antagonists, and the eyes and head are rotated to the left, a position which indicates paresis of the muscles which produce rotation of them to the right. Under these circumstances, when the dog endeavors to advance he begins to move round his tail, a movement which has been called "*mouvement de manége*," and which is the equivalent of hemiplegia in man. It is, therefore, probable that some of the compulsory movements described as automatic kinesioneuroses really belong to the synkineses, as at present defined.

(6) *Disorders of the Associated Movements of Articulation.*

But when the muscles which are bilaterally associated in their action are small, and when minor nervous discharges only are requisite to throw them into action, the connection of the muscles of the two sides with one hemisphere may be brought into such habitual use that the connection with the other hemisphere, although still existing, is held practically in abeyance. The muscles concerned in executing the movements of articulation, for instance, are bilaterally associated; the necessary adjustments demand great delicacy of execution, but no great muscular exertion; the muscles engaged in executing the most delicate

of these adjustments are small, and consequently these muscles fulfil all the conditions just mentioned.

It is now a matter of almost daily observation that the muscular adjustments concerned in articulate speech are regulated from the left hemisphere; but it by no means follows that the regulation of all the functions performed by these muscles is similarly restricted. The contractions of the laryngeal muscles concerned in vocalization, for instance, are not necessarily interfered with, because the delicate adjustments required in articulate speech are abolished; hence complete loss of the power of articulate speech is perfectly compatible with entire absence of voluntary paralysis of any of the muscles engaged in articulation. It is not the power of producing voluntary contractions of these muscles which is lost, but the power of producing highly complex combinations of these contractions. If we suppose that v and v' are the spinal nuclei of the nerves ($v\ n$, $v\ n'$) which supply the muscles of articulation, the two nuclei are practically fused into one by transverse commissural fibres (c''); and consequently impulses which start from the left cortex (C'), and pass through the fibres (3') to the spinal nucleus (v) of the right side, readily reach the left nucleus (v') through the commissural fibres (c''). But as the muscles concerned in articulation act always bilaterally and symmetrically, the channels of communication between the spinal nuclei of their nerves and the cortex of one hemisphere are brought into habitual use; while the channels of communication between these nuclei and the opposite hemisphere become partially obliterated from disuse, and probably not thoroughly developed from the first. The channels of communication between the right cortex (C) and the nuclei v and v', for instance, are represented by the dotted line (3 3), and the commissural fibres which convey impulses from the left to the right nucleus by the dotted line (c''), in order to indicate that these channels are only partially open. Destruction of the communication (3') between the left cortex (C') and the right nucleus (v) is followed by loss of articulate speech, a condition which is called *aphasia*.

If the lesion destroy the portion of the cortex of the left hemisphere —the posterior part of the third frontal convolution—from which the fibres of communication spring, this condition is permanent, except perhaps in young people, in whom the corresponding part of the right hemisphere becomes educated and developed for the purpose. But if the lesion involve only the channel of communication (3') between the left cortex and the right nucleus, the loss of speech is only temporary. The corpus callosum consists of fibres ($c\ c$) which connect symmetrical parts of the two hemispheres; and the portion of it which connects

the third frontal convolution of the two sides is represented in Fig. 72 by the dotted line to show that, although the connection exists, it is partially closed through disuse. When, however, the communication through (3′) is interrupted, impulses generated in the third left frontal convolution make their way through the fibres of the corpus callosum to the corresponding part of the right hemisphere, and after a time through the dotted line (3) which connects the latter with the left nucleus, and, after another interval, through the partially open commissural fibres which connect the left (*v*) with the right nucleus (*v′*). so that the power of speech is gradually reacquired. A lesion, however, which destroys both the channel of communication (3′) between the third left frontal convolution and the spinal nuclei, and the fibres of the corpus callosum (*c c*, dotted line) connecting the right and left third frontal convolutions, will influence speech as powerfully and permanently as disease of the gray substance of the third left frontal convolution itself. Such a lesion effectually cuts off the third left frontal convolution, in which the higher mechanism which regulates the muscular adjustments concerned in articulation is organized, from the spinal nuclei; and the only means by which speech can be then restored is the organization of a new mechanism in the corresponding part of the right hemisphere, a method which must always be slow, and which can only take place, at least to any considerable extent, in the plastic tissues of young people.

CHAPTER VII.

GENERAL SYMPTOMATOLOGY (*continued*).

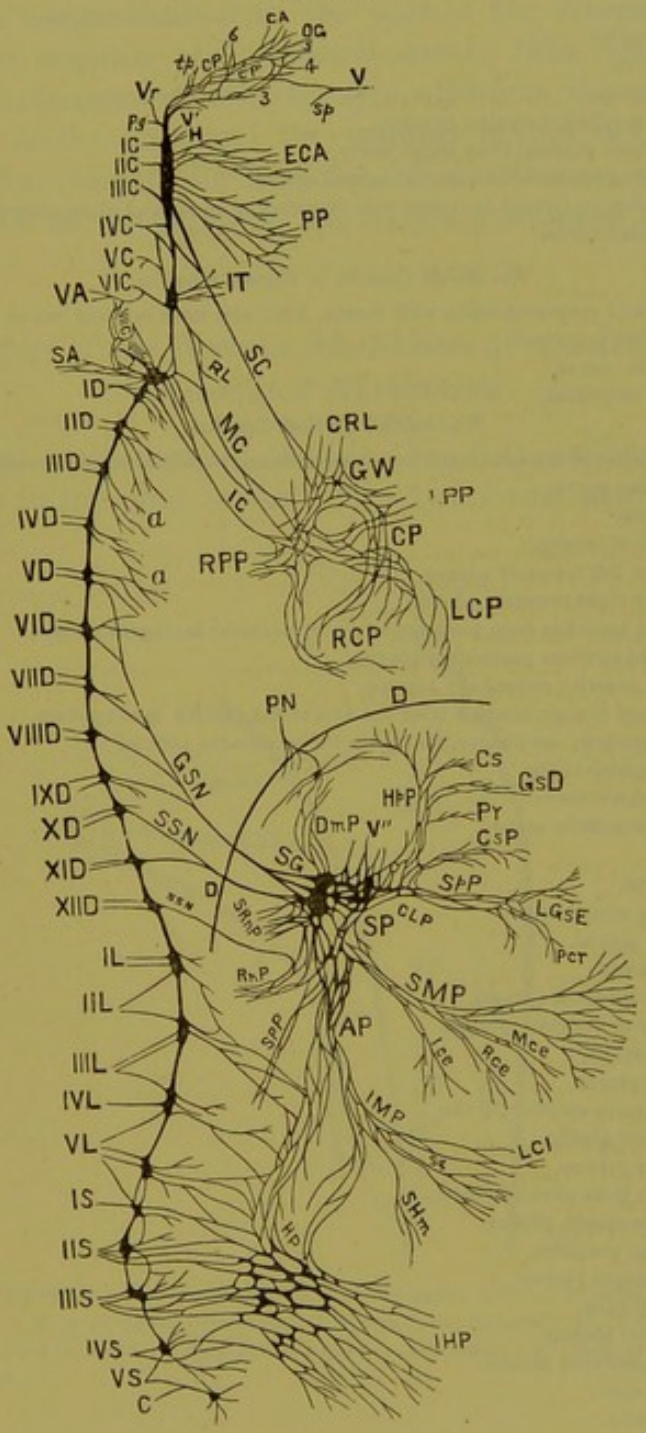
II. The Visceral Kinesioneuroses.

The motor affections of internal organs present many peculiarities in comparison with those of the organs of external relation. These peculiarities depend in great part upon the fact that the muscular apparatus of the internal organs is formed of unstriated muscular tissue, which differs from the striated muscle in its mode of contraction, and in several other respects. An unstriated muscular fibre does not respond to mechanical and electrical stimuli by a prompt contraction of short duration, but a long latent period precedes the contraction, which itself lasts for a considerable time, while relaxation takes place only in a slow and gradual manner. Another peculiarity of the contractions of unstriated muscles is the rhythmic and automatic manner in which they occur; these characteristics being well exemplified by the peristaltic action of the intestines and ureters. It is very probable that these movements depend upon the presence of local ganglia on the walls of the organs, although there are not wanting facts to show that the power of undergoing such contractions is an inherent property of the unstriated muscular fibres themselves. The functions of these intramural ganglia are regulated by means of accelerating and retarding nerve fibres from centres situated in the cerebro-spinal system, so that arrest of the contractions of unstriated muscles may be caused by a destructive lesion of the accelerator or an irritative lesion of the inhibitory fibres, while spasm may be caused by an irritative lesion of accelerator fibres or a destructive lesion of inhibitory fibres.

The movements of the internal organs are regulated chiefly by the ganglia and plexuses of the sympathetic system, but these are so inextricably connected with the cerebro-spinal system that it is impossible to draw any line of demarcation between the two nervous mechanisms.

The sympathetic system of nerves consists of a vertebral and prevertebral portion. The *vertebral* portion is composed of a series of ganglia, united by a longitudinal cord (Fig. 73, IC to C) which descends along each side of the vertebral column from the head to the coccyx. The *prevertebral* portion consists of the numerous ganglia

FIG. 73.



SUPERIOR CERVICAL GANGLION OF THE SYMPATHETIC: ITS CONNECTIONS AND BRANCHES.
(Reduced from FLOWER.)

IC to IVC, Branches of communication to four upper cervical nerves
PS, " " petrosal ganglion.
V r, " " ganglion of root of pneumogastric.
V', " " ganglion of trunk of pneumogastric.
H, " " hypoglossal nerve.
CP, Carotid plexus.
C'P, Cavernous plexus.
CA, Branches accompanying internal carotid artery.
OG, " to ophthalmic ganglion.

th, To tympanic branch of glosso-pharyngeal.
3, to third nerve.
4, to fourth nerve.
5, to fifth nerve.
6, to sixth nerve.
V, Vidian nerve to spheno-palatine ganglion.
S *p*, Large superficial petrosal from facial nerve.
EAC, Accompanying branches of external carotid artery.
PP, Pharyngeal plexus, formed by union with branches of vagus and glosso-pharyngeal nerves.
SG, Superior cardiac nerve.

The Middle Cervical, or Thyroid Ganglion.

IVC to VIC, Branches of communication with fourth, fifth, and sixth cervical nerves.
IT, Inferior thyroid branches.
MC, Middle cardiac nerve.
RL, To recurrent laryngeal.

The Inferior Cervical Ganglion.

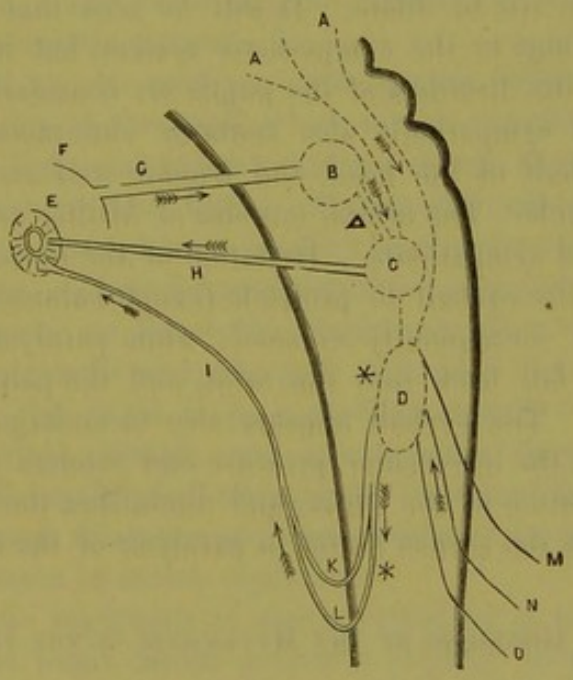
VIIC to VIIIC, Branches of communication with seventh and eighth cervical nerves.
IC, Inferior cardiac nerve.
CP, Cardiac plexus.
GW, Ganglion of Wrisberg.
LCP, Posterior, or left coronary plexus.
RCP, Anterior, or right coronary plexus.
CRL, Cardiac branches from pneumogastric or recurrent laryngeal nerves.
APP, To right anterior pulmonary plexus.
LPP, To left anterior pulmonary plexus.
ID to IID, Branches of communication from the first to the twelfth dorsal nerves.
a, *a*, To aorta, vertebræ, œsophagus, and posterior pulmonary plexus.
GSN, Great splanchnic nerve.
SSN, Small splanchnic nerve.
SSN′, Smallest splanchnic nerve.
D, Diaphragm.
PN, Phrenic nerve.
SP, Epigastric, or solar plexus.
CLP, Cœliac plexus.
Cs, Cystic plexus.
GSD, Gastro-duodenal plexus.
C *s* P, Gastric or coronary plexus.
Py, Pyloric plexus.
SpP, Splenic plexus.
LGsE, Left gastro-epiploic plexus.
Pcr, Pancreatic plexus.
HpP, Hepatic plexus.
V″, Branches from pneumogastric.
DmP, Diaphragmatic plexus.
SG, Semilunar ganglion.
SRaP, Suprarenal plexus.
RaP, Renal plexus.
SpP, Spermatic plexus.
SMP, Superior mesenteric plexus.
Mce, Middle colic.
Rce, Right colic.
Ice, Ileo-colic.
AP, Aortic plexus.
IMP, Inferior mesenteric plexus.
LCI, Left colic plexus.
Sz, Sigmoid plexus.
SHm, Superior hemorrhoidal plexus.
1L to VL, Branches of communication with the five lumbar nerves.
IS to VS, " " " five sacral nerves.
C, " " " coccygeal nerve.
HP, Hypogastric plexus.
IHP, Pelvic, or inferior hypogastric plexus, giving branches to all the pelvic viscera.

and plexuses of the head, chest, abdomen, and pelvis. The cerebro-spinal nerves communicate with the cord of the sympathetic at their exit from the cranium and vertebral canal. The fourth and sixth cranial nerves communicate with the sympathetic in the cavernous sinus, the olfactory in the nose, and the auditory in the meatus auditorius internus. The sympathetic branches of distribution accompany the arteries, so that all the organs of the body are supplied by sympathetic nerves.

1. *Disorders of the Nervous Mechanism of the Iris and other Associated Mechanisms.*

The nervous mechanism of the iris (Fig. 74) consists of a (1) contractor centre in the crus cerebri (C), which forms part of the nucleus

FIG. 74.



A A, psychical impression; B, centrum optici; C, oculo-motor centre; D, dilator centre (spinal); E, iris; G, optic nerve; H, oculo-motor (sphincter); I, sympathetic (dilator); K, L, anterior roots; M N O, posterior roots; △, seat of lesion causing reflex pupillary immobility; *, probable seat of lesion causing myosis. (After ERB.)

of the third nerve; (2) a cortical contractor centre (A), situated probably in the angular gyrus; (3) a dilator centre in the medulla (D); and (4) a cortical dilator centre (A), situated probably in the posterior part of the first frontal convolution. The cortical centres are connected with the contractor and dilator spinal centres by centrifugal fibres which descend in the pyramidal tract; the spinal contractor centre is con-

nected with the sphincter of the iris (E) by efferent fibres (H) which pass in the third nerve; and the dilator nucleus with the dilator muscle by means of efferent fibres which descend in the cervical region of the cord, emerge along with the anterior roots of the eighth cervical (K) and first dorsal (L) nerves, ascend in the cervical sympathetic, and ultimately find their way to the dilator fibres through the cavernous plexus, the lenticular nucleus, and the ciliary nerves. The contractor nucleus is connected with the surface by afferent fibres which pass from the retina (F) in the optic nerves and tracts (G) to the corpora quadrigemina (B), and then bend down to join the contractor nucleus in the floor of the aqueduct of Sylvius. The afferent and efferent fibres of the contractor nucleus form a reflex loop, so that when light falls on the back of the eye the iris contracts. The dilator nucleus appears to be connected with the surface of the body generally by afferent fibres (M N O), so that strong irritation of any part of the surface of the body causes the iris to dilate. It will be seen that only part of this mechanism belongs to the sympathetic system, but it will conduce to clearness if all the disorders of the pupils are considered in this place.

The cervical sympathetic also contains *vaso-motor fibres* for the corresponding half of the head, and *trophic and secretory* fibres for the salivary glands. The orbital muscles of Müller are also innervated from the cervical sympathetic. Irritation of the fibres supplying these muscles causes the eyeball to protrude (exophthalmos), and the palpebral aperture is consequently widened; while paralysis of them allows the eyeball to fall back into the orbit, and the palpebral aperture is then narrowed. The eyeball appears also to undergo trophic change, which increases the intraocular pressure and renders the cornea more convex in irritation of the fibres, and diminishes the intraocular pressure and renders the cornea flatter in paralysis of the fibres.

a. Disorders of the Movements of the Iris.

The disorders of the movements of the iris may be divided into the following varieties, namely, (1) contraction of the pupil, or myosis; (2) dilatation of the pupil, or mydriasis; (3) immobility of the pupil with normal size; (4) clonic spasm of the muscles of the iris; and (5) reflex disorders of the pupil.

(1) *Myosis.*—Three forms of myosis may be distinguished, namely, (*a*) *spastic* or *spasmodic myosis* caused by spasm of the sphincter; (*b*) *paralytic myosis*, caused by paralysis of the dilator fibres; and (*c*) *combined spasmodic* and *paralytic myosis*, caused by simultaneous spasm of the sphincter and paralysis of the dilator fibres. In the first

two of these varieties the pupil is in a medium degree of contraction and movable, and consequently they may be named the *medium* or *labile myoses*. In the last of the three the pupil is in the highest degree of contraction and immovable, and consequently it may be named *maximum* or *stabile myosis*.

(*a*) *Spastic myosis*, if in high degree, prevents the pupil contracting to light or during efforts at accommodation. The pupil does not dilate by shading the eyes, but a moderate degree of dilatation is caused by all excitants of the dilator centres or fibres, such as a strong sensory impression or emotional disturbance. A minimum dilatation is produced by mydriatics, and a maximum contraction by myotics.

(*b*) *Paralytic myosis* does not prevent the pupil contracting to the stimulus of light or during efforts at accommodation, but dilatation does not occur in irritation of the dilator centres or fibres. A medium degree of dilatation is produced by mydriatics and a maximum contraction by myotics. The pupil is more contracted, as a rule, in spasmodic than in paralytic myosis.

(*c*) *Combined spastic and paralytic myosis* causes a maximum degree of contraction of the pupil, which is also completely immovable to the stimulus of light and accommodation, as well as to those which act on the dilator centres and fibres. Mydriatics cause a medium degree of dilatation, but myotics have no effect on the size of the pupil.

(2) *Mydriasis.*—Three forms of mydriasis may also be distinguished, namely, (*a*) *spastic* or *spasmodic mydriasis*, (*b*) *paralytic mydriasis*, (*c*) *combined spasmodic* and *paralytic mydriasis*. In the spastic and paralytic form the pupil is in a medium degree of dilatation, and movable to certain stimuli, and consequently they may be named *medium* or *labile mydriasis;* while in the combined form it is in a condition of maximum dilatation and immovable, and consequently it may be called *maximum* or *stabile mydriasis*.

(*a*) *Spasmodic mydriasis* is characterized by a medium degree of dilatation of the pupil, which contracts slightly to light and during efforts at accommodation, but does not dilate on irritation of the dilator centre either through sensory nerves or psychical impressions. The pupil is difficult to contract by myotics, but a maximum dilatation is readily produced by mydriatics.

(*b*) *Paralytic mydriasis* is characterized by a medium degree of dilatation of the pupil, which fails to contract to the stimulus of light or during efforts at accommodation, but dilates further on sensory or psychical irritation of the dilator spinal centre. A maximum dilatation is readily produced by mydriatics, but a medium contraction alone is produced by myotics.

(*c*) *Combined spasmodic and paralytic mydriasis* is characterized by the pupil being in a maximum degree of dilatation, and completely reactionless to all kinds of stimuli. It is not possible to obtain a further dilatation of the pupil by mydriatics, but a medium degree of contraction is produced by myotics.

(3) *Complete immobility of the pupil with normal size* is caused by paralysis of both the dilator and contractor muscles of the iris. When the muscles of the iris are alone affected the condition is named (*a*) *iridoplegia*, but when all the internal muscles of the eye are paralyzed the condition is named (*b*) *ophthalmoplegia interna*.

(*a*) *Iridoplegia* is characterized by the pupil being midway between dilatation and contraction, and being immovable to every form of stimulus. Hutchinson states that the power of being acted upon by myotics and mydriatics is only completely lost when the substance of the iris itself is disorganized.

(*b*) *Ophthalmoplegia interna* is characterized by the pupil being midway between dilatation and contraction, and completely reactionless, while in addition the power of accommodation to near vision is lost.

(4) *Clonic spasm of the muscles of the iris* is named *hippus* or *chorea of the iris;* it consists of quickly alternating contractions and dilatations of the pupil, which depend probably upon a clonic spasm of the sphincter. It sometimes accompanies nystagmus, while at other times it is observed during the regressive period of paralysis of the third nerve.

(5) *Disorders of the reflex movements of the iris* may be divided into those of (*a*) the *irido-dilator*, and (*b*) the *irido-contractor* reflex arc.

(*a*) *Disorders of the irido-dilator reflex arc* may be subdivided into those caused by lesion (j) of the efferent and (jj) of the afferent portion of the reflex arc.

(j) *Disorder of the efferent portion of the reflex dilator arc* is met with in locomotor ataxia. A strong sensory irritation of any part of the body is followed in healthy persons by dilatation of the pupils, but this reaction fails to take place in many cases of locomotor ataxia, the reflex arc being interrupted by lesion of the efferent fibres of the dilator centre in their descending course through the cervical portion of the cord.

(jj) *Disorder of the Afferent Portion of the Reflex Dilator Arc.*— When there is complete anæsthesia of the lower part of the body from spinal disease, irritation of the anæsthetic area is not followed by dilatation of the pupils, but in cerebral anæsthesia, simulated anæsthesia, and probably, also, in hysterical anæsthesia, the reaction is not interfered

with. This reaction, therefore, may be found a useful test in distinguishing spinal anæsthesia from the other forms.

(*b*) *Disorders of the irido-contractor reflex arc* may be divided into those which are caused by disease of (j) the efferent fibres of the arc which pass in the third nerve, the short root of the lenticular ganglion, and the ciliary nerves; (jj) the afferent fibres of the arc, which pass from the retina through the optic nerves and tracts to the corpora quadrigemina; and (jjj) the loop which joins the corpora quadrigemina with the nucleus of the third nerve.

(j) *Disorder of the Efferent Portion of the Irido-contractor Reflex Arc.*—When the pupil fails to react to the stimulus of light from lesion of the efferent fibres of the reflex arc, the sphincter of the iris is found to be paralyzed, and the other symptoms which indicate partial or complete paralysis of the third nerve are generally present.

(jj) *Disorder of the Afferent Portion of the Irido-contractor Reflex Arc.*—In atrophy with blindness of the optic nerves the sphincters of the iris lose their tone, and, provided the sympathetic mechanism be free from disease, the pupils dilate, and they fail to contract to the stimulus of light, but still retain the power of contracting when the eyes are converged. In unilateral atrophy with blindness both pupils fail to contract when light is admitted to the affected side, but both contract readily when light is admitted to the sensitive eye, because the central cord of the contractor reflex arc is connected with its fellow of the opposite side by commissural fibres. In blindness from a lesion situated above the corpora quadrigemina, contraction of the pupil to light is retained, because the reflex arc is unaffected and consequently the presence or absence of reflex contraction of the pupil to light in cases of blindness is a valuable diagnostic sign in determining the localization of the lesion.

(jjj) *Reflex Immobility of the Pupil* (*the Argyll-Robertson Pupil*).—In this condition there is absence of reflex contraction of the pupil to light, while the associated contraction with accommodation is retained, and vision may be normal. The absence of any paralysis of the sphincter and of blindness, shows that the efferent and afferent fibres of the reflex arc are unaffected, and consequently the lesion must be situated in the fibres which connect the corpora quadrigemina with the nucleus of the third nerve (Fig. 75); the interruption taking place most probably near the descending root of the fifth nerve. This symptom is met with almost exclusively in locomotor ataxia and general páralysis of the insane.

b. Disorders of the Vaso-motor and Other Mechanisms of the Cervical Sympathetic and Cilio-spinal Region of the Spinal Cord.

The symptoms caused by disease of the cervical portion of the sympathetic differ according as the lesion is (1) an irritative or (2) a depressive one.

(1) *Irritative Phenomena.*—When the sympathetic centres in the medulla oblongata, or the efferent fibres which connect them with the periphery, are irritated, the pupil becomes dilated (spasmodic mydriasis); the palpebral aperture is increased in size; the eyeball becomes slightly protruded (exophthalmos); the temporal artery is contracted, and feels like a hard cord under the finger; the skin of the half of the face and the ear on that side is pale and cold to the touch; and the temperature in the external meatus, and probably in the cavities of the mouth and nose on that side, is lowered as compared with that on the opposite side. The phenomena of irritation of the sympathetic are somewhat transient, so that we do not possess any very accurate information with regard to the state of the secretions in such cases; but I have observed, in cases of cervical pachymeningitis with dilated pupils, that the face has often an oily appearance, as if the secretion of sweat were not so much increased in quantity as altered in quality. There are no very accurate observations with regard to the condition of the secretion of tears, saliva, or of that from the mucous membrane of the nose.

(2) *Depressive Phenomena.*—When the sympathetic centres in the medulla, or the fibres which connect them with the periphery, are paralyzed, the pupil is contracted (paralytic myosis); the intraocular tension is diminished and the cornea flattened; the eyeball is retracted or falls back into the orbit, and its pressure against the eyelids being thus lessened, the palpebral fissure becomes narrower; the temporal artery is dilated and tortuous; the skin of the face and side of the head on the affected side may be congested, especially at first; the temperature in the external meatus, the mouth, and the nostril is increased on the diseased as compared with corresponding parts on the healthy side; and the secretions of tears, saliva, and sweat, as well as that from the mucous membrane of the nose, are diminished on the side of the lesion; while the skin of the face looks, in long-standing cases, more flabby and wrinkled and older than that of the opposite side. When the disease has become chronic, the vessels on the affected side of the face may cease to be dilated; and when the patient exerts himself so that the cutaneous vessels of the body generally become dilated, the healthy side of the face becomes flushed and covered with

perspiration, while the affected half retains its normal appearance and remains free from moisture. The diseased side may also remain dry when the patient takes a hot bath. Bilateral paralysis of the sympathetic centres in the medulla oblongata, such as sometimes occurs in progressive muscular atrophy, is attended by a great flow of viscid saliva corresponding to the paralytic secretion obtained by experiments on animals.

Morbid Anatomy and Physiology.—Organic lesions of the sympathetic may be divided into those which implicate (1) the centres in the medulla oblongata or the conducting fibres in their descending course through the cervical portion of the spinal cord (Fig. 74, D to K, L); and (2) the fibres in their course through the rami communicantes of the eighth cervical and first dorsal nerves (Fig. 74, K, L) and the cervical sympathetic.

(1) *Lesions of the Medulla Oblongata and of the Cervical Portion of the Spinal Cord.*—Oculo-pupillary phenomena are not mentioned as being present in reported cases of lesions of the medulla oblongata, but a case is under my care just now in which there is decided paralytic myosis and diminution of the palpebral fissure of the right eye, and in which the accompanying symptoms would seem to indicate that the lesion is situated in the medulla. The patient had an apoplectic attack four or five weeks ago, and he is now suffering from left-sided hemiplegia and crossed hemianæsthesia, the right half of the face and the left half of the body being anæsthetic. In addition, the patient is quite unable to maintain the erect posture, although he is not completely paralyzed even on the left side of the body; and when he is placed on his legs between two attendants he staggers from side to side, and throws his legs about in the most irregular manner when he attempts to move. The staggering and incoördination are most probably caused by implication of the fibres coming from the inferior peduncles of the cerebellum. The crossed hemianæsthesia is likewise best explained by supposing that the lesion is situated in the medulla, causing simultaneous injury of the sensory conducting paths in their passage through the medulla, and of the ascending root of the fifth nerve. The excessive flow of saliva which is present in advanced cases of bulbar paralysis is most probably caused by paralysis of the sympathetic centres in the medulla oblongata. Dilatation of both pupils, along with paralysis of the four extremities, and a remarkably small and slow pulse (48 per minute), was observed by Rosenthal in a person who had been stabbed in the neck in the neighborhood of the sixth cervical vertebra. Oculo-pupillary phenomena, sometimes of the irritative and at other times of the depressive variety, have been frequently

observed in cervical spinal pachymeningitis and in fractures of the cervical spine; and the fact that these phenomena are also frequently present in locomotor ataxia would seem to indicate that the efferent fibres pass downwards through the posterior columns of the cord, while the absence of these symptoms from cases of progressive muscular atrophy and acute spinal atrophic paralysis shows that the efferent fibres are not likely to pass through the gray anterior horns.

(2) *Lesions of the Rami Communicantes and of the Cervical Sympathetic.*—The rami communicantes are sometimes implicated in cases of cervical pachymeningitis as they emerge from the spinal cord with the anterior roots of the eighth cervical and first dorsal nerves, and in the early stages of such cases the irritative phenomena may be present, but these soon give place to the depressive symptoms. The rami communicantes are divided in cases of rupture of the brachial plexus, and in these cases the paralytic oculo-pupillary, vaso-motor, and secretory phenomena are always well marked. These symptoms are also frequently met with in aortic aneurisms and mediastinal tumors from pressure on the inferior ganglion of the cervical sympathetic, in compression of the cervical sympathetic from goitre and other tumors, and in these cases the irritative symptoms may be present; but the paralytic phenomena are much more frequently met with. The depressive phenomena may also be caused by division of the cervical sympathetic from gunshot wounds. The irritative and depressive phenomena of the sympathetic are also met with in certain functional diseases, such as hemicrania.

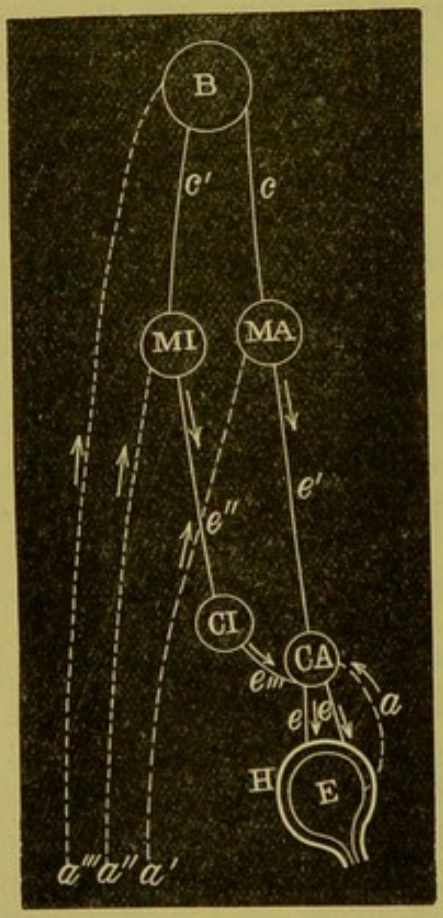
The explanation of these symptoms is to be found in the facts, established by experiments on animals, that the cilio-spinal region of the cord and the cervical sympathetic contains not only dilator fibres for the iris, but also vaso-motor fibres for the bloodvessels of the side of the face, and secretory fibres for the lachrymal, salivary, and nasal secretions. It would also appear that the cervical sympathetic contains trophic fibres for the eyeball, as, after section of it, Brown-Séquard found gradual atrophy of the eye to take place on the side operated upon. The diminution of the intraocular tension and the flattening of the cornea which occur in paralysis of the sympathetic fibres in man is probably caused by lesion of these trophic fibres. The cervical sympathetic also contains fibres which regulate the contractions of the muscles of Müller in the orbit; and when these fibres are irritated the muscles are maintained in a state of contraction, and the globe is protruded; and when they are paralyzed the muscles relax and allow the globe to fall back into the orbit, and the palpebral fissure is diminished.

2. *Disorders of the Nervous Mechanism of the Heart.*

a. The Cardiac Nervous Mechanism.

The nervous centres which preside over the movements of the heart consist of intracardiac ganglia, centres in the medulla oblongata, and centres in the cortex of the brain. The positions of these centres may be illustrated by the annexed diagram (Fig. 75). The cardiac muscle

Fig. 75.



Schema of the Action of the Cardiac Nervous Mechanism.

CA, intramural motor ganglia of the heart; CI, intramural inhibitory ganglia; MI and MA, centres in the medulla oblongata, the former being inhibitory, the latter motor; B, centre in cortex of brain; *a*, afferent fibre to intramural ganglion cell; *a'*, afferent-motor or excito-motor fibre; *a''*, afferent inhibitory or excito-inhibitory fibre; *a'''*, afferent fibres connecting periphery with cortex of brain; *e'*, effero-motor fibre; *e''* and *e'''*, effero-inhibitory fibres; *e*, *e*, efferent fibres of the reflex arc of the motor intramural ganglia; *c*, *c'*, fibres connecting the cortex of the cerebrum with the centres in the medulla oblongata. The arrows indicate the direction of the conduction.

is represented by H, and the endocardium by E; CA represents the intramural ganglia; MA and MI the motor accelerating and the motor inhibitory centres respectively; and B the cortical centre, which is

represented not only as a motor centre, but also as receiving sensory fibres from the heart and from the periphery of the body. These centres are connected with the periphery and with one another and the heart in various ways: *Firstly.* Afferent fibres (not represented in the diagram) connect the endocardium and the muscular fibres with a cardiac sensory cortical centre. These fibres pass from the heart through the cardiac plexuses, and the great cardiac nerve to the middle ganglion of the cervical sympathetic; but their subsequent course through the spinal cord and brain is not known. Irritation of these fibres in any part of their course causes cardiac pain or angina. *Secondly.* Afferent fibres (a') connect the endocardium with the intramural motor ganglia (CA), and from the latter issue fibres (e) which are distributed to the cardiac muscle (H). This constitutes a simple reflex mechanism. *Thirdly.* Afferent fibres (a'') connect the periphery of the body with the cardiac accelerator centre in the medulla, and irritation of these renders the action of the heart more frequent. These fibres are called excito-motor, but *affero-accelerating* fibres would best characterize their functions. *Fourthly.* Afferent fibres (a''') connect parts of the periphery with the cardio-inhibitory centre in the medulla, and irritation of these renders the action of the heart slower. These fibres are called *excito-inhibitory*, but *affero-inhibitory* would best characterize their functions. It is possible that each of the centres in the medulla is connected by afferent fibres with the endocardium and the muscular fibres of the heart, but these are not represented in the diagram. *Fifthly.* Afferent fibres (a'''') connect the surface of the body, either directly or indirectly, with the cortical motor centre (B). *Sixthly.* Intercentral fibres (e') connect the motor accelerating centre (MA) in the medulla and the intramural cardiac ganglia (CA); they convey efferent impulses which increase the activity of the heart, and are consequently called *accelerating fibres.* *Seventhly.* Intercentral fibres (e'' and e''') connect the inhibitory centre (MI) in the medulla and the intramural ganglia (CA); they carry impulses which arrest the action of the heart, and are therefore called *cardio-inhibitory* fibres. Various physiological facts seem to indicate that between the cardio-inhibitory centre in the medulla (MI) and the intramural motor ganglia (CA) intermediate ganglia (CI) are interposed, which, from their action, are called *intramural* cardio-inhibitory ganglia. *Eighthly.* Intercentral fibres (c and c') connect the cortex of the opposite hemisphere of the brain with the cardio-motor and cardio-inhibitory centres in the medulla. These fibres may respectively be called *centrifugo-motor* and *centrifugo-inhibitory* fibres.

b. Disorders of the Cardiac Nervous Mechanism.

(1) disorders of the sensory nerves of the heart (angina pectoris).

Angina pectoris occurs in paroxysms which are separated from one another by longer or shorter intervals. The attack begins by a sudden, shooting, tearing, or burning pain, which is felt at the lower part of the sternum and shoots over the left side of the chest and neck, or along the sternum and down the left or both arms. At the same time a feeling of oppression or constriction is felt across the chest, which is accompanied by a sense of suffocation and inability to breathe. During the paroxysm the arteries feel like cords, and the pulse is small and wiry, or feeble, irregular, and intermittent; the surface of the body is pale and cold; the face is pale, sunken, and covered with sweat; and the expression is one of great alarm and fear; while it has an unaccountable feeling of anxiety and fear of impending death. At the end of the attack the pulse becomes full and soft; the skin becomes warm, red, and covered with abundant perspiration. The paroxysm is generally of short duration, lasting only a few minutes; but sometimes the attack is made up of a series of paroxysms, each being followed by a remission or a complete intermission of the distressing symptoms. The paroxysms recur, as in epilepsy, at extremely variable intervals, and the course of the disease is always chronic. The spinous and transverse processes of the cervical and upper dorsal vertebræ and the region of the inferior angle of the scapula are sometimes tender to pressure, either during the attacks or continuously.

It would appear that the essential condition which underlies angina pectoris is loss of the balance which ought to be maintained between the propulsive powers of the heart and the resistance to be overcome, the conditions being altered in such a way that the muscular walls of the heart are subjected to strain. This state may result from all conditions which weaken the muscular power of the heart, such as the anæmia caused by calcification of the coronary arteries, and fatty degeneration of its muscle; and from all conditions which cause an obstruction to the onward flow of blood, such as aortic obstruction, and diminution of the calibre of the arterioles generally, either by disease of their walls or spasm of their muscular coats. It will, therefore, be apparent that the symptoms of angina will accompany many, probably most, of the organic disorders of the heart.

(2) disorders of the simple reflex cardiac mechanism.

The heart may be thrown into action after it has been removed from the chest by stimulating the endocardium, a fact which shows that the

afferent fibres connect this membrane with the intramural ganglia, which in their turn are connected by efferent fibres with the muscular fibres. Poisons which act on the muscular substance of the heart, as the salts of potassium, lactic acid and its salts, destroy this mechanism by rendering the muscle incapable of responding to a nervous stimulus. Such poisons arrest the action of the heart in *diastole*. Landois has shown that weak solutions of these agents, when injected into the endocardium of the frog, stimulate this reflex mechanism, and accelerate the action of the heart, while stronger solutions paralyze it. Irritation of this reflex mechanism in disease accelerates the action of the heart, while all depressive influences upon it render the action of the heart slow and feeble, and angina is found associated with the last state.

(3) DISORDERS OF THE CARDIO-INHIBITORY MECHANISM.

(*a*) *Disorders of the Inhibitory Fibres of the Vagus.*—The beat of the heart may be checked or stopped in diastole by stimulating the peripheral part of the divided vagus nerve. The inhibitory fibres (e'') of the vagus are paralyzed by curara, the action of the heart is then accelerated, and is not rendered slow by stimulation of the vagus. Electrical stimulation of the sinus venosus will, however, still inhibit the cardiac beats, and to account for this action the existence of intramural cardio-inhibitory ganglia are assumed. Nicotine stimulates the same fibres which curara paralyzes and consequently the action of these drugs is strictly antagonistic. But atropia paralyzes the intramural inhibitory mechanism, while physostigma stimulates, so that these drugs are also antagonistic in their action on the heart. But although atropia and nicotine, and curara and physostigma have opposite effects on the cardiac rhythm, yet they are not mutually antagonistic. Atropia and physostigma will check the action of nicotine and curara respectively, but the latter drugs have no effect in checking the action of the former. Muscarin and jaborandi produce standstill of the heart which may be removed by atropia, yet neither of the former drugs has any influence upon the action of the latter. From this it is inferred that muscarin and jaborandi stimulate the inhibitory fibres of the vagus, but do not affect the intramural cardio-inhibitory mechanism.

Irritation of the vagus is indicated by a full, hard, retarded pulse, increased force of the cardiac beats, disorders of phonation and deglutition, and sometimes temporary arrest of the heart's action. Paralysis of the vagus is indicated by increased rapidity of the pulse, which may beat as high as from 216 to 240 times in a minute. This form of

accelerated action of the heart is sometimes associated with attacks of bronchial asthma.

(*b*) *Disorders of the Cardio-inhibitory Centre in the Medulla.*—In acute and chronic diseases of the base of the brain the rhythm of the heart is greatly changed, and this is probably due to irritation or paralysis of the cardio-inhibitory centre in the medulla. In tubercular meningitis, for instance, the pulse, which is at first very slow, becomes very quick towards the terminal period of the disease. Great acceleration of the pulse is also met with in the various forms of bulbar paralysis, and in cases of locomotor ataxia with gastric crises and other symptoms indicative of disease of the medulla.

(*c*) *Disorders of the Reflex-inhibitory Mechanism.*—It would appear that powerful stimulation of any part of the body will produce reflex inhibition of the heart. Crushing of a frog's foot will, for instance, stop the cardiac beats, and in man the fainting which occurs during severe pain is caused by an inhibitory action on the heart. Injury of the intestines, however, appears to exercise a more powerful effect on the heart than that of any other part of the body. If the abdomen of a frog be laid bare and the intestines be sharply struck, the heart will stand still in diastole, and the same effect is produced by strong stimulation of the mesenteric nerves, but this action fails in both cases if the vagi are previously divided. Disease of the abdominal organs often gives rise, by reflex irritation of the vagi, to a kind of angina, which Landois has named *angina pectoris reflectoria*.

(4) DISORDERS OF THE EXCITO-MOTOR OR ACCELERATOR MECHANISM.

Stimulation of certain parts of the spinal cord, and of certain fibres of the sympathetic, causes acceleration of the heart's action, but section of these nerves does not render the action of the heart slower, and consequently the accelerator and inhibitory fibres are not to be regarded as antagonistic, this view being still further confirmed by the fact that simultaneous stimulations of these fibres do not neutralize each other. When the accelerating fibres are irritated by disease the symptoms are the same as are caused by stimulation of the simple reflex motor mechanism.

(5) ANGINA PECTORIS VASO-MOTORIA.

The symptoms of angina are sometimes caused by irritation of the vaso-motor nerves, but this condition will be considered with the angio-neuroses.

3. *Disorders of the Nervous Mechanism of Respiration.*

a. The Respiratory Nervous Mechanism.

The nervous mechanism of respiration consists of nerve centres and their connection with one another, with the muscles of respiration, and with the periphery of the body.

The respiratory nerve centres are situated in the spinal cord and medulla oblongata, and in the cortex of the brain. The spinal centres are the nuclei of origin of the thoracic, phrenic, and spinal accessory nerves, and to these may be added the nuclei of origin of the nasal branches of the facial nerve, and of some of the fibres of the superior laryngeal nerve. The respiratory centres in the medulla are situated beneath the fourth ventricle, at the nuclei of origin of the pneumogastric and spinal accessory nerves. This centre controls and regulates the spinal centres, inasmuch as destruction of it arrests all respiratory movements. The cortical motor respiratory centres appear to be situated on the internal surface of the hemisphere. The following are some of the conducting fibres of the respiratory mechanism: *Firstly.* Afferent fibres from the surface of the body generally influence the respiratory cortical centres, and consequently any sudden impression upon the surface of the body interferes with the respiratory rhythm. *Secondly.* Afferent fibres from certain parts of the body influence the respiratory centre in the medulla. Breathing is arrested in inspiration by cold suddenly applied to the surface of the body, and it is temporarily arrested in expiration by a sudden irritation of the mucous membrane of the nose, the irritation being conveyed through the branches of the fifth nerve. *Thirdly. Affero-accelerating* fibres to the respiratory centre are contained in the trunk of the vagus. If the vagi are divided respiration becomes much slower, fuller, and deeper than normal, but if the central end is stimulated the respiration is again quickened, and a strong stimulus arrests it in the position of deep inspiration. *Fourthly. Affero-inhibitory* fibres to the respiratory centre pass in the superior and inferior laryngeal nerves, and probably, also, in the splanchnic nerves, and powerful stimulation of these fibres produces respiratory arrest in expiration. *Fifthly.* Efferent fibres connect the cortical motor centres with the respiratory centres in the medulla, and probably also directly with the spinal centres. *Sixthly.* The respiratory centre in the medulla is connected with the spinal centres probably by cells as well as by fibres, and the former of these centres is probably only the upper expanded termination of a continuous gray column which forms the spinal nuclei. *Seventhly.* The spinal nuclei are connected with the muscles of respira-

tion by the thoracic, phrenic, and spinal accessory nerves, and by the nasal branches of the facial, and some of the fibres of the superior laryngeal nerve. *Eighthly.* It is probable that there is no localized sensory cortical centre for respiration, but the distress caused by obstruction to the breathing is a complex sensation caused by irritation of the afferent fibres of the respiratory muscles and various other afferent fibres.

The rhythmical action of the respiratory centre is probably automatic and not reflex in its nature, although its action is variously modified by afferent impulses. The exciting cause of the respiratory movements is the presence of a certain amount of oxygen and carbonic acid in the blood, respiration becoming stronger the less the quantity of oxygen and the more of carbonic acid it holds. The rhythm of respiration is, however, modified and regulated by impulses passing through the afferent inhibitory and accelerating fibres. Brener and Hering believe that distention of the lungs acts as an excitant to the inhibitory fibres, and thus induces an expiratory act, and that contraction of the lung excites accelerating fibres and thus initiates an inspiration. It is also probable that we must assume the existence of an inspiratory and an expiratory centre in the medulla.

b. Disorders of the Respiratory Nervous Mechanism.

(1) *Accelerated Breathing.*—When the blood is deficient in oxygen and charged with carbonic acid, the respiratory centre in the medulla is stimulated and the respiratory rhythm is *accelerated.* The breathing is also often accelerated in hysteria and other nervous disorders, and in febrile diseases.

(2) *Dyspnœa.*—When respiration is accompanied by a distressing consciousness of want of breath, arising from undue irritability of the nervous mechanism, as in hysteria, or from insufficient aëration of the blood, as in organic diseases of the heart, the accessory muscles of respiration are thrown into action, and the condition is called *dyspnœa.* When there is an obstruction to the passage of air to and from the lungs, the limits of distention and contraction are reached slowly and with effort, and consequently the dyspnœa is declared not so much by the frequency of the respiratory movements as by the increasing effort required to accomplish the act and by an alteration in the ratio of the various stages of the respiratory rhythm.

(3) *Apnœa.*—By blowing air into the lung or by forced voluntary breathing the blood becomes saturated with oxygen and poor in car-

bonic acid, and the respiratory movements are temporarily arrested. This condition is termed *apnœa*.

(4) *Asphyxia*.—When the deficiency of oxygen in the blood is very great the excitability of the respiratory motor centre in the medulla becomes destroyed, and the respiratory movements are arrested. This condition is termed *asphyxia*.

(5) *Cheyne-Stokes Respiration*.—In various cerebral, cardiac, and renal affections the breathing becomes intermittent. After a prolonged pause the respiratory rhythm becomes established, and the respiration gradually increases in depth and frequency up to a certain point, beyond which they become gradually shallower and slower until the pause, which may last from one-half to three-quarters of a minute. The duration of the whole cycle is usually from one to two minutes. This peculiar kind of breathing, with its gradually ascending and descending intensity and periodical pause, is named the Cheyne-Stokes respiration, after the observers who first described it. The usual explanation of this phenomenon is that when the excitability of the respiratory centre is greatly diminished the blood must become surcharged with carbonic acid in order to excite it, the increased inspiratory efforts thus induced diminish the venous state of the blood, and the respiration becomes less powerful, until it is finally arrested for a time until the blood becomes again surcharged with carbonic acid. Langendorff's experiments prove that arrest of the flow of blood to the medulla will induce this kind of breathing in animals, and it is, therefore, likely that an alteration in the nutritive quality of the blood, or a spasm of the arteries of the medulla, may help to cause it.

(6) *Asthma nervosum* consists of paroxysmal attacks of difficulty of breathing, which are caused by a spasmodic contraction of the muscular tissue of the bronchial tubes. The expectoration of frothy mucus which generally accompanies an attack would seem to indicate that the symptoms are also in part caused by an œdematous swelling of the mucous membrane of the smaller bronchi. Those who are subject to nasal catarrh will readily understand the suddenness with which such a swelling may appear, and the degree of obstruction and discomfort to which it may give rise. The asthmatic paroxysm may be excited by direct irritation of the trunk of the vagus, in other cases it is caused by reflex irritation of the sensory nerves of the lungs themselves, or of those of remote organs, such as the stomach, intestines, or uterus. An attack sometimes results from central irritation, and it is then generally associated with hysteria. In some cases an asthmatic attack is accompanied by a feeling of great oppression and great acceleration of the pulse, which may beat from 132 to 148 in the minute, and in these

cases it is probable that there is a simultaneous irritation of the pulmonary and paralysis of the cardio-inhibitory fibres of the vagus.

Sneezing, coughing, respiratory spasm, and respiratory paralysis will be considered hereafter.

4. *Disorders of the Nervous Mechanism of the Bladder and Rectum.*

a. The Nervous Mechanism of the Bladder and Rectum.

The walls of the bladder and rectum contain muscular fibres to expel their contents, while at the mouth of each there is a sphincter which is maintained in a state of tonic contraction, and thus prevents

Fig. 76.

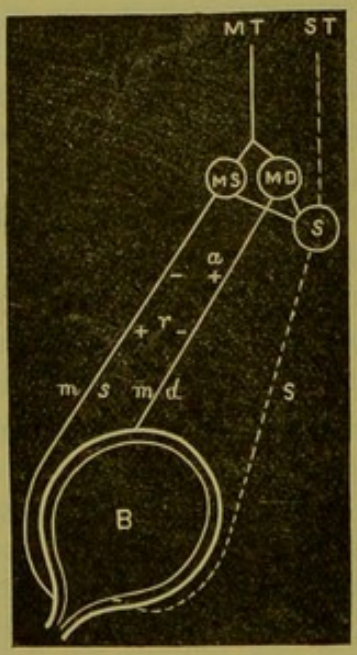


Diagram Showing the Probable Plan of the Centre for Micturition. (After Gowers.)

MT, Motor tract, ST, Sensory tract in the spinal cord; MS, Centre, and *m s*, Motor nerve for sphincter; MD, Centre, and *m d*, Motor nerve for detrusor; *s*, Afferent nerve from mucous membrane to S, sensory portion of centre; B, Bladder. At *r* the condition during rest is indicated, the sphincter centre in action, the detrusor centre not acting. At *a* the condition during action is indicated, the sphincter centre inhibited, the detrusor centre acting.

the continual escape of their contents. It is probable that the nervous arrangements for the regulation of both the urinary and rectal functions are the same, and it will consequently suffice if we describe the nervous mechanism of the bladder.

This nervous mechanism consists of nerve centres and their connections with one another and with the viscus. It is probable that two centres exist in the spinal cord—an automatic centre (Fig. 76, MS)

situated in the segments corresponding with the second, third, and fourth sacral nerves, and maintaining the tonic contraction of the sphincter, and a reflex centre (Fig. 76, MD) situated on a little higher level for the expulsion of the urine by inducing a contraction of the detrusor vesicæ. Another centre is situated in the cortex of the brain, and by its means the automatic and reflex lumbar centres are brought under voluntary control. The cortical centre is connected with the lumbar centres by means of centrifugal fibres (MT), and the lumbar automatic and reflex centres are connected with the bladder by efferent fibres, some of which (*m s*) connect the automatic centre (MS) with the sphincter and others (*m d*) connect the reflex centre with the detrusor. These centres are also connected with the periphery by means of afferent fibres, some of them being reflex and others sensory. The reflex afferent fibres (*s*) ascend from the mucous membrane of the bladder, to reach the lumbar centres through the posterior gray horns (S), while other afferent fibres ascend along the centripetal conducting paths (ST) to reach the cortex of the brain, and then to become connected with the cortical centre.

When the bladder is empty or only partially full the sphincter is maintained in a state of continuous contraction by the action of the automatic centre (MS). But when it becomes distended a strong impression is made upon the afferent nerves of the mucous membrane and impulses are conveyed to the lumbar centre and to the brain. Now the effect of afferent impulses upon an automatic centre is to inhibit its action, while afferent impulses conveyed to a reflex centre find vent along efferent channels. When, therefore, afferent impulses are conveyed to the lumbar centres, the action of the automatic centre is inhibited and the sphincter relaxes, while that of the reflex centre is increased and the detrusor vesicæ contracts. The action of the local automatic and reflex mechanisms is rendered more definite and certain when the afferent impulses reach the brain. A desire to urinate is excited; voluntary impulses are then conveyed by centrifugal channels to the inferior centres, the action of the automatic centre is either increased and that of the reflex centre inhibited so as to check the reflex tendency to urinate, or the action of the automatic centre is inhibited and that of the reflex increased, and urination is accomplished.

b. Disorders of the Vesical Nervous Mechanism.

The nervous mechanism of the bladder may be affected in various ways, but those disorders generally declare themselves either by incontinence or retention. Affections of the nervous mechanism of the

bladder may be divided into those caused by (*a*) spino-peripheral and (*b*) those caused by cerebro-spinal lesions.

(*a*) *Spino-peripheral Lesions.*—A destructive lesion of the automatic centre of the sphincter, or of its efferent fibres, gives rise to *paralytic* or *atonic* incontinence, while irritative lesions occasion *spasmodic* retention. A destroying lesion of the reflex centre causes *paralytic* or *atonic* retention, and irritative lesions of the reflex arc, generally caused by peripheral irritation of the afferent fibres, as in cystitis, give rise to *spasmodic* incontinence.

(*b*) *Cerebro-spinal Lesions.*—Destroying lesions of the cortical centre or of the centrifugal conducting paths arrest voluntary control over the bladder the urine is discharged at irregular intervals, and cannot be restrained: but if the spino-peripheral apparatus is free from disease the urine does not escape in a continuous stream. If the centripetal cerebral conducting paths are also affected, the patient is unconscious of the act of urination. Irritative lesions of the cerebro-spinal apparatus may give rise at one time to spasmodic incontinence, and at other times to spasmodic retention. The disturbances of the functions of the bladder met with in cases of hysteria and during epileptic attacks are probably caused by cerebral discharges along the cerebro-spinal conducting paths.

5. *Disorders of the Nervous Mechanism of the Genital Organs.*

a. The Nervous Mechanism of the Genital Organs.

Sexual Functions.—The sexual functions are governed by a cortical centre, a reflex centre situated in the upper part of the lumbar enlargement, and local automatic ganglia connected with the bloodvessels of the corpora cavernosa. It is not necessary to describe in detail the connections of these centres with one another and with the periphery. The lumbar reflex centre is stimulated to action by (*a*) irritation of the sensory nerves of the glans penis, and (*b*) discharge from the cortex of the brain with its associated emotional excitement. On stimulation of the reflex centre efferent impulses are conveyed along the *nervi erigentes*, which cause vascular dilatation and erection by inhibiting the action of the local automatic ganglia. A still more prolonged irritation of the lumbar centre produces ejaculation.

b. Disorders of the Nervous Mechanism of the Genital Organs.

The sexual functions may be disordered by (*a*) spino-peripheral, or (*b*) cerebro-spinal lesions.

(*a*) *Spino-peripheral Lesions.*—Destroying lesions of the lumbar centre or of the nervi erigentes cause impotence, while irritative lesions

of the reflex arc, generally stimulation of the afferent portion of the arc, cause erection without sexual desire, named *priapism*, or partial erections with strong sexual desire, named *satyriasis*. In the latter condition the cortical centres are also stimulated either directly from the original source of irritation or indirectly from the erections produced. This condition is met with in cases of locomotor ataxia.

(*b*) *Cerebro-spinal Lesions.*—Destroying lesions of the cortical centres or of the centrifugal conducting paths render the patient *impotent*, but erections and ejaculations may still occur so long as the reflex mechanism in the cord is intact. If the centripetal paths are interrupted the subject is insensible of an erection. The act of coition for instance, affords no pleasure to many hysterical women, and it is probable that in them there is a functional arrest of centripetal impulses to the cortex of the brain. Irritative lesions of the cortical centre or of the centrifugal conducting path may give rise to satyriasis, nymphomania, or to priapism. Satyriasis in the male, or nymphomania in the female, are not unfrequently observed in the insane, caused probably by irritation of the cortex; and irritation of centripetal fibres is also likely to give rise to the same condition. But although irritation of centrifugal channels occasions priapism, it is not likely to cause satyriasis. Lesions in the upper dorsal and lower cervical regions of the cord are often accompanied by priapism, doubtless caused by irritation of centrifugal conducting paths.

6. *Disorders of the Nervous Mechanisms of Other Viscera.*

It would occupy too much space to enter upon an analysis of the motor affections of the other viscera, such as the various segments of the intestines and the uterus with its appendages. Complicated disorders of these and other organs occur from lesions of the centres of innervation in the cortex of the brain, medulla oblongata, and spinal cord, as well as from lesions of the intramural ganglia, and the fibres which connect these different centres with one another and the periphery. These lesions give rise to atony or hypertony, spasm or paralysis, or want of motor coördination, similar in principle to the disorders of the nervous mechanisms we have just been considering.

III. Vascular Kinesioneuroses or Angioneuroses.

1. *The Vaso-motor Nervous Mechanism.*

The calibre of the bloodvessels throughout the body appears to be regulated by means of a nervous mechanism which is essentially similar to those which regulate the movements of the hollow viscera. The

centres consist of local ganglia in the walls of the arteries, spinal centres, vaso-motor centre in each lateral half of the medulla, and cortical motor centres which are situated in the motor area of the cortex. The vaso-motor centre in the medulla is probably only the upper expanded end of a column of gray matter which represents the spinal centres. The following are some of the conducting fibres of the vaso-motor mechanism: *Firstly.* Afferent fibres from the periphery of the body generally alter the arterial tonus. A medium degree of irritation of the sensory nerves causes an increase, but strong irritation causes great diminution of the vascular tone. *Secondly.* Afferent fibres which run in the vagus, and which, on being irritated, cause a diminution of the vascular tone, and these are consequently named *depressor fibres.* In the rabbit these fibres arise from the heart, and form a separate branch which runs alongside the carotid artery and the cervical sympathetic. *Thirdly.* Afferent fibres which run in the superior laryngeal nerve and the sympathetic, and excitation of which causes a marked increase of vascular tone, and consequently these fibres are named *pressor fibres.* *Fourthly.* Afferent fibres connect the muscular fibres of the middle coat of the arteries with the intramural ganglia, and excitation of these causes first contraction and then dilatation of local vascular areas. *Fifthly.* Centrifugal fibres connect the cortical centres of one hemisphere with the vaso-motor centre of the opposite half of the medulla. *Sixthly.* Efferent fibres connect the vaso-motor centre in the medulla with the spinal centres, but it is probable that the connection between these centres is in considerable part effected by means of gray matter. *Seventhly.* Efferent fibres connect the vaso-motor fibres in the medulla and the spinal vaso-motor centres with the intramural ganglia, these fibres passing outwards partly through the rami communicantes and the sympathetic, and partly through the anterior roots and the spinal nerves. These fibres are of two kinds: excitation of one set of fibres causes the arteries to contract, and these are consequently named *vaso-constrictor fibres;* but excitation of the other set causes the arteries to dilate, and these are consequently named *vaso-dilator fibres.* In many parts of the body the vaso-dilator fibres run in separate channels from the vaso-constrictor fibres. Stimulation of the chorda tympani, for example, causes dilatation of the bloodvessels of the submaxillary glands and of the anterior part of the tongue. In the extremities the nerve trunks contain both kinds of nerve fibres. *Eighthly.* Efferent fibres connect the local ganglia with the muscular fibres of the middle coat of the arteries.

The centre in the medulla oblongata is the general vaso-motor centre for all the arteries of the body, and by its action, which is probably of

a reflex nature, it maintains all the arteries of the body in a medium state of contraction, constituting *arterial tonus*.

2. *Disorders of the Vaso-motor Nervous Mechanism.*

Spasm of the vessels—angiospasm or vascular hypertony—may be caused by irritation of vaso-constrictor or paralysis of vaso-dilator fibres; while dilatation of vessels—angioparalysis or vascular atony—may be caused by paralysis of vaso-constrictor or irritation of vaso-dilator fibres. Both irritation and paralysis of vaso-motor fibres may be direct from lesion of the fibres or of the centres, or reflex from lesion of afferent fibres. Alterations of the calibre of the vessels of the surface of the body may be caused by (*a*) peripheral, (*b*) spinal, and (*c*) cerebral lesions; while special mention will be made (*d*) of the vaso-motor disorders of the viscera.

a. Peripheral Angioneuroses.

Reflex vascular disorders may be produced by lesions of the afferent fibres which pass from the vessels to the local ganglia, or of afferent fibres which pass to the ganglia situated higher up. These reflex disorders may consist of spasm or dilatation, although it is not known under what conditions the one or the other state is caused. Dilatation of the vessels of the conjunctiva, for example, is caused by neuralgia of the first division of the trigeminus, but it is not known whether the action is reflex or direct, or whether the vascular dilatation is caused by paralysis of vaso-motor constrictor or irritation of vaso-dilator fibres. Division of large nerve trunks is followed by redness and increased temperature of the parts supplied by the injured nerve, which is caused by paralysis of the vaso-constrictor nerves. But degeneration of the nerve fibres induces trophic changes in the affected extremity, which causes the material exchanges to be diminished and the circulation to be less active, and consequently less heat is generated, while more is radiated owing to the dilatation of the vessels, and the temperature falls below the normal. Paralysis of the vaso-motor nerves of an extremity occurs sometimes in the absence of any other symptom of nervous disease.

b. Spinal Angioneuroses.

Lesions of the vaso-motor centres in the medulla and spinal cord, or of their respective conducting paths, may increase or diminish the arterial tone either locally or generally. Injuries and diseases of the spinal cord which cause paraplegia are generally associated by a pri-

mary increase of temperature of the paralyzed limbs. In hemiparaplegia of spinal origin there is a primary increase of temperature in the paralyzed as compared with the non-paralyzed limb.

Injuries of the cervical portion of the cord near the medulla oblongata cause a remarkable elevation of the temperature of the body, and in these cases the temperature may continue to rise after the injury, and may even increase considerably after death. This remarkable rise of temperature probably depends upon sudden paralysis of the vasomotor system, or upon interference with the action of a heat-regulating centre in the medulla oblongata. In locomotor ataxia, again, which is a disease of the afferent or sensory portion of the spinal cord, the lower extremities are often cold from vascular spasm, and in some cases of this disease a local alteration of the vessels occurs which is so remarkable as to require special mention.

Tabetic Ecchymoses.—In the course of locomotor ataxia, patches of discolored skin are found scattered irregularly over the lower extremities and lower part of the trunk. These patches are at first of a bright red color, but soon become purple, and pass, like ordinary ecchymoses, through various shades of brown, green, and yellow, until they finally fade from the circumference to the centre, and disappear in from four to six days from the commencement. These ecchymoses appear suddenly towards the termination of severe paroxysms of lancinating pains and gastric crises. They are irregularly circular in form, and vary in size from a few lines to more than an inch in diameter. Several of these patches are found at the same time, as many as three or four of them being observed on each lower extremity.

The *local asphyxia* of Renaud belongs probably to the angioneuroses, but, as it often terminates in gangrene, its description will be found amongst the trophoneuroses.

c. Cerebral Angioneuroses.

Vaso-motor disorders are daily observed under the influence of various emotions, the most familiar of these being the blush of shame and the pallor of fear. Fainting is associated with pallor of the surface, and alternating conditions of pallor and redness are often observed in various neuroses, such as hysteria and epilepsy. At other times the vascular alteration, instead of being diffused, occurs in patches—what has been observed under the name of cerebral maculæ.

Taches cerebrales consist of red blotches and mottlings on the chest or abdomen of epileptics, and those suffering from Graves's disease and other neuroses. When the affected portion of skin is rubbed, or, in strongly marked cases, is merely touched by the finger, the surface soon

becomes suffused with bright red marks, which spread to some distance around the point touched, and persist for several minutes.

d. Visceral Angioneuroses.

The vaso-motor nerves of the thoracic viscera are derived from the inferior cervical and superior thoracic ganglia, and from the spinal cord by communicating branches from the third to the seventh dorsal vertebræ. The vaso-motor nerves of the abdominal viscera exist chiefly in the splanchnic nerves. Section of the splanchnic nerves occasions a great diminution of the arterial pressure from dilatation of the vessels and engorgement of the abdominal viscera. Irritation of the distal end causes contraction of the vessels and consequent elevation of the blood pressure. A part of the vaso-motor nerves of the abdominal viscera probably passes in the vagus. Experimental injury of the lumbar portion of the spinal cord in animals has been found to cause congestion and even extravasation of blood in the suprarenal capsules, and hemorrhagic foci have been found in these organs in cases of acute myelitis. Crushing of the pons and basal ganglia in animals has been found to cause congestion and ecchymoses in the lungs, pleura, kidneys, and mucous membrane of the stomach and bowels, and Eulenburg found intestinal hemorrhage after bruising the cortex of the occipital lobe of the brain. Congestion and extravasations of blood in the internal organs are not uncommon as complications of cerebral apoplexy. The various menstrual disorders which are so frequently associated with emotional disturbances are no doubt the result of functional disorders of the vaso-motor nerves, and the vicarious hemorrhages of the stomach, intestines, lungs, and other organs probably also depend upon disorder of vaso-motor innervation. Both quantitative and qualitative anomalies in the condition of the urine probably depend upon disorder of the vaso-motor nerves of the kidneys. Bernard found that injury of the upper part of the floor of the fourth ventricle causes polyuria and albuminuria, while injury of the lower part of the floor of the fourth ventricle causes temporary glycosuria. Injuries of the spinal cord, in the cervical and thoracic ganglia, or of the large nerve-trunks, such as the sciatic nerve, are also followed by glycosuria. If the pneumogastric nerve is divided in the neck, stimulation of the upper end is followed by dilatation of the vessels of the liver, and the appearance of sugar in the urine. The most reasonable explanation of these phenomena is that vaso-motor paralysis of the hepatic artery causes engorgement of the hepatic vessels, which gives rise to an increased production of

sugar. Certain forms of enlargement of the liver and spleen are probably caused by paralysis of vaso-motor nerves. Section of the efferent fibres of the semilunar and splenic plexuses in animals causes enlargement of the spleen, while irritation of these fibres reduces its size and renders the organ paler. Extirpation of the coeliac and mesenteric plexuses causes, besides other phenomena, congestion and enlargement of the liver, and it is probable that the congestion of this organ which takes place during attacks of migraine is of vaso-motor origin.

CHAPTER VIII.

GENERAL TREATMENT.

Nervous diseases must be treated according to the same general principles as all other diseases, and it is therefore unnecessary to enter upon a detailed description of treatment in this place. The treatment of nervous diseases may be divided into that which is directed: (1) to prevent disease; (2) to remove the exciting cause of the disease; (3) to remove the anatomical cause; and (4) to allay or remove serious symptoms.

1. PROPHYLACTIC TREATMENT.

Prophylactic treatment consists of a special application of hygienic rules to the cases of those who manifest inherited or acquired proclivities to diseases of the nervous system. The children of parents who have suffered from severe nervous diseases, like hysteria, epilepsy, or neuralgia, ought to be specially guarded against being subjected to severe mental strain and emotional excitement in youth, and especially during the period of sexual development. The children of such parents are generally quick in their perceptive faculties and are possessed even of great intellectual activity, and they ought not to be allowed to enter upon competitive examinations at schools except under the strictest precautions. Parents and teachers ought to pay great attention to such symptoms as headache, sleeplessness, horrible dreams and night startings, and loss of flesh and appetite, which are the more usual symptoms of an overstrained nervous system. Of these symptoms, sleeplessness is probably the most important as a danger signal, inasmuch as, on the one hand, it is generally caused, especially in the absence of pain, by exhaustion of the nervous system, and, on the other hand, it becomes a powerful cause of further exhaustion, because the nervous energies used up during the day fail to be restored at night. Plenty of muscular exercise, so long as it is thoroughly enjoyed and stops short of inducing fatigue, is the most powerful means we possess of fortifying the nervous system in young people. Those who inherit a predisposition to nervous disease also require an abundance of plain and nourishing diet, and a due exposure to sunlight and fresh air.

2. REMOVAL OF THE EXCITING CAUSE.

When a disease of the nervous system has been induced by unfavorable circumstances of climate, exposure to variations of temperature, or excessive fatigue, these conditions must, if possible, be corrected; and when the disease has been caused by wounds, contusions, or compression of nervous tissues, these causes must be removed, and the damage done to the tissues repaired as much as possible by surgical interference. If the disease is caused by a morbid poison like syphilis, malaria, gout, rheumatism, or the metallic poisons, the treatment must be directed to remove these poisons from the system or to neutralize their action.

3. REMOVAL OF THE ANATOMICAL CAUSE.

The nutrition of diseased nervous tissues may be favorably influenced by agents which act directly on the nervous tissues themselves, the connective tissues which surround the nervous tissues, the vaso-motor nerves and centres of the bloodvessels themselves, and the blood. The agents by which nutrition can be influenced may be divided into (*a*) internal and (*b*) external remedies.

(*a*) Internal Remedies.

Internal remedies produce their action after gaining admission into the circulation either by being absorbed through the mucous membranes, skin, or subcutaneous tissues after injection, or by direct injection into a vein. The following are some of the remedies of this class: *Strychnia* and the preparations of nux vomica increase the irritability of the gray substance of the spinal cord and diminish its specific resistance, and they are usefully administered when the irritability is depressed. Strychnia is a powerful remedy in atonic dyspepsia, constipation with flatulence, paralysis of the sphincters, nocturnal incontinence of urine, and sexual debility; but does not possess much value in the treatment of organic diseases of the spinal cord, being valueless in chronic spinal affections and positively injurious in all acute organic affections. *Conium* depresses the irritability of the spinal cord and of the motor nerves, and its use has been recommended by Dr. Crichton Browne in acute mania, and it has likewise been found useful in tetanus. *Calabar bean* lessens and ultimately destroys the irritability of the gray substance of the spinal cord, causing anæsthesia, loss of reflex excitability, and paralysis. It has been found useful in tetanus and in hemicrania.

Its alkaloid, *physostigma*, increases the irritability of the terminal fibres of the vagus, and kills by paralyzing the respiration. *Belladonna* and its alkaloid, *atropine*, increase the irritability of the gray substance of the spinal cord, and stimulate in a special manner the respiratory and vaso-motor centres, the cardiac acceleratory nerve or its centre, and the pupillary fibres of the sympathetic to the eyes. It paralyzes the motor nerves, first affecting those of the trunk, the terminations of the vagi both in the heart and lungs, the terminations of the secretory nerves of the salivary glands and of the sweat glands, the terminations of the inhibitory fibres of the splanchnics, and the terminations of the nerves supplying the iris. In large doses it depresses the functions of the afferent nerves. This drug has been found useful in checking profuse sweating, especially the night sweats of phthisis, and the secretion of milk. It is also useful in habitual constipation, whooping cough, incontinence of urine, and nocturnal emissions; and its use has been recommended by Brown-Séquard in chronic organic spinal diseases on the grounds that it contracts the arterioles of the cord. It is also useful in allaying pain, but is inferior to opium. *Ergot* is given in chronic spinal affections, being supposed to have the power of contracting the arterioles. *Opium* lessens the irritability of the sensory conducting paths and of the perceptive centres. Small doses first increase the irritability, but the primary increase is soon followed by a secondary stage of depression, and if a large dose be administered the first stage of increased irritability is so transitory that it may be overlooked. Opium may be administered in small doses so as to obtain the primary or stimulant action, and given in this way it is found useful in the treatment of nervous exhaustion caused by mental anxiety and overwork. It is, however, most frequently given in large doses, and is the most powerful remedy we possess for allaying pain and procuring sleep. *Hydrate of chloral* in small doses induces sleep, and in large doses profound coma. It paralyzes sensation and reflex action and causes arrest of respiration, or paralyzes the heart. Chloral and allied agents are given in relatively large doses with the view of depressing the irritability of the sensory mechanism and procuring sleep. Chloral has been found useful in the treatment of tetanus and of poisoning by strychnia, and as a sedative and hypnotic in cases in which the irritability of the nervous mechanism is increased. *Bromide of potassium* and its allies depress the irritability of the gray matter of the brain and spinal cord, and in large doses diminish sensibility and reflex excitability. They are very valuable remedies in epilepsy and various other spasmodic affections. Preparations of *zinc*, *arsenic*, and *phosphorus* appear to exercise a favorable influence on the nutrition of the

nervous system, and they have consequently been named "nervine tonics." These agents have been found useful in cases of neurasthenia, epilepsy, chorea, hysteria, and various spasmodic affections. *Silver salts* also belong to the same class of agents, and they have been employed in the treatment of epilepsy and tabes dorsalis. *Iron*, by improving the quality of the blood, is a most useful remedy in various nervous affections, and large doses of the carbonate are particularly efficacious in neuralgic affections. *Quinine* is a specific against all nervous affections which result from malaria, but it is also useful in the treatment of neuralgia, and especially in neuralgia of the supra-orbital branch of the fifth nerve. *Milk, whey, and grape "cures,"* and courses of *mineral waters*, act beneficially in many nervous diseases by exercising a favorable influence on general nutrition. *Iodide of potassium* has probably no special action on the nervous tissues themselves, but its well-known action in quickening the absorption of inflammatory effusions renders it an invaluable agent in the management of many nervous diseases, and it is an indispensable agent in the treatment of syphilitic nervous diseases. *Mercurial preparations* are also useful in the treatment of inflammatory diseases, more especially when the membranes of the brain or spinal cord or the sheaths of nerves are affected. Mercurial treatment is, however, chiefly directed against syphilitic nervous diseases.

(*b*) External Remedies.

Cold steadily applied lowers the irritability and retards the conductivity of the nervous tissues, and consequently it is used as a remedy in the acute stage of all inflammatory affections of the nervous system. Chapman's vaso-motor therapeutics is founded on the assumption that cold applied to the spine lowers the irritability of the vaso-motor, along with the other centres of the spinal cord, and thus causes dilatation of the bloodvessels whose nervous supply is derived from the portion of the cord over which the cold is applied. Chapman's ice-bag is the best method of applying cold to the spine, and very convenient bags are made for applying ice to the head or any other part of the body.

Warmth increases the irritability and accelerates the conductivity of the nervous tissues, and when combined with moisture it relaxes the tissues, and thus exercises a soothing influence in painful and spasmodic affections. Warmth may be applied by means of hot-water fomentations, poultices, Priessnitz's compresses, hot sand-bags, and India-rubber bags filled with hot water.

Cold baths tend directly to lower the irritability of the nervous tis-

sues and to depress the nervous functions, and, when their use is attended with benefit, it is due to their indirect action, or to the reaction of the tissues. This reaction is aided by the presence of salt in the water, and consequently the sea bath is less depressing than the ordinary bath. The cold bath is a powerful therapeutic agent in neurasthenia, hysteria, and various other functional affections, and it may occasionally be found useful in chronic organic affections. *Shower, douche, and sponge baths* produce powerful nervous stimulation by the impact of water on the body, in addition to the effects of the cold immersion. Shower and sponge baths are useful in the treatment of functional nervous affections; while the douche is employed for its local effects on paralyzed parts, and in rousing a patient from drunkenness or opium poisoning. *Warm baths* act as a sedative to the cutaneous nerves, and through them to the nervous system generally.

If the temperature be indifferent (90° to 97° F.) the bath acts mainly as a nervous sedative, while warm and hot baths (97° to 108° F.) produce great vascular excitement and act as powerful nervous stimulants. If the symptoms of irritation preponderate the indifferent baths should be selected, and if the symptoms of depression are prominent the warm and hot baths are to be preferred. *Brine, vapor, hot-sand, hot-air, gaseous,* and *chalybeate baths* all act on the same principle as hot baths, and are to be used when powerful stimulation is required. The *needle bath* is also a powerful stimulant and is used when the symptoms of depression predominate.

Mud baths act like warm baths, but are much less exciting than warm-water spring baths, and they are consequently adapted for the treatment of spinal irritation and of the various forms of spasmodic paralysis.

The *Turkish bath* combines many of the properties of the hot and cold bath. The great heat to which the body is subjected tends doubtless to induce debility, but the subsequent free application of cold water excites the cutaneous nerves and braces the system, so that the tonic effects of the cold bath are secured. The systematic kneading to which all the muscles of the body are subjected promotes their own nutrition and that of the nerves which supply them, and thus the effects of the bath are combined with those which result from careful and regulated exercise, and systematic gymnastics. The Turkish bath is, indeed, a powerful therapeutic agent, and has a wide range of usefulness in the treatment of various nervous affections.

Experience has shown that certain climates and regions exercise a favorable influence on various nervous diseases, especially on those of functional origin. Sea air has a very invigorating effect on the system,

and it is well adapted for the cure of nervous exhaustion from overwork in those who are otherwise strong and healthy. Feeble and irritable people, however, obtain greater benefit from residence in a mountainous district. Mountain exercise has a very enlivening effect upon the nervous system, and the higher and drier the district the more marked are the tonic effects. The Engadine and Davös Platz are favorite mountain residences, and the Scottish mountains, the English lake district, and Ilkley, in Yorkshire, are also favorite places of resort, although they have neither the elevation nor the dry atmosphere of the Swiss mountains.

Bloodletting and counter-irritation are as useful in the treatment of inflammatory affections of the nervous system as in those of other organs, but as they present nothing special in their action or mode of application when employed in the treatment of nervous diseases, they do not require further consideration at present. The counter-irritants usually employed are cutaneous faradization, sinapisms, vesicants, issues, moxæ, and the actual cautery. *External frictions* are carefully employed in various nervous affections. Frictions with soothing liniments, warm oil, and ointments containing opium or belladonna, are used to allay pain in neuralgic and other painful affections. Frictions with spirituous liniments, either alone or combined with other stimulants, such as ammonia and camphor, are used in the treatment of various forms of paralysis.

Seclusion of patients from their friends is of great value in the treatment, not only of the graver psychoses, but also of many cases of hysteria, neurasthenia, melancholia, and other functional affections. With reference to the treatment of neurasthenia, Dr. S. Weir Mitchell says: "Once separate the patient from the moral and physical surroundings which have become part of her life of sickness, and you will have made a change which will be in itself beneficial, and will enormously aid in the treatment which is to follow." One of the most frequent questions which the physician has to decide in the treatment of nervous diseases is whether a patient is to take *exercise* or *rest*. This question is readily decided in most cases of organic disease, the general rule being that physiological rest should be enjoined in all acute, and a certain amount of graduated exercise in chronic affections. In the minor degrees of neurasthenia and melancholia a moderate degree of exercise may be prescribed, but it should be always short of fatigue, and in aggravated cases the most absolute rest should be enjoined, while the place of exercise is to some extent supplied by massage.

Massage is a general term which includes methodical rubbing, strok-

ing, kneading, and clapping the surface of feeble and paralyzed parts—a method which is often successful in the treatment of organic and functional paralyses. Massage has been extensively used in the Manchester Royal Infirmary by my colleague, Dr. Morgan, and with very gratifying results. The directions for carrying out the process as practised by Dr. Morgan are briefly the following:

1. Pinch the surface of the skin from below upwards, with the view of stimulating the cutaneous nerves. Suppose the lower extremities to be paralyzed, the skin of the foot is first drawn up into successive folds and lightly pinched. The skin over the whole of the leg, and finally that of the thigh is gone over in the same way.

2. Give passive movements to the joints, first moving each of them separately and then all together.

3. Shampoo the limb well. This is done by lubricating it with some bland oil, which is well rubbed into the surface by the tips of the fingers passing from below upwards, the spaces between the muscular groups being specially selected for rubbing.

4. Work the muscles well with the fingers. Each muscle, or group of muscles, is grasped between the ball of the thumb and the fingers, and well kneaded and rolled. During this process the limb must be placed in a position to approximate the ends of the muscles operated upon, and thus relax them to the utmost.

5. Slap the muscles well with the ulnar border of the hand.

6. Grasp the lower portion of the limb between the hands, and then draw them slowly and firmly upwards. This movement should be repeated some twelve or fifteen times. In this manner the veins are emptied and the circulation quickened.

The *Weir Mitchell treatment*, which has been carried out so successfully by Dr. Playfair, of London, in the treatment of neurasthenia, is a combination of seclusion, rest, massage, and hyperfeeding. With regard to the carrying out of this treatment Dr. Playfair remarks: "The removal of the patient from her home surroundings, and her complete isolation in lodgings with only a nurse in attendance, is a matter of paramount importance. This is a point on which I am most anxious to lay stress, since it is the great crisis to the patient and her friends, and constant appeals are made to modify this, which I look upon as an absolute *sine quâ non*. I attribute much of the success which I have been fortunate enough to obtain in my cases, to a rigid adherence to this rule." Great care must be exercised in the selection of a suitable nurse, and if the case is not going on favorably much advantage is often obtained by a change of nurse. The patient is ordered to bed for from six weeks to two months, and during the greater part of

this period such actions as sewing, reading, and writing are strictly forbidden. In aggravated cases the patient is not allowed even to turn over in bed without aid. She is fed by the nurse, and even the calls of nature are obeyed in the recumbent posture. With regard to the use of massage and feeding, Dr. Playfair says: "Massage consists in a systematic and thorough kneading and movement of the whole muscular system for about three hours daily, the result of which at first is to produce great fatigue, and subsequently a pleasant sense of lassitude. Subsidiary to this is the use of the faradic current for about ten to twenty minutes, twice daily, by which all the muscles are thrown into strong contraction, and the cutaneous circulation is rendered excessively active. The two combined produce a large amount of muscular waste, which is supplied by excessive feeding, and in consequence of the increased assimilation and improved nutrition we have the enormous gain in weight and size which one sees in these cases, it being quite a common thing for a patient to put on from one to two stones in weight in the course of five to six weeks. The feeding at regular intervals constitutes a large part of the nurse's work. At first from three to five ounces of milk are given every few hours, and the first few days the patient is kept on an exclusively milk diet. By this means dyspeptic symptoms are relieved, and the patient is prepared for the assimilation of other food. This is added by degrees, *pari passu* with the production of muscular waste by massage, which is commenced on the third or fourth day. By about the tenth day the patient is shampooed for an hour and a half twice daily, and by this time she is able to take an amount of food that would appear almost preposterous did not one find by experience how perfectly it is assimilated and how rapidly flesh is put on. It is the usual thing for patients to take, when full diet is reached, in addition to two quarts of milk daily, three full meals, viz., breakfast, consisting of a plate of porridge and cream, fish or bacon, toast and tea, coffee and cocoa; a luncheon, at 1 P. M., of fish, cutlets or joints, and a sweet, such as stewed fruit and cream, or a milky pudding; dinner at 7 P. M., consisting of soup, fish, joints, and sweets, and in addition a cup of raw meat soup at 7 A. M. and 11 P. M. It is really very rare to find the slightest inconvenience from this enormous dietary. Should there be an occasional attack of dyspepsia, it is at once relieved by keeping the patient for four and twenty hours on milk alone."

The results obtained by means of this treatment have been so striking and remarkable that not the slightest detail in the method of carrying it out is unimportant. After the first few days of treatment a considerable quantity of soup made from raw beef is added to the milk diet.

The following is Dr. Mitchell's formula for this soup: "Take one pound of raw fillet of beef, chop it finely, and place it in a bottle with a pint of water and five drops of hydrochloric acid. Stand the mixture in ice all night, and in the morning set the bottle in a pan of water at 110° F., and keep it two hours at this temperature. It is then to be thrown on a stout cloth, and strained until the remaining mass is nearly dry. The filtrate is given in two or three doses in the course of twenty-four hours. If the raw taste prove very objectionable, the beef to be used may be quickly roasted on one side, and then the process is completed in the manner above described. The soup thus made is for the most part raw, but has also the flavor of cooked meat."

Swedish gymnastics are a mere modification of ordinary gymnastics, the object being to aid the recovery of paralyzed or rather paretic parts by bringing the muscles into methodical exercise. The patient is instructed to make systematic voluntary efforts to move the paralyzed muscles, and when a certain degree of voluntary movement is attained the effect is increased by the opposition of a practised assistant. If, for example, the flexors of the forearm are to be exercised, the forearm is first extended, and the patient endeavors to flex it whilst the assistant opposes flexion with more or less force. In the case of associated movements it is often necessary to aid the contraction of one group of muscles so as to overcome the contraction of their antagonists, which are also excited to action during the voluntary effort to contract the paralyzed group. The assistant must then aid the paralyzed muscles by passively extending the healthy antagonist muscles. The action of the healthy antagonist muscles can be weakened, not only by the hands of an assistant, but also by fixing elastic bands or spiral wires to the limb so as to aid the action of the paralyzed groups. In the Swedish system great attention is paid to the development and strengthening of the extensors of the body.

Ordinary gymnastic exercises are also useful in the treatment of various nervous affections. By their use the nutrition of the muscular system is improved, the heart propels the blood more efficiently through the organism, the blood itself becomes of better quality, and the nervous system participates in the general improvement.

Mechanical Vibrations.—Some years ago, Dr. Mortimer Granville was led to try the effects of rapidly tapping the skin over the fifth nerve in a case of facial neuralgia by means of a Bennett's percussion hammer, using the pleximeter as a shield. The results obtained were so encouraging that he gradually extended the application of mechanical vibration as a therapeutic remedy to various other nervous diseases. With the view of better regulating the number and strength of the

blows which can be delivered in a given time, Dr. Granville had an instrument constructed which he names "percuteur." The newest and most efficient percuteur is worked by electricity, but it is important to remember that no electricity passes through the patient; the electricity is converted into mechanical motion, and its only use is to enable the performer to deliver a regulated number of blows on any desired part, just as was done more roughly by means of Bennett's percussion hammer. Dr. Granville believes that by means of mechanical vibrations he can relieve a considerable number of cases of neuralgia; that much amendment occurs by its use in the early stages of locomotor ataxia and lateral sclerosis; and that by it he can even arouse torpid nerve centres to action, and consequently that it is of great use in the treatment of neurasthenia.

Electricity in the forms of the galvanic and induced currents is one of the most useful remedies we possess in the treatment of the diseases of the nervous system. Only the briefest possible account can here be given of the methods of application and uses of electricity in the treatment of disease, and the student is referred for further information to books specially devoted to this subject, the valuable work of Dr. De Watteville on "Medical Electricity" being particularly recommended to the English reader.

In selecting the position upon which the electrodes are to be placed, two methods of application have been adopted, viz., the *direction* and *polar* methods. In the *direction* method one rheophore is placed over the plexus and the other over the trunk of the nerve. When the positive pole or anode is centrally placed the current is called a *descending* current, and when the cathode occupies that position the current is called an *ascending* current. In the *polar* method one rheophore is placed on an indifferent part of the body, such as the sternum, and the other is placed over the nerve or muscle which it is desired to stimulate. It does not appear that there is any essential difference in principle between these two methods. When the cathode is used as the exciting pole in the polar method, the same kind and degree of contraction is obtained as when the cathode is peripherally placed in the direction method; and, conversely, when the anode is used as the exciting pole in the polar method, the reactions obtained are the same as those produced when the anode is peripherally placed in the direction method. In practice, therefore, it appears to be indifferent whether the polar or direction method be employed, but those positions should be chosen which will best insure that the tissue or organ which it is desired to influence shall be thoroughly permeated by the current. With reference to the size of the electrodes, the general rule is that, when a

purely local effect on a nerve or muscle is wanted for diagnostic purposes, the electrodes should be small; but when an alterative effect on deeply seated organs is required large electrodes, thoroughly moistened, should be employed. The current, instead of being passed through the skin, may be sent directly to a nerve or muscle which it is desired to stimulate by means of needles introduced into its substance. This method, which is only of limited application in therapeutics, is called *electric acupuncture*.

Galvanization.—There are three methods of applying the galvanic current, according as it is desired to obtain its sedative, alterative, or stimulant action. According to the first method the rheophores are maintained immovable on the skin, or the affected extremities are placed in tepid salt water, with which the conducting wires of the battery are in contact. This is the *stabile* method of application. In the second or *labile* method of application the cathode is made to glide over the skin in the direction of the nerves and muscles to be acted on. The third or *interrupted* method is based upon the fact that the constant current acts as a stimulus to both nerves and muscles at the moment of making and breaking contact. In using this method one pole is held immovable on one point, whilst the other is made repeatedly to touch the part to be stimulated. The current may also be interrupted by means of a commutator. A still more powerful effect is produced by suddenly reversing the direction of the current by means of the commutator—a descending current, for instance, being suddenly changed for an ascending current. This method has been called by Remak, who introduced the practice, *voltaic alternatives*.

Faradization.—When it is desired to limit the current to the skin the rheophores must be used dry, and the skin should also be dusted with toilet powder or some other absorbent powder so as to diminish its conductivity. The electrode used should be metallic, either in the form of a cylinder, disk, or wire brush, and the useful method of application is what is known as the *electric hand*. A moist rheophore is applied over the sternum or other indifferent part of the body, and the other is held in one hand of the operator while he passes the back of the disengaged hand over the part which it is desired to excite. When the deeper tissues are to be reached through the skin the rheophores should consist of well-moistened sponges in cylinders, or metallic disks covered with wet leather, and the skin itself should be thoroughly moistened with a mixture of warm water and salt. In order to electrize a muscle completely the rheophores should be applied over its fleshy body, and they should either cover its whole surface or be applied in succession to all points of its surface. When the body of the muscle is thick a

strong current should be used; otherwise the superficial layers alone are excited. The easiest way to apply the current to a muscle is to hold the handles of the rheophores in the right hand, one between the thumb and index finger, and the other between the middle and ring fingers. The two electrodes are then moved up and down over the whole surface of the muscle so as to secure a thorough contraction of every part of it. The faradic current is passed through a nerve in order to obtain contraction of the muscle or group of muscles which it supplies. The polar method is adopted for this purpose, one electrode being placed over the sternum or some indifferent part of the body, and the other over the course of the nerve at the motor points, as indicated in Ziemssen's diagrams.

Central galvanization aims at bringing the whole of the central nervous system under the influence of the current. The cathode is held immovable over the epigastrium, while the anode is passed over the forehead and top of the head, along the inner border of the sterno-mastoid, over the course of the pneumogastric and sympathetic in the neck, over the nape of the neck, and along the whole length of the spine.

General faradization aims at bringing the whole muscular system under the influence of the current. One pole, usually the negative, is placed on some insensitive part of the body or fixed to a copper plate upon which the soles of the feet are placed, while the other is either sponged over the whole body or kept stationary at certain points.

Galvano-faradization is obtained by joining the induction coil and the galvanic battery in one circuit. The negative pole of the one is connected by means of a wire with the positive of the other, while the electrodes are attached to the two extreme poles. I have had repeated opportunities for observing the excellent results which are obtained by this method in the practice of my colleague, Dr. Morgan, who was the first to devise this method. Dr. De Watteville, who appears to have adopted this method independently of Dr. Morgan, speaks very highly of its power as a therapeutic agent.

Uses of Electricity.—The faradic current is a powerful stimulant to both nerves and muscles, and when a simple stimulant effect is alone desired this current is, as a rule, more applicable than the galvanic current. This current may, therefore, be used as a stimulant either to act on the skin directly in cases of anæsthesia, or to act indirectly on remote organs in a reflex manner; or it may be used as a powerful neuro-muscular stimulant in the various forms of paralysis. The galvanic current acts as a stimulant to nerves and muscles both on making

and breaking contact, and, in addition, produces a profound alteration of nutrition during the time the current is interruptedly passing through an organ. The latter action has been called by Remak its catalytic action, and it is to it probably that the constant current owes the many advantages it possesses over the faradic current in the treatment of many of the diseases of the nervous system. The stimulant action of the constant current is, however, very important both as a means of diagnosis and in the treatment of paralysis. We have seen that while paralyzed muscles manifest the reaction of degeneration, they are more sensitive to the action of the galvanic than of the faradic current, and in these cases the constant current should be selected to stimulate the nutrition of the affected muscles. With this exception, however, the faradic is a more powerful agent in the direct treatment of paralyzed muscles than the constant current. But the catalytic action of the constant current renders it an exceedingly valuable agent in modifying the nutrition of the nerves and trunks of the central organs of the nervous system, and it may even be used in the treatment of cases where the irritability of portions of the nervous system is increased, and to which, therefore, the faradic current is wholly inapplicable. As examples of the numerous applications of the constant current may be mentioned its employment for the relief of pain in neuralgia and the assuaging of various forms of spasm, not to speak of its numerous applications, both locally and generally, in the treatment of chronic diseases of the brain, spinal cord, special senses, and viscera.

4. TO ALLAY OR REMOVE SERIOUS SYMPTOMS.

The fourth indication of treatment is to allay or remove serious symptoms, and of these the distressing symptom of *pain* is by far the most prominent and important. Opium is the most powerful remedy we possess for the alleviation of pain, and it acts most effectually when administered in the form of subcutaneous injection of morphia. Bromide of potassium, chloride of ammonium, chloral hydrate, croton chloral, atropia, quinine, and various other agents may at times be used in preference to opium in cases of neuralgia. The constant current is also a valuable agent for assuaging pain, and when it succeeds it should be preferred to all other remedies, inasmuch as its use is unattended by any evil after-consequences. Cold, continuously applied, has been used as a palliative for the removal of pain in neuralgia, while at other times the local application of warmth affords greater relief, acting, probably, by relaxing the tissues in which the nerve fibres are embedded. *Con-*

tinuous pressure over a nerve is often useful in neuralgia and in cases of motor spasm, the action being produced by arrest of the conduction through the nerve. Ointments and liniments of aconite, veratrine, opium, or atropine are useful as local applications for the removal of pain.

Surgical operations have been successfully undertaken for the removal of pain or spasm. These operations are performed with the view of arresting the conduction of nerves in cases of neuralgia or in local spasms. The operations are *neurotomy*, in which the nerve is simply divided; *neurectomy*, in which a portion of it is dissected out; and *nerve-stretching*, in which the nerve is powerfully stretched. The conditions under which each of these operations ought to be undertaken, will be mentioned in another part of this work.

SPECIAL PATHOLOGY OF THE NERVOUS SYSTEM.

CHAPTER I.

GENERAL DISEASES OF THE PERIPHERAL NERVES.

1. HYPERÆMIA OR CONGESTION OF THE NERVES.

Congestion of the nerves is caused by exposure to great cold, as has been proved by S. Weir Mitchell, who found that exposed nerves which had been frozen became congested and swollen on thawing.

Symptoms.—The symptoms caused by freezing of nerves were first studied by Waller, who froze the ulnar nerve by placing the elbow in a freezing solution. The first symptoms are pain, anæsthesia, paralysis, and increase of temperature in the region of distribution of the nerve. In the thawed portion a very painful sensation is felt which spreads backwards to the brachial plexus, and may even produce vertigo and faintness. The symptoms which follow the thawing of the nerve are hyperæsthesia, numbness, formication, and partial loss of power, but no elevation of temperature in the area of distribution of the affected nerve.

2. INFLAMMATION OF NERVES—NEURITIS AND PERINEURITIS.

Etiology.—Inflammation of peripheral nerves is caused by wounds, contusions, rupture, laceration, or sudden compression of the nerve, or by exposure to cold and wet. Neuritis may also be caused by extension of inflammation from the surrounding tissues and organs. The most notable diseases which set up neuritis are caries of bones, inflamed tendinous sheaths, acute and chronic inflammation of joints, abscesses, and malignant growths. It is also frequently developed after acute diseases such as the acute exanthemata, typhoid fever, and diphtheria, and after chronic diseases like rheumatism and syphilis. Inflammation of nerves is also met with in lepra anæsthetica, herpes zoster, and vari-

ous other cutaneous affections, and it may appear in the absence of any recognizable cause, being then called *idiopathic neuritis.*

Symptoms.—The symptoms of neuritis differ according as the inflammation is (*a*) *acute* or (*b*) *chronic.*

(*a*) *Acute neuritis* comes on soon after exposure of the nerve to one or other of the causes of the disease. It is ushered in by a well-marked feeling of chilliness, or by an actual rigor, which is accompanied by headache, sleeplessness, and smart fever. In inflammation of a mixed or sensory nerve the patient experiences a severe and almost intolerable pain in the region of the affected nerve, and which often radiates into the areas of other branches of the same plexus, or into more remote nerve territories. The pain is deep-seated, tearing, boring, or burning, and it is described as being *almost continuous*, although there may be remissions and paroxysmal exacerbations, the latter being specially apt to occur at night. Every movement of the limb augments the sufferings of the patient, and pressure over the affected nerve causes intense pain, which radiates from it in all directions. The tract of the inflamed nerve is sometimes indicated by a red line like that which occurs in inflammation of a superficial lymphatic vessel. The skin over the whole area of distribution of the nerve is at first extremely sensitive to the slightest contact, and the patient complains of numbness and formication of this area, but the hyperæsthesia soon gives place to anæsthesia. In the early stage of the disease muscular spasms and twitchings may be present, but these are soon replaced by paralysis of the muscles supplied by the nerve. The degree of paralysis varies according to the severity of the inflammation, but in all aggravated cases the muscles undergo rapid atrophy, and manifest "the reaction of degeneration." In excitable patients an attack of acute neuritis may be attended by slight delirium, and an emotional condition is sometimes induced which may be mistaken for an attack of hysteria.

(*b*) *Chronic neuritis* occurs either as a sequel of an acute attack, or it arises insidiously with obscure symptoms, which gradually or suddenly attain great intensity. Pain is the earliest and most constant symptom; it varies considerably in character and intensity, being sometimes dull and tensive, at other times of a lancinating, tearing character, and radiating towards the periphery. The pain is continuous, although it is frequently interrupted by paroxysmal exacerbations, which generally occur at night and prevent sleep. It is increased by every kind of exertion and movement, and by everything which excites the activity of the heart. In the first stages of the disease the patient complains of numbness and of formication, and of unpleasant pricking sensations when the skin is touched or struck, but in the later stages of

the disease anæsthesia is established, which may vary in degree from a slight blunting of sensibility up to complete loss of all forms of sensibility.

Symptoms of motor irritation, such as tension, sudden contractions, cramps, or persistent contractures of the muscles, are present in the early stage of the disease, but these are succeeded by paralytic phenomena. The muscular spasms are sometimes direct and sometimes reflex. Reflex spasms are met with in the facial muscles when the fifth nerve is affected, and the sphincter of the iris and ciliary muscle may be the subjects of spasm when the ophthalmic branch is implicated; and even in neuritis of mixed nerves reflex spasms are sometimes so violent that, for example, the finger-nails may become buried in the skin of the palm from spasm of the flexors.

The affected nerve is swollen, and when it occupies a superficial position it can be felt as a continuously thick cord. In other cases the thickening occurs at certain intervals, and then fusiform or moniliform swellings may be felt in the course of the nerve—a condition which has been named *neuritis nodosa*. The nerve is always sensitive to pressure, and compression of the swollen portions gives rise to eccentric pains and formication.

The electrical reactions of the affected nerves and muscles may remain normal or be even increased in slight cases; but in rheumatic and syphilitic cases, and, indeed, in all aggravated forms of the disease, the muscles undergo atrophy and manifest the "reaction of degeneration."

Trophic disorders of the skin and nails and swelling and stiffness of the joints are frequently observed in chronic neuritis; hysterical or epileptic convulsions and tetanus frequently result from neuritis.

3. ATROPHY OF NERVES.

Atrophy of nerves may, as we have already seen, be *congenital* in those cases where portions of the body are incompletely formed, *idiopathic* when there is a simple wasting of the nerve fibres, as occurs in atrophy of the optic nerves in tabes dorsalis, and *secondary* when the nerve fibres are separated from their trophic centres.

Symptoms.—It may be laid down as a general rule that whenever atrophy of a nerve exists its function is lowered or lost. Secondary atrophy of motor nerves may be distinguished by electrical examination, and primary atrophy of the optic nerves may be recognized by an ophthalmoscopic examination.

4. HYPERTROPHY OF NERVES.

Hypertrophy of nerves is a mere anatomical curiosity, and is not known to be connected with any definite symptoms.

5. NEUROMATA.

Etiology.—Some individuals appear to be predisposed to the formation of neuromata, and phthisical and scrofulous persons seem to be particularly liable to their formation. Isolated neuromata are more common in women than in men, while multiple neuromata are almost exclusively met with in men. They occur at all ages, and are often congenital.

The best known of the exciting causes are blows, intermittent pressure, penetration and retention of foreign bodies, and various injuries. Neuromata are also frequently found in the cicatrices formed after nerves have been divided or injured, and swellings of the ends of the nerves are often met with in the stumps of amputated limbs. Chronic neuritis, syphilis, lepra, and elephantiasis may lead to the formation of tumors in nerves, and in a large number of cases no definite cause can be traced.

Symptoms.—The symptoms of neuromata are variable, many of them being quite painless, and others causing intense and persistent suffering. Isolated neuromata give rise to severe and incurable neuralgia, and they have consequently been called *tubercula dolorosa*. The pain may be tearing, lancinating, aching boring, or burning; it is almost always remittent or completely intermittent, but when the paroxysm comes on the pain gradually increases in intensity, and radiates from certain points towards the periphery. The pain is increased by cold and damp weather, by pressure, or the slightest movement of the affected limb, and in women frequently by the return of the menses or by pregnancy. It may often be made to disappear temporarily by firm pressure on the nerve above the tumor. The pain is generally more severe in small tumors of cutaneous branches than in larger tumors of deeper nerve-trunks. In addition to pain, feelings of numbness and formication, and sensations of heat or cold, are often felt in the area of distribution of the affected nerve. In some cases anæsthesia may be present, and appears not infrequently in the form of *anæsthesia dolorosa*.

Motor disorders are rare, but sometimes occur in the form of tremors, spasms, and contractures, and these may ultimately give place to complete paralysis. The course of the disease is very variable. True neuromata may remain stationary for many years without causing any

serious symptoms, and multiple neuromata are sometimes so free from pain that they are only discovered accidentally during life or at the autopsy. In other cases active symptoms, present for a time, may remit and ultimately cease, and in a few cases the tumor disappears. In other cases the tumor gives rise to persistent paralysis, and tumors of the cauda equina cause anæsthesia of the lower extremities of variable distribution, paraplegia, and various trophic disorders. In other cases the symptoms are such as to give rise to the greatest suffering, and to cause sleeplessness, cachexia, and even death from exhaustion.

Treatment.—The treatment must be directed to remove the cause, and surgical interference is frequently necessary in cases of injury. At other times the treatment must be directed against articular rheumatism, inflammation of tendons, syphilis, and other morbid conditions.

In *simple congestion* the steady application of ice along the track of the nerve, elevation of the part, and absolute rest suffice to arrest the disease.

In *acute neuritis* energetic antiphlogistic treatment must be adopted, consisting of local depletion, application of ice, purgatives, favorable position of the parts, and absolute rest. Large doses of quinine may prove useful, and large doses of morphia or atropia are necessary to allay pain.

In *subacute* and *chronic neuritis* cold may be applied in the first stages, but depressing treatment must soon be changed for counter-irritation by means of the faradic brush, painting with iodine, or blistering. In very chronic and obstinate cases recourse may be had to energetic counter-irritants, such as the moxa and the actual cautery. Hot baths, such as those of Wilbad, Gastein, Teplitz, and Wiesbaden, as well as mud and strong saline baths, have been found useful in the treatment of chronic neuritis. The galvanic current is a most effectual remedy, and the best method of applying it appears to be the steady application of the anode for a few minutes each day, over the affected spot. In severe cases the part must be kept at absolute rest in an appropriate position, and the patient should be warned against taking active exercise, or exposing himself to cold and wet. When all active symptoms have subsided the return of motion to paralyzed muscles may be promoted by massage and the faradic current.

In congenital atrophy no treatment is of any avail, and in idiopathic atrophy a simple delay in the progress of the disease is all that can be expected from treatment. Secondary atrophy may be arrested, and

even complete recovery may take place, provided the primary disease can be removed.

The only successful treatment of neuromata is afforded by the extirpation or the destruction of the tumor. Extirpation succeeds best when the growth can be removed whilst the nerve is left intact. When extirpation is impracticable the tumor may be destroyed by caustics or electrolysis, but these methods are not very successful. When the tumor cannot be removed, palliative treatment to alleviate the sufferings of the patient must be adopted.

CHAPTER II.

DISORDERS OF COMMON SENSATION AND OF SPECIAL SENSE.

I. ANÆSTHESIA AND ANALGESIA IN THE REGION OF DISTRIBUTION OF INDIVIDUAL NERVES AND PLEXUSES.

When a purely sensory or mixed nerve is divided a certain degree of anæsthesia is generally produced in the cutaneous area supplied by it, but the extent and degree of this anæsthesia are very variable, and by no means correspond with the anatomical distribution of the sensory filaments of the nerve. Arloing and Tripier found that section of one of the four nerve branches by which the digit of a dog or cat is supplied caused no recognizable diminution of its cutaneous sensibility, section of two branches caused only slight general diminution of sensibility, section of three branches caused decided diminution of sensibility, but complete anæsthesia of any part of the digit was only established when the four branches were divided. They also found that on the fifth digit of the dog, which is supplied from branches from the ulnar and radial nerves, partial anæsthesia only is caused by section of one or other of these nerves, while on the fifth digit of the cat, which is supplied by branches of the ulnar nerve alone, section of the nerve caused complete anæsthesia. It would seem, therefore, that the skin is not mapped out into functionally independent territories corresponding to the anatomical distribution of the filaments of the sensory nerves. This functional intercommunication between neighboring nerve territories is explained by the free anastomosis which is known to take place, first, between branches of different nerves of considerable size, and second, between those nerves in the terminal networks described by Jacobowitsch and Beale. Arloing and Tripier have described a third method by means of which nerve fibres from one nerve district may be distributed to another territory. After division of one of the branches of the median nerve of the dog or cat, for instance, they found that the animal gave manifest evidence of pain when the peripheral portion of the divided branch was pinched, and even division of the whole nerve did not entirely prevent these manifestations of sensibility. The peripheral portion of the divided branch was cut out and examined microscopically a few days after section, and it was found

that although by far the greater number of fibres were degenerated, yet a few of them had remained healthy. A portion of the proximal end of the divided nerve was also examined microscopically, and a few degenerated fibres were discovered amongst a large number of healthy ones. From these observations the authors concluded that the few fibres which remained healthy in the peripheral portion of the nerve still retained their connection with their trophic centres, while the few which were degenerated in the proximal portion were severed from their centres. As the trophic centres of these fibres must have been situated in the spinal ganglia or in the gray matter of the cord, they must have reached the divided branch from the peripheral side of the point of section, and have been pursuing a centripetal course, and the authors justly assume that these fibres conferred recurrent sensibility upon the peripheral part of the nerve. They also believe that these fibres, after ascending a short distance in the nerve, are distributed to the skin, and thus take an important part in supplying one district with nerve fibres from neighboring nerve territories. It has also been suggested by Jacobi that the spinal ganglia form a centre in which an anastomosis takes place between fibres from different cutaneous nerve territories. But whether these explanations be satisfactory or not, it is at least certain that, after division of a principal nerve trunk, complete anæsthesia is either absent or is limited to a very small portion of skin, while the area of partial anæsthesia is much less than that of the anatomical distribution of the sensory branches of the nerve. A detailed description of the distribution of anæsthesia in diseases and injuries of individual nerves will be given when we come to discuss paralysis of peripheral nerves, because the presence of anæsthesia serves to distinguish peripheral paralysis from the atrophic paralysis of spinal origin. The distribution of anæsthesia in disease of individual nerves may also be gathered in a general way from the distribution of neuralgia of the same nerves, and it is therefore unnecessary to pursue the subject further at present.

II. HYPERÆSTHESIA AND HYPERALGESIA IN THE REGION OF DISTRIBUTION OF INDIVIDUAL NERVES AND PLEXUSES.

The most characteristic feature of the hyperæsthesia and hyperalgesia caused by disease of peripheral nerves is that they are apt, by radiating to neighboring nerve territories, to extend over a much wider area than the anatomical distribution of the sensory branches of the affected nerve. The distribution of hyperæsthesia in disease of individual nerves may, however, be gathered from the distribution of the pain in neuralgia without further description.

III. NEURALGIA IN THE REGION OF DISTRIBUTION OF INDIVIDUAL NERVES AND PLEXUSES.

1. Neuralgia of the Fifth Nerve (Trigeminal Neuralgia).

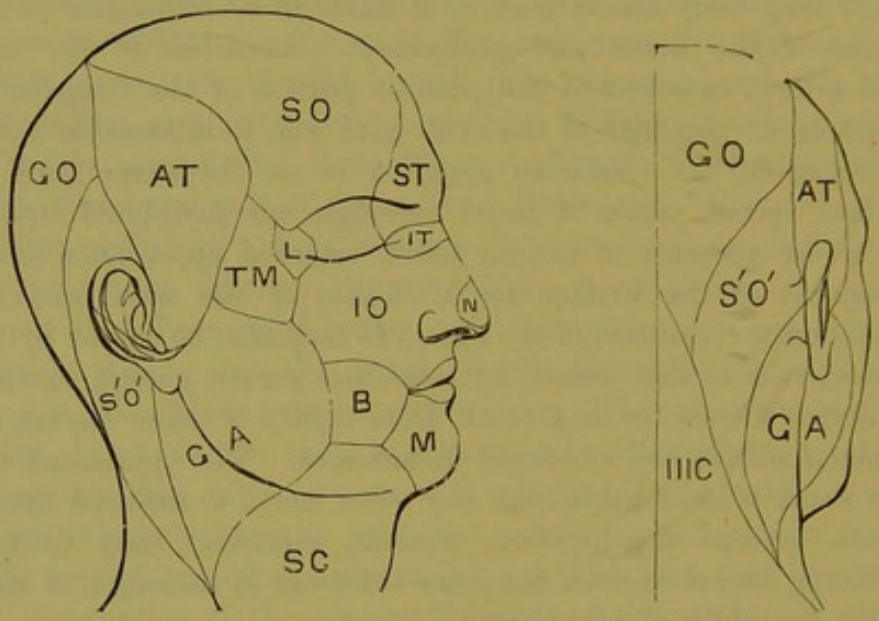
Etiology.—Trigeminal neuralgia occurs with unusual frequency in epileptic families, and in the female sex. The passage of the nerve through long bony canals renders it liable to be implicated in various affections of the bones and periosteum. Aneurism of the internal carotid artery, exostoses of the petrous portion of the temporal bone, and tumors at the base of the skull give rise to intractable neuralgia by pressure on the Casserian ganglion, or on the nerve itself. The important special causes of facial neuralgia are peripheral irritations, such as the presence of carious teeth, retarded appearance and false development of the wisdom teeth, disease of the nasal and frontal sinuses, and overexertion of the eyes. It may also be caused by remote irritation, such as that caused by intestinal worms, genital excitement, and uterine disease, or may result from injury of other nerves, excessive mental strain, and emotional disturbance. The ophthalmic branch of the nerve is more liable than any other nerve to malarial neuralgia. Anæmia, arterial degeneration, wounds, cicatrices, and diseases of neighboring tissues exercise the same influence in neuralgia of the fifth as in the neuralgiæ of other nerve territories.

Symptoms.—Facial neuralgia consists of attacks of pain in the area of distribution of the fifth nerve (Fig. 77) or of one of its branches, which are apt to recur on the slightest exposure to the exciting cause, or even in the absence of such cause. Each attack is made up of recurring paroxysms of severe pain, separated by intervals of comparative but not entire freedom from pain. The actual outburst of severe pain may be preceded by obscure feelings of discomfort, itching or formication in the side of the face, flying pains about the teeth, or by a feeling of general malaise and shivering, but at other times a severe dart of pain shoots along the course of one of the branches of the nerve without being preceded by any warning.

Each paroxysm consists of a succession of quick lightning-like darts of pain, which emanate from one or two foci and radiate towards the periphery. At first one or two of these flashes may be followed by a comparatively free interval, but they recur with increasing severity and quickness, until they at last blend into an uninterrupted pain of great intensity, during the continuance of which the patient suffers indescribable agony. After one or two minutes the intensity of the shooting pain

abates, but the patient continues to suffer during the interval from a dull aching pain, which occasions great discomfort and prevents sleep. The character of the pain during the paroxysm varies; it may be burning, boring, cutting, crushing, or stabbing, although the lightning-like shocks are most frequently met with. The intensity of the disease may vary from an attack which consists of a few darts of pain or a

Fig. 77.



Sensory Nerves of the Head and Face. (After Flower.)

First division of the fifth:
- SO, Supraorbital.
- ST, Supratrochlear.
- IT, Infratrochlear.
- L, Lachrymal.
- N, Nasal.

Second division of the fifth:
- IO, Infraorbital.
- TM, Temporo-malar.

Third division of the fifth:
- B, Buccal.
- M, Mental.
- AT, Auriculo-temporal.

Branches of the cervical plexus:
- GO, Great occipital.
- S'O', Small occipital.
- GA, Great auricular.
- SC, Superficial cervical.
- IIIC, Third cervical.

little tingling of the face, and manifests no tendency to recur, up to a disease of the most obstinate character, that recurs repeatedly and with great severity during the whole of life, and in which an attack may be determined by such a slight exciting cause as a current of cold air on the cheek, or such actions as chewing, coughing, washing the face, or slight emotional disturbances.

Painful points are observed during the attacks, and sometimes even in the periods of intermission, corresponding generally to the localities where the nerve becomes more superficial, either in issuing from a bony canal or in penetrating fasciæ.

The *concomitant symptoms* of trigeminal neuralgia are very numerous. During a severe paroxysm the pain often radiates to other nerve territories, extending to the other branches of the fifth, when one only is primarily affected, or to the occipital, cervico-brachial, or intercostal sensory nerves. The skin is generally hyperæsthetic in the early stages of the disease, and often anæsthetic in chronic cases. Disturbances of special sense have occasionally been observed in facial neuralgia, consisting of photophobia, amblyopia, or even amaurosis, and disorders of the senses of hearing, tasting, and smelling.

Motor disorders are almost always present in aggravated cases, and consist of clonic and tonic spasms of all the muscles of the affected side of the face, or of a few only of them, such as those of the eyelid or the angle of the mouth, while the muscles of mastication and of the tongue are sometimes attacked.

Vaso-motor disturbances are manifested by pallor and coldness in the early stage of the attack, but these are quickly followed by intense redness and elevation of temperature, and then the skin becomes glossy and œdematous. The redness extends to the mucous membranes supplied by the affected nerve, the conjunctiva being specially liable to become red and congested; the carotid, facial, and temporal arteries of the affected side may be seen to pulsate strongly, and the side of the face is covered by beads of perspiration.

Secretory disorders are represented by an increased flow of tears, by arrest or increase of the secretion of the mucous membrane of the nose, and occasionally by augmented salivary secretion.

The *trophic disturbances* consist of swelling of the face, changes in the color and texture of the hair, herpes zoster frontalis, erysipelas, subacute inflammation of the periosteum and of the fibrous membranes in the neighborhood of the painful points, neuroparalytic ophthalmia, iritis, and glaucoma. In aggravated and long-continued cases the incessant pain and its attendant sleeplessness undermine the constitution, the general nutrition becomes impaired, and at last the patient suffers from marasmus and nervous exhaustion.

The *psychical disorders* present are mental irritability and despondency, and hysterical seizures, whilst occasionally patients have committed suicide to escape from their sufferings.

Varieties of Trigeminal Neuralgia.

a. Ophthalmic or Supraorbital Neuralgia.

In this form several branches of the first division of the fifth nerve (Fig. 78) are all affected, or the pain is limited to some particular branch.

The painful points are:

(1) The *supraorbital point*, at or near the supraorbital foramen: (2) the *palpebral*, in the upper eyelid; (3) the *nasal*, at the point of

FIG. 78.

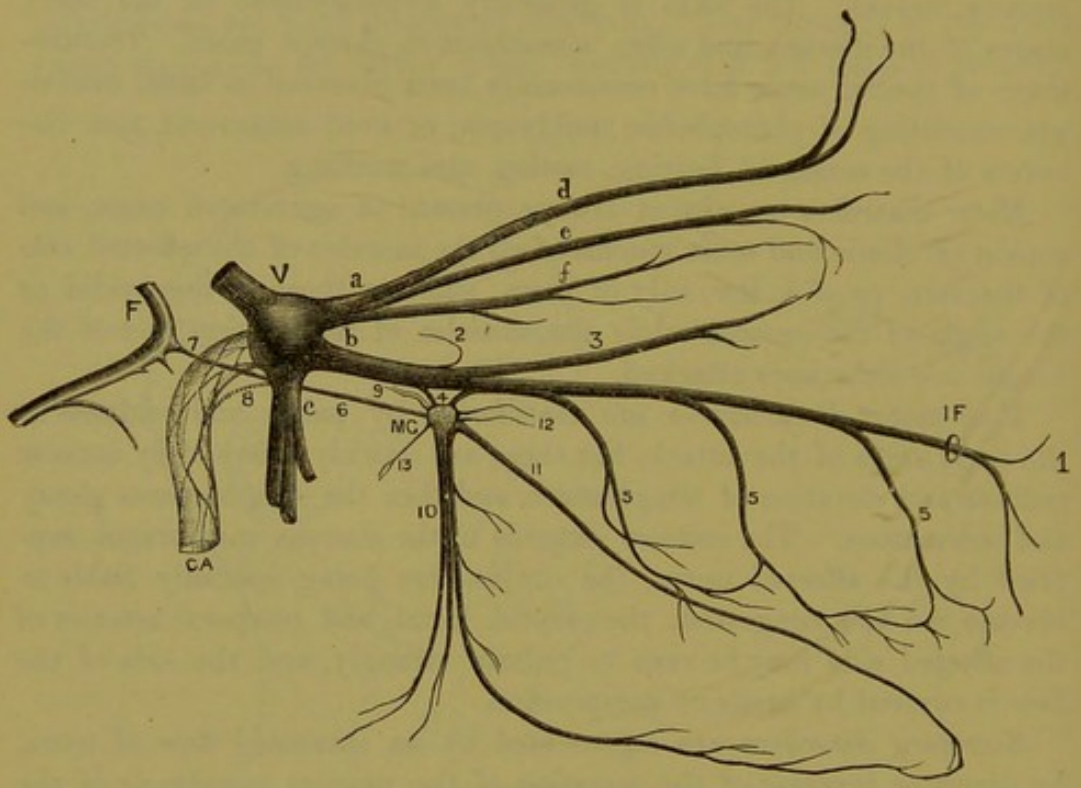


DIAGRAM OF THE FIRST AND SECOND (SUPERIOR MAXILLARY) DIVISIONS OF THE FIFTH NERVE, ITS CONNECTIONS AND CHIEF BRANCHES. (From HERMANN'S "Physiology.")

V, Placed over Casserian ganglion.

a, First or ophthalmic division, with *d* its *frontal*, *e* its *lachrymal*, and *f* its *nasal* branches

b, Second or superior maxillary division, branches of which are marked as follows:

1, Its *terminal* branches, *nasal*, *labial*, and *palpebral*.

2, *Recurrent* branch to the *dura mater*, and *middle meningeal artery*.

3, *Orbital* branch.

4 is placed between the two *spheno-palatine* branches (which descend to Meckel's ganglion)

5, Dental branches.

MG, Meckel's ganglion.

6, The Vidian nerve (constituting the motor and sympathetic root of Meckel's ganglion).

7, The *great superficial petrosal* nerve, from the geniculate ganglion of the facial nerve, joining the Vidian.

8, The *sympathetic* branch from the plexus on the carotid artery, joining the *great superficial petrosal*, and forming with it the *Vidian nerve*.

9, Ascending branches of Meckel's ganglion.

10, Descending palatine branches.

11, Naso-palatine branch.

12, Upper nasal branches.

13, Pharyngeal branch

F, Facial nerve. CA, Carotid artery. IF, Infraorbital foramen.

emergence of the long nasal branch at the junction of the nasal bone with the cartilage; (4) the *ocular*, a somewhat indefinite focus within

the globe of the eye when the ciliary nerves are affected; (5) the *trochlear*, at the inner angle of the orbit. The characteristic features of ophthalmic neuralgia are pain in the forehead, extending downwards to the upper eyelid and root of the nose, hyperæmia of the conjunctiva, lachrymation, and a painful spot at the supraorbital foramen. Malarial neuralgia almost always assumes this form; the attacks recur with great regularity, and are very intense.

b. Supramaxillary Neuralgia.

When all the branches of the second division of the fifth nerve (Fig. 78) are affected the pain is situated in the cheek, eyelid, lateral portion of the nose and upper lip (infraorbital nerve), in the zygomatic arch and anterior temporal region (orbital nerve), in the upper row of teeth (dental branches), and in the nasal cavities and gums (naso-palatine and posterior palatine nerves).

Infraorbital neuralgia is the most common variety of the second division of the nerve, and the characteristic pain is localized in the cheek, upper lip, upper row of teeth, and the neighborhood of the zygomatic arch. An obstinate form of neuralgia has been described by Gross which appears to have its seat in the remnants of the alveolar processes or the overlying gum in elderly persons who have lost their teeth.

The painful points of supramaxillary neuralgia are: (1) The *infra-orbital*, corresponding to the emergence of the nerve from its bony canal; (2) the *malar*, on the most prominent part of the malar bone; (3) an indeterminate focus somewhere in the line of the gum of the upper jaw; (4) the *superior labial*, also indeterminate; (5) the *palatine*, rare, but occasionally the seat of intolerable pain.

c. Inframaxillary Neuralgia.

When all the branches of the third division of the fifth nerve are affected the pain is localized in the region of the lower jaw and lower row of teeth (inferior dental nerve), in the chin (mental branch), in the tongue and mucous membrane of the mouth (lingual nerve), in the cheek (buccal nerve), and in the temporal region, anterior part of the auricle of the ear, and external auditory meatus (auriculo-temporal nerve).

The painful points are: (1) The *temporal*, a little in front of the ear; (2) the *inferior dental*, opposite the point of emergence of that nerve; (3) the *lingual*, on the side of the tongue; (4) the *inferior labial*. The *parietal* point, a little above the parietal eminence,

corresponds to the inosculation of various branches of the nerve, and may occur in all forms of trigeminal neuralgia.

FIG. 79.

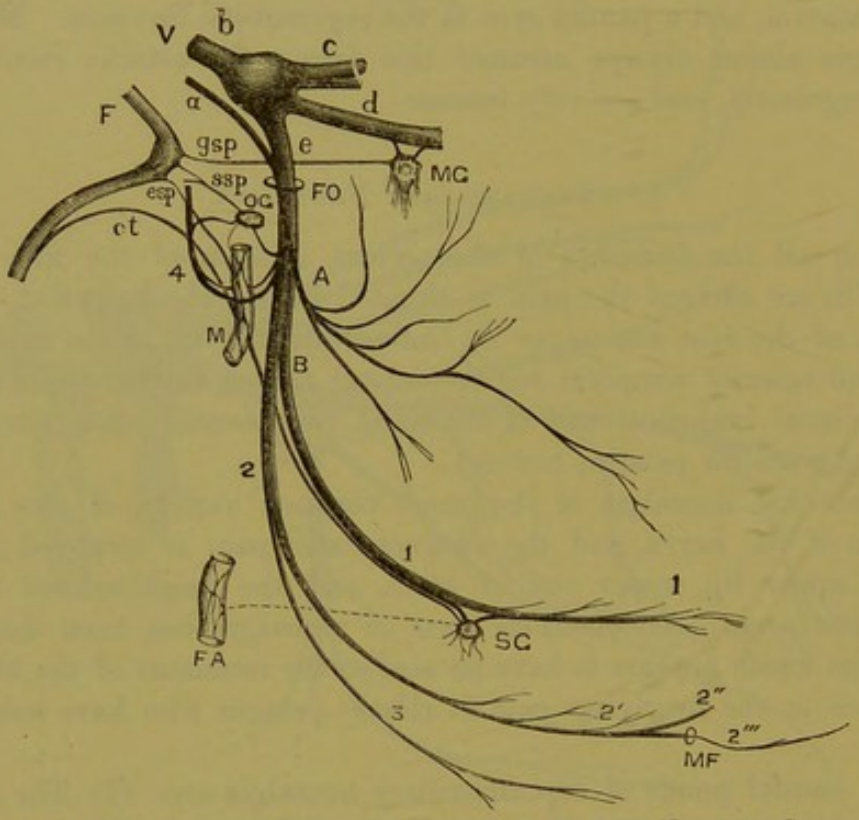


DIAGRAM OF THE THIRD (INFERIOR MAXILLARY) DIVISION OF THE FIFTH NERVE, ITS CONNECTIONS AND CHIEF BRANCHES. (From HERMANN'S "Physiology.")

V, Fifth nerve. *b*, Its largest sensory root, with Casserian ganglion.

a, Its smaller motor root joining *e*, the third division of the Casserian ganglion, to form the inferior maxillary nerve.

A, Anterior division of inferior maxillary nerve (mainly motor) supplying branches to the muscles of mastication, and a terminal *buccal* branch to the mucous membrane of the mouth.

B, Posterior division (mainly sensory); its branches are marked —

1, Lingual nerve; 1′, Branches to the tongue.

2, Inferior dental nerve; 2′ Its twigs to the teeth; 2″, Incisor branch; 2‴, Mental branch.

3, Mylohyoid branch to digastric and mylohyoid.

4, Auriculo-temporal nerve.

F, Facial nerve. *ct*, Its *chorda tympani* branch, joining the lingual and running to the submaxillary ganglion SG, of which it forms the motor root.

OG, Otic ganglion:

ssp, *Small superficial petrosal* nerve, connecting *otic ganglion* and facial nerve.

M, Middle meningeal artery, from the plexus upon which sympathetic filaments pass to the *otic* ganglion

esp, *External superficial petrosal* nerve, connecting the plexus on the middle meningeal artery with the facial nerve;

gsp, *Great superficial petrosal* nerve, connecting the facial with *Meckel's* ganglion.

FA, Facial artery, from the plexus upon which sympathetic filaments pass to the submaxillary ganglion.

FO, Foramen ovale. MF, Mental foramen.

SG, Submaxillary ganglion

d. Epileptiform Neuralgia.

In this form of facial neuralgia lightning-like pains of the most violent nature succeed each other with the greatest rapidity for a few seconds or minutes, and then suddenly vanish. These short attacks

Fig. 80.

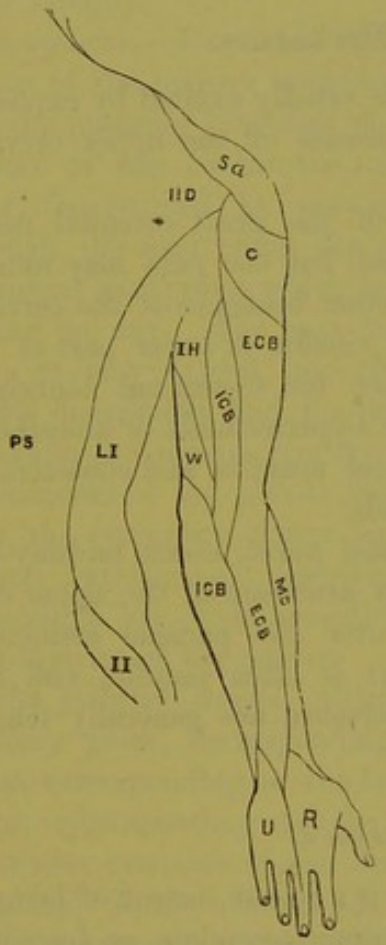
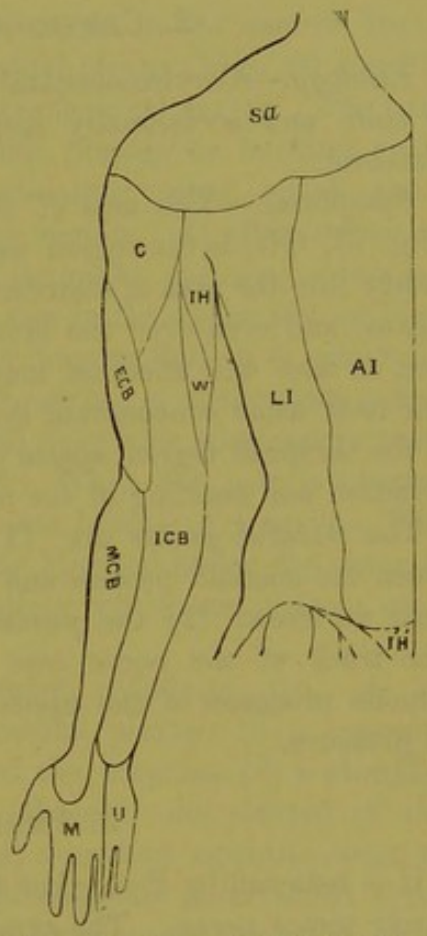


Fig. 81.



Figs. 80 and 81.—Cutaneous Nerves of the Trunk, Upper Extremity. (After Flower.)

Sa, Supraclavicular nerve.
IID, Second dorsal.
PS, Posterior branches of the spinal nerves.
LI, Lateral branches of the intercostal nerves.
AI, Anterior branches of the intercostal nerves.
II, Iliac branch of ilio-inguinal nerve.
I'H', Ilio-hypogastric nerve.
C, Circumflex nerve.
IH, Intercostal humeral
W. Nerve of Wrisberg
I'CB, Internal cutaneous branch of musculo-spiral nerve.
ECB, External cutaneous branch of musculo-spiral nerve.
ICB, Internal cutaneous nerve.
MC, Musculo-cutaneous nerve.
R, Radial nerve.
U, Ulnar nerve
M, Median nerve.

may, however, recur and follow each other in quick succession for a period of hours, days, or even weeks, when a respite follows, and the paroxysm disappears for days, weeks, or even years, although relapses are sure to recur after a longer or shorter time. This form of neuralgia is of centric origin, and occurs in families with a strongly marked neuropathic tendency, while it is often accompanied by epilepsy.

2. Cervico-occipital Neuralgia.

Etiology.—Cervico-occipital neuralgia is usually excited by exposure to cold, and occasionally results from disease of the upper cervical vertebræ.

Symptoms.—The area of distribution of the great occipital nerve (Fig. 77, GO) is the region usually affected, but the pain may radiate widely into the area of distribution of the other branches of the cervical plexus, and even over the brows, temples, cheeks, or lower part of the face, so that the affection may be mistaken for trigeminal neuralgia. The most usual concomitant symptoms are hyperæsthesia or anæsthesia of the occipital region, spasm of the cervical muscles, and considerable irritation and swelling of the cervical glands.

The *painful points* are: (1) the *occipital point*, about midway between the mastoid process and the spinous processes of the upper cervical vertebra; (2) the *parietal point*, over the parietal eminence. The track of the nerve over the occiput is often painful, and the spinous processes of the upper cervical vertebra are generally tender on pressure.

3. Phrenic Neuralgia.

It is believed by Peter that the phrenic is a mixed instead of being a purely motor nerve. The symptoms of phrenic neuralgia, as described by this author, are severe pains at the point where the nerve descends over the scalenus anticus muscle in its course through the chest, at the anterior and lower part of the thorax, and along the line of attachment of the diaphragm. Pain in the shoulder is a constant and characteristic symptom. Phrenic neuralgia is usually a symptom of epilepsy, hysteria, and angina pectoris, and is met with occasionally as a separate affection.

The *painful points* are: (1) The spinous processes of the upper cervical vertebræ; (2) the phrenic nerve in its course along the subclavicular fossa; (3) the line of attachment of the diaphragm, especially anteriorly between the seventh and tenth ribs; (4) a point over the cartilage of the third rib.

4. Cervico-brachial Neuralgia.

Etiology.—The most important exciting causes are the various injuries to which the upper extremities are so peculiarly exposed. It may also be caused by lead-poisoning and malaria, and may be a symptom of central diseases, as tabes hemiplegia and progressive muscular atrophy.

Symptoms.—Cervico-brachial neuralgia occurs in the area of distribution of the sensory branches of the brachial plexus (Figs. 80 and 81) and the posterior branches of the four lower cervical nerves. The pain is more or less continuous, and is of a dull, boring, or burning character, interrupted by paroxysms of lancinating pains, which shoot through the arm along the course of the nerves. It often occurs in nocturnal paroxysms which last through the night and may disappear during the day. The pain may have its seat in the upper arm or forearm, or may extend into the hand and fingers, but it is generally widely distributed, and the intimate interweaving of the various nerve trunks in the plexus renders it difficult to determine what nerve roots or branches of the plexus are implicated. When the pain is situated near the periphery it may be limited to one branch of a nerve. The violent burning pain described by S. Weir Mitchell under the name of "causalgia" is often present in the neuralgiæ which result from gunshot injuries of the nerves.

The *painful points*, which are somewhat indefinite, are: (1) an *axillary point*, corresponding to the brachial plexus; (2) a *scapular point*, corresponding to the lower angle of the scapula; (3) a *shoulder point*, corresponding to the emergence through the deltoid of the cutaneous branches of the circumflex; (4) a *median cephalic point*, at the bend of the elbow; (5) an *external humeral point* about three inches above the elbow; (6) a *superior ulnar point*, over the ulnar nerve in its course between the olecranon and the epitrochlear; and (7) a *radial point*, where the nerve becomes superficial at the lower and external aspect of the forearm. Painful points may occasionally be developed by the side of the lower cervical vertebræ, corresponding to the posterior branches of the lower cervical nerves.

The *concomitant* symptoms consist of cutaneous hyperæsthesia, numbness, and formication, or a considerable degree of anæsthesia. Radiating pains are felt in the region of distribution of the cervical plexus, and of the upper dorsal and intercostal nerves.

The *motor disorders* consist of twitchings of the muscles of the upper extremities, and in aggravated cases they may be maintained in

a state of persistent spasm. In chronic cases some of the muscles may be enfeebled or completely paralyzed, but neuritis is then probably present.

The *vaso-motor* disorders are coldness and pallor, or redness and heat of the affected extremity.

The *trophic disorders* consist of eruptions of herpes, but the aggravated cases which are caused by gunshot and other injuries of the nerves are complicated by pemphigus, obstinate ulcers, glossy skin, and changes in the growth of the nails and hair.

5. Dorso-intercostal Neuralgia.

Etiology.—Women are especially liable to this form of neuralgia. It comes on usually between the ages of twenty and forty years, in nervous, hysterical, and anæmic subjects, and its usual exciting causes are oversuckling, menorrhagia, and leucorrhœa.

The exciting causes of dorso-intercostal neuralgia are exposure to cold, injuries of various kinds, neuritis, neuromata, disease of the vertebræ or ribs, aortic aneurisms, pulmonary phthisis, dilatation of the venous plexus in the interior of the vertebral canal, and diseases of the spinal cord, such as transverse myelitis, spinal meningitis, spinal and meningeal tumors, and locomotor ataxia.

Symptoms.—The pain of intercostal neuralgia is seated in the area of distribution of the sensory branches of the twelve pairs of dorsal nerves; it is of a dull, and tensive character, and is usually continuous, but it is occasionally interrupted by tearing, lancinating, or burning pains. The pain is aggravated by all violent respiratory movements, and by slight pressure on the skin, but it is often relieved by firm and steady pressure. The pain not infrequently radiates towards the back and arm, or into the loins or lower extremities, and this form of neuralgia may be associated with brachial and lumbo-abdominal neuralgia, or with angina pectoris. The seat of true intercostal neuralgia is the skin of the anterior and lateral wall of the thorax and abdomen as far down as the symphysis pubis, and when the first two nerves are attacked the pain extends to the axilla and inner surface of the arm. When the posterior branches are affected the pain is seated in the back and loins as far down as the crista ilii. Dorso-intercostal neuralgia is generally unilateral except when it is a symptom of spinal disease, and the area of distribution of one or two branches on the left side from the fifth to the ninth nerve is the part most usually affected.

The *concomitant* symptoms consist of hyperæsthesia of the affected skin, while a circumscribed patch of anæsthesia has occasionally been

met with. Spasm of the intercostal muscles, giving rise to catching breathing like that of pleurisy, is often present. Of the vaso-motor and trophic disorders, herpes zoster is the most common; it may be present without neuralgia in young persons, but the neuralgia precedes and outlasts the eruption in old persons.

The *painful points* are: (1) a *vertebral point*, close to the vertebral column over the point of emergence of the nerve from the intercostal foramen; (2) a *lateral point*, over the spot in which the lateral perforating branch becomes subcutaneous; and (3) an *anterior* or *sternal point*, over the spot where the anterior perforating branch pierces the muscle close to the sternum, and in the abdomen over the rectus muscle. The whole length of the intercostal nerves is often sensitive, and several of the spinous processes of the corresponding vertebra tender to pressure.

Mastodynia.

Etiology.—Neuralgia of the female breast forms a special variety of intercostal neuralgia. It may appear at puberty in persons having a strong neurotic tendency, while anæmia, chlorosis, and hysteria act as predisposing causes. It is also liable to come on during pregnancy, although a large proportion of the pains felt in the gland during this period are caused by mechanical distention. The exciting causes of mastodynia are irritation of cracked nipples, shrinking of the nipples during lactation, injuries of the gland, and neuromata and painful tubercles of the nerves.

Symptoms.—The pain of "irritable breast" is very violent; it is described as tearing, cutting, boring, and lancinating, and appears in paroxysms which are usually of short duration, but may last several hours. The breast feels heavy, and the patient cannot lie on the affected side, while the slightest contact, even the pressure of the clothes, is unbearable. There is generally a great deal of hyperæsthesia, and the paroxysms are sometimes accompanied by vomiting. The pain radiates into adjoining regions, and the severity of the paroxysm is increased during the catamenial period.

Painful points may be found on the nipple or on the sides of the breast, but they are indefinite. The spinous processes of the second to the sixth dorsal vertebra are generally tender to pressure.

6. Lumbar Neuralgia.

Etiology.—The special exciting causes of lumbar neuralgia are compression of the nerves by herniæ, accumulation of feces, cancer in the pelvis or in the vertebral column, diseases of the uterus and vagina,

coitus, psoas abscess, chronic spinal diseases, and diseases of the vertebral column.

Symptoms.—Lumbar neuralgia includes all forms having their seat in the area of distribution of the sensory branches of the first four pairs of lumbar nerves. The pain is usually limited to one or two of the branches of the plexus. Lumbar neuralgia may be divided into (1) lumbo-abdominal and (2) femoral neuralgia, while it will be convenient to describe in this place (3) neuralgia of the external generative organs, even although these organs and the neighboring parts are supplied by branches from the sacral as well as from the lumbar nerves.

a. Lumbo-abdominal Neuralgia.

The pain in this variety is seated in the loins, hypogastrium, mons veneris, and scrotum or labia majora, and extends over the crista ilii as far as the buttock, while the inguinal region may be occasionally affected. The usual concomitant symptoms are spasm of the cremaster, vomiting, herpes, and increased sexual desire with priapism and ejaculation of seminal fluid.

The *painful points* are: (1) *Vertebral points*, corresponding to the posterior branches of the respective nerves; (2) an *iliac point*, at the middle of the crista ilii; (3) an *abdominal point*, at the side of the linea alba above the symphysis pubis; (4) an *inguinal point*, in the groin near the exit of the spermatic cord; (5) a *scrotal* or *labial point*, in the scrotum or labium majus. The pain frequently radiates into neighboring nerve territories.

b. Femoral Neuralgia.

(j) *Neuralgia of the lateral cutaneous nerve of the thigh* is situated in the outer and part of the posterior aspect of the thigh as far as the knee.

The *painful points* are: (1) Spot over *anterior superior spinous process*, which is constant; (2) spots along the outer aspect of the thigh, which are less constant.

(jj) *Crural neuralgia* is situated in the middle and inner part of the anterior surface of the thigh, the anterior surface of the knee, and the inner surface of the leg, and of the foot as far as the great toe The concomitant symptoms are hyperæsthesia or anæsthesia of the skin, especially in the vicinity of the knee-joint, a feeling of numbness or formication in the region of distribution of the saphenous nerve, and a feeling of weariness on exertion, or paresis of the muscles of the thigh.

The *painful points* are: (1) One in the fold of the groin, where the nerve emerges from the pelvis; (2) one at the inner side of the patella, where the saphenous nerve becomes subcutaneous; (3) one in front of the ankle-joint; and (4) one at the base of the great toe.

(jjj) *Obturator* neuralgia is situated in the inner side of the thigh, and extends as far as the knee-joint. It is associated with obturator hernia, and the concomitant symptoms are formication on the inner surface of the thigh, and a feeling of stiffness and immobility of the adductors of the thigh.

c. Neuralgia of the External Organs of Generation.

(j) *Neuralgia of the Penis or Clitoris.*—The pain has its seat in the glans and extends to the root of the penis. The pain is violent, lancinating, and burning, and may be unilateral or bilateral; it is increased by sexual intercourse and urination, and it may be accompanied by priapism and frequent ejaculations. The clitoris is sometimes the seat of painful erections in the early stage of tabes dorsalis.

(jj) *Neuralgia scrotalis vel labialis* is a common symptom of lumbo-abdominal neuralgia. The scrotum or labium majus is often tender to touch.

7. Neuralgia of the Sacral and Coccygeal Nerves.

Neuralgia affecting the sensory branches of the sacral and coccygeal nerves may be divided into (*a*) sciatica, (*b*) plantar neuralgia, and (*c*) coccygodynia.

a. Neuralgia Ischiadica (Sciatica).

Etiology.—Heredity does not appear to exercise much influence in the production of sciatica. The largest number of cases occur between forty and fifty years of age or during the period of commencing bodily degeneration, and after the age of thirty years males are much more frequently attacked than females. The exciting causes are exposure to cold, gunshot wounds, and injuries of the nerve by blows, falls on the buttock, or by compression of the nerve in difficult labors, especially when the forceps is used. Other causes are mechanical pressure on the nerve from sitting on hard seats, enlargements and displacements of the uterus, pregnancy, accumulation of feces in the sigmoid flexure, and pelvic tumors. Portal congestion, habitual constipation, and all conditions which give rise to congestion of the hemorrhoidal and other pelvic veins may cause sciatica, while it may also result from the poisons of rheumatism, gout, and syphilis.

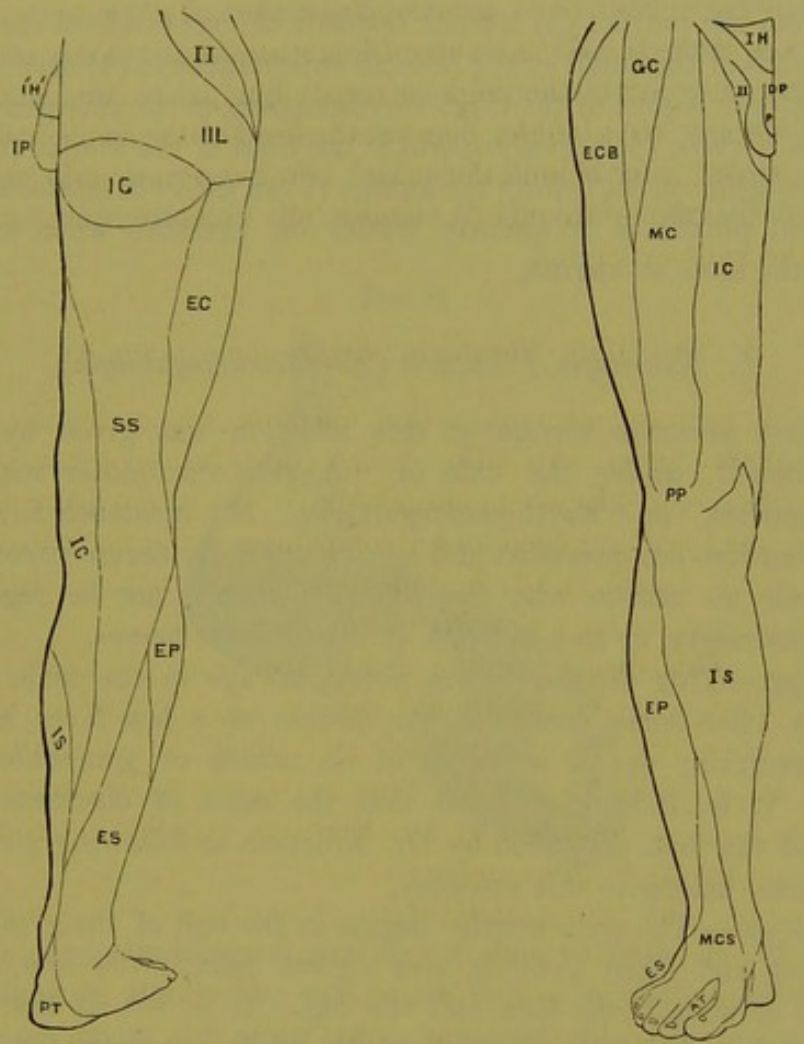
Symptoms.—The pain is seated in the area of distribution of the sensory branches of the small and great sciatic nerves (Fig. 82). An attack of sciatica is generally preceded by premonitory symptoms, such as a sensation of fluid trickling over the skin, or a feeling of cold or heat, formication, and a sense of stiffness and dragging. The symptoms of true neuralgia now make their appearance, and consist of lightning-like pains which increase in intensity and recur with greater and greater frequency, until a violent paroxysm of almost continuous pain is established. After a time the violence of the pain abates and the patient has an interval in which he is comparatively but not completely free from pain, but after a longer or shorter period he is seized with another violent paroxysm. The pain is usually superficial, but at times it may be felt in and between the muscles, or even in the bones; it proceeds from one or more fixed points, and generally shoots along the branches of the nerve to the periphery, while a nocturnal exacerbation is frequently experienced. It is aggravated by all movements of the limb, and such actions as coughing, sneezing, straining at stool, or even simple contact of the bedclothes may bring on a paroxysm, and consequently the limb is maintained in a fixed position with all the joints slightly flexed.

The pain is most commonly felt in the posterior surface of the thigh, commencing in the neighborhood of the sciatic foramen, and extending to the popliteal space and calf of the leg. The next part most frequently affected is the peroneal region, including the anterior and external surface of the leg and the dorsum of the foot. Sometimes the whole area of distribution of the sciatic nerves is affected, and the violence of the pain may shift from one region to another. Violent pains are also felt in the sacrum and loins from implication of the posterior branches of the sacral nerves, while the pain may also shoot into the lumbar nerves and their branches, into the sciatic nerve of the opposite side, or into more remote nerve territories.

The *concomitant* symptoms are hyperæsthesia, or partial anæsthesia, and diminution of the electric sensibility of portions of the skin, cramps of particular groups of muscles, which are especially severe at night when the patient is falling asleep, and in long-standing cases considerable diminution of motor power, which renders the gait limping. Complete paralysis only occurs when there is neuritis. The most usual vaso-motor and trophic disorders are pallor and coldness, or redness and heat of the surface, increased secretion of sweat, wasting or occasionally hypertrophy of some of the muscles.

The *painful points* are: (1) a series of points reaching from the lower end of the sacrum up to the crista ilii, representing the posterior

FIG. 82.



CUTANEOUS NERVES OF THE LOWER EXTREMITY. (After FLOWER.)

Lumbar Plexus.

IH, Ilio-hypogastric nerve.
II, Ilio-inguinal.
IIL, Second lumbar nerve.
GC, Genito-crural.
EC, External cutaneous.
MC, Middle cutaneous.
IC, Internal cutaneous.
IS, Internal saphenous.
PP, Plexus patellæ.

Sacral Plexus.

DP, Dorsalis penis of pudic.
IP, Inferior hemorrhoidal of pudic.
P, Superficial perineal of pudic and inferior pudendal of small sciatic.
IG, Inferior gluteal of small sciatic.
SS, Small sciatic.
EP, Branches from external popliteal.
ES, External saphenous.
MCS, Musculo-cutaneous.
AT, Branches of anterior tibial.
PT, Branch of posterior tibial.

branches; (2) a point opposite the emergence of the great and small sciatic nerves from the pelvis; (3) a point opposite the spots where the ascending branches of the small sciatic become subcutaneous; (4) several points at the posterior aspect of the thigh corresponding to the emergence of the cutaneous nerves; (5) a *fibular point* at the head of the fibula; (6) an *external malleolar point* behind the ankle; (7) an *internal malleolar point*. The sacral plexus is frequently tender on pressure when examined through the anus or vagina.

b. Neuralgia Plantaris (Erythemomegalalgia).

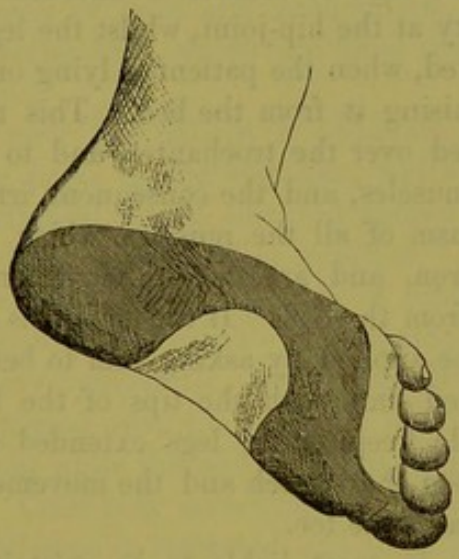
The first accurate account of this affection was given by Dr. S. Weir Mitchell, under the title of "A rare vaso-motor neurosis of the extremities," or "Erythemomegalalgia," but inasmuch as the vaso-motor disorders are preceded and accompanied by severe paroxysms of pain, I see no reason why the affection should not be regarded as *plantar* neuralgia, or as a *neuritis of the plantar nerves*.

Etiology.—This disease occurs nearly always in the male sex, and comes on after some constitutional disease as a low fever, after prolonged exertion, or as a sequel of an attack of gonorrhœal rheumatism. It is, indeed, probable that the cases of obstinate pain in the sole of the foot, described by Dr. Elliotson as following gonorrhœal rheumatism, belong to this category.

Symptoms.—The pain usually begins in the ball of the great toe, or in the heel, and often extends over a great part of the sole, and may reach the dorsum of the foot, and the leg. It is felt at first towards night, and is relieved by the night's rest, while it is increased by walking, the erect posture, or even by allowing the foot to hang down. The pain in the early stage of the disease consists of a deep-seated aching, but after a time it becomes of a burning character, and is then aggravated by warmth and relieved by cold and the recumbent posture. The most characteristic symptom of the affection, however, is a flushing of the painful area, which comes on with exertion or when the feet are allowed to hang down, and is accompanied by swollen veins and violent throbbing of the arteries. The shaded part in the annexed diagram shows distribution of the reddened surface in one of Dr. Mitchell's cases. In the worst cases the extremity is pale and cold when the patient is at rest. In aggravated cases the pain is so severe as to render walking all but impossible, and when persisted in intense redness and swelling are occasioned, the patient sleeps with his feet uncovered, and may even be reduced to crawl on his hands and knees, or is obliged to be carried about in order to avoid placing his feet on the ground.

The feet are generally bathed in sour-smelling sweat, and the skin of the sole may have a sodden appearance, becoming somewhat glazed during the paroxysm of pain and redness. The disease is sometimes progressive, and in its later stages may be associated with evidences of spinal disease, such as girdle pains and partial paralysis with atrophy of some of the muscles of the leg, while the faradic contractility has been found diminished in the muscles of the limb most affected. The

FIG. 83.



disease is generally bilateral, and in one case observed by Dr. Mitchell the hands were attacked as well as the feet. In each of several cases which came under my observation a tender spot was present in the centre of the heel, the whole course of the external plantar nerve was tender to pressure, and painful points were found between the heads of the metatarsal bones, over the bifurcation of the branches of the plantar nerves for the digits.

c. Neuralgia of the Coccygeal Nerves (Coccygodynia).

Etiology.—This affection occurs generally in women in consequence of injury to the coccyx from a fall, or during labor. It may also be caused by exposure to cold, or may originate spontaneously.

Symptoms.—The chief symptom of coccygodynia consists of pain in the region of the coccyx when the patient sits or walks, and it is often felt during micturition and defecation, especially if there be much straining. Pressure on the coccyx, by the finger, aggravates this pain.

named *gastric crises*, is a very frequent and distressing symptom of locomotor ataxia.

Symptoms.—Gastralgia is characterized by paroxysmal attacks of pain in the epigastric region which may radiate upwards to the back between the shoulders, or to the middle of the sternum. The attack comes on suddenly, without premonitory symptom, and the pain, which is very severe, generally intermits after a few minutes, but soon recurs with greater intensity, and after repeated intermissions and recurrences it finally disappears. Pressure over the cartilages of the false ribs on the left side, or on the corresponding intercostal spaces, may cause pain, and the spinous processes of some of the dorsal vertebræ may be tender on pressure. The pain of gastralgia is relieved by firm and uniform pressure, and tenderness of the epigastrium is generally absent. The upper portions of the recti muscles are strongly contracted during the attack and the abdominal walls are rendered tense and unyielding, whilst the epigastric region is usually retracted. The pulse is generally slow and feeble; the arterial tension is low; the extremities are cold and pale; and towards the end of the attack the patient may suffer from chilliness and a feeling of oppression and faintness like that of angina pectoris reflectoria. The attack often terminates by copious vomiting, the food contained in the stomach is first ejected, and then large quantities of watery fluid mixed with bile and blood, and in aggravated cases, mucus; while if the urine be examined during or soon after the attack it is often found to contain a small quantity of albumen.

Tabetic gastric crisis is a variety of gastralgia which begins during an attack of the lancinating pains of locomotor ataxia. The patient complains of pain which starts from the groins, and passes up each side of the abdomen to become fixed in the epigastrium, while at the same time severe lightning pains dart from between the shoulders and radiate round the base of the thorax. Severe vomiting now sets in, just as occurs in all severe cases of gastralgia. The patient suffers during the attack from a profound malaise; the action of the heart is accelerated, and the lightning pains are unusually severe. The attack may last without respite for two or three days, and may recur every two or three weeks although the usual interval between them is not less than a month.

d. Neuralgia Hepatica (*Hepatalgia*).

Etiology.—Hepatic colic is usually the result of the passage of biliary calculi through the cystic and common ducts, but colic of similar character sometimes occurs in neurotic subjects in the absence of any signs of biliary obstruction and it is regarded as of purely neuralgic origin.

Symptoms.—The symptoms of hepatic colic are more or less severe pain which comes on suddenly and lasts with irregular intermissions and exacerbations from a few hours to a few days. The pain is often very severe, and of an aching, cutting, or tearing character, and is usually attended with a feeling of constriction or cramp. It is generally referred to the pit of the stomach or to the umbilicus, whence it radiates to the back and between the shoulders, but never downwards. The patient suffers during a severe attack from faintness, nausea, and vomiting; the action of the heart is weakened, the surface of the body is cold, and in severe cases there are symptoms of collapse.

e. Neuralgia Hypogastrica.

Hypogastric neuralgia may be divided into the following varieties according to the branches of the plexus affected.

(1) Neuralgia Ani.

Etiology.—Neuralgia of the rectum generally results from fissures of the anus, but it may occur in the absence of any recognizable local cause; and it is a common symptom of locomotor ataxia.

Symptoms.—The symptoms consist of severe paroxysms of cutting pains coming on suddenly and spontaneously after exposure to cold. The pains are situated an inch within the anus, and they are much increased by defecation. The pain is associated with hyperæsthesia or anæsthesia of the skin of the perineal region, spasm of the sphincter ani and bladder, and difficulty of micturition.

(2) Neuralgia Uteri (Hysteralgia).

Etiology.—The causes of uterine neuralgia are prolapse of the uterus, uterine tumors of all kinds, ulceration of the cervix, profuse and intractable leucorrhœa, ascarides in the rectum, scybala impacted in the rectum, and calculus in the ureter or kidney. Sometimes the source of irritation may be in a remote part of the body, and in some cases paroxysms of uterine neuralgia may occur in the absence of any discoverable disease of the pelvic organs.

Symptoms.—The patient complains of paroxysmal attacks of intense pain situated deeply in the pelvis which is aggravated by movement, by the maintenance of the erect posture, and by pressure on the cervix. The pain often radiates to the inguinal and lumbar regions of one side, and it is often, although not always, worse at the menstrual period.

Vaginismus is a condition of excessive sensibility of the vaginal orifice which renders coitus impossible. It is often associated with spasm of the constrictor vagina and of the levator ani, and attempts at coition may induce general hysterical convulsions.

(3) Ovarian Neuralgia and Hyperæsthesia (Ovarialgia).

Etiology.—Ovarian neuralgia may be caused by any of the sources of irritation already enumerated as the causes of uterine neuralgia. The left ovary is the one usually affected. It is frequently associated with grave hysterical symptoms, and it is difficult to determine whether the ovarian pain is to be regarded as the cause or an effect of the hysteria.

Symptoms.—The symptoms of ovarian neuralgia consist of pain, which is sometimes so acute that the patient cannot tolerate the slightest touch, and which is localized partly in the hypogastrium and partly in the iliac fossa. In many cases there is more or less complete anæsthesia of the abdominal walls and pain is only elicited on deep pressure over the affected ovary, which can generally be felt by the hand in the other variety of ovarian neuralgia. Pressure exaggerates this iliac pain, and causes it to radiate towards the epigastrium and throat. The radiation of the pain towards the epigastrium is often accompanied by nausea and vomiting, and, if the pressure be continued, palpitations of the heart, increased frequency of the pulse, and a sensation of "globus hystericus" supervene. If the pressure be continued, the patient, according to Charcot, suffers on the affected side, in addition to the symptoms just described, from loud sibilant sounds in the ear, obscurity of the sight of the eye, and a sensation as of blows from a hammer on the temporal region.

(4) Neuralgia Testis.

Etiology.—Neuralgia of the testis may be caused by morbid growths in the organ, and calculus in the ureter, while it is not uncommon in neurotic subjects as the result of self-abuse.

Symptoms.—The symptoms of neuralgia of the testis consist of spontaneously arising paroxysms of severe pain which is situated partly in one testicle and partly in the epididymis and cord. The organ is tender to pressure and contact, and the patient complains of a dull aching pain in the intervals between the paroxysms. During the paroxysm the testicle is strongly retracted from spasm of the cremaster muscle, and the patient suffers from fainting and vomiting. The affection is usually very obstinate, and it is often accompanied by great

mental depression which may end in confirmed hypochondriasis or melancholia.

(5) Cystalgia.

Etiology.—Neuralgia of the bladder usually results from the presence of calculus, or malignant disease, but it occasionally results in women from long-continued menorrhagia combined with anæmia.

Symptoms.—Neuralgia of the bladder consists of severe paroxysms of pain, which is situated at the neck of the bladder, and is accompanied by a frequent desire to micturate.

(6) Neuralgia Urethralis.

Etiology.—Neuralgia of the urethra may occur in the absence of any local source of irritation, and is then generally a symptom of locomotor ataxia.

Symptoms.—Neuralgia of the urethra consists of paroxysms of severe pain in the course of the urethra. The pain is accompanied by an urgent desire to pass water, probably caused by spasm of the detrusor muscle, but the patient frequently experiences difficulty in voiding the urine, probably because the sphincter is also spasmodically contracted.

IV. DISORDERS OF THE NERVES OF SPECIAL SENSE.

1. Diseases of the Olfactory Nerve.

The sense of smell is excited by the contact of odoriferous particles with that part of the mucous membrane of the nose to which the olfactory nerves are distributed. The smell induced by simple diffusion of odoriferous particles is very imperfect, but the sensation is much intensified by the forcible impact of those particles against the mucous membrane which is obtained by the process of *sniffing*.

Tests.—In testing the sense of smell care should be taken not to irritate the fifth nerve, and consequently substances having a pungent odor should be avoided. The best substances are volatile oils, the fetid gum resins, and other substances having a penetrating odor like camphor and musk. Our perception of flavors being due to the sense of smell and not to that of taste, it is necessary to get the patient to partake of substances possessing a delicate *aroma* or *bouquet*, such as roast beef, cheese, or wine. Electricity is not of much use in testing the sense of smell.

a. Hyperosmia or Olfactory Hyperæsthesia.

The acuteness of the sense of smell may, like that of the other senses, be increased by education, and hysterical patients are often able to discriminate smells which are quite inappreciable to others.

b. Hyperalgesia of the Sense of Smell.

The sense of smell is frequently perverted in insanity, and other grave diseases of the nervous system. Hysterical subjects may manifest a decided aversion to perfumes which are agreeable to most people, or a predilection for odors, like that of asafœtida, which are repugnant to others. An unaccountable aversion to a particular perfume is sometimes one of the earliest symptoms of locomotor ataxia.

Illusions or hallucinations of smell, which are generally of a repugnant kind, are often complained of by the insane. The most usual odors complained of are those of sulphur and putrid substances, and these patients are often led to believe that they are surrounded by dead and decaying bodies. A bad smell sometimes constitutes the aura of epileptic and epileptiform seizures, and it may likewise be a symptom of an intracranial tumor.

c. Anosmia or Olfactory Anæsthesia.

Anosmia consists of a diminution or complete loss of the sense of smell, and when both sides are affected the sense of taste is likewise impaired. The sense of smell is diminished in disease of the fifth nerve owing to nutritive changes occurring in the mucous membrane of the nose, and it is also diminished in facial paralysis because the act of *sniffing* becomes impossible, owing to the paralysis of the dilators of the alæ nasi. It is likewise impaired by local disease, such as constriction of the nostrils, nasal polypi, acute and chronic inflammation of the mucous membrane, strumous and syphilitic ozæna, and occlusion of the nasal and pharyngeal cavities. The sense of smell is frequently blunted in old age, and loss of smell is occasionally observed as an early and persistent symptom of locomotor ataxia. Loss of smell is sometimes caused by injuries of the head, the affection being thus described as *traumatic anosmia*. The most frequent injury to cause anosmia, in the absence of fracture of the cranium, is a blow or fall on the occiput, and a blow on the forehead is sometimes followed by loss of smell. Anosmia of the left nasal cavity is sometimes associated with right hemiplegia and aphasia, the result of embolism of the middle cerebral artery.

In other cases left-sided anosmia is associated with aphasia in the absence of any paralysis, and in some of these cases at least the disorder of speech was a sensory aphasia. In cerebral hemianæsthesia, whether of hysterical origin or caused by organic disease, smell is lost on the same side as the other sensory disorders. Loss of smell is also caused by tumors in the anterior fossa of the skull, or in the anterior cerebral lobes, and by basal meningitis, exostoses, or caries of the bones. It is at other times a congenital affection, and then usually results from absence of the olfactory bulbs.

Morbid Anatomy and Physiology.—Diminution or loss of smell may be caused by disease of the local olfactory organ, the peripheral end-organs, the olfactory nerves, the conducting apparatus through the olfactory bulbs and hemispheres of the brain, or of the cortical centres themselves. With regard to local disease it will suffice to say, that it is very probable the ramifications of the olfactory nerves themselves become secondarily affected in chronic inflammation and other diseases of the mucous membrane. Loss of smell may, however, be caused by disease of the peripheral end-organs of the olfactory nerve in the absence of disease of the mucous membrane. Smell is sometimes permanently lost in consequence of the impression of a very powerful stench, and Prévost found degeneration of the olfactory nerves and atrophy of the bulbs in old people in whom the sense of smell was diminished. The anosmia which accompanies locomotor ataxia is caused, most probably, by a neuritis of the olfactory nerves, while that which accompanies cerebral tumors is most frequently caused by compression of the olfactory nerves and bulbs. Traumatic anosmia is generally caused by blows on the occiput; the elastic bones of the cranium yield to some extent to the blow; the whole of the encephalon above the tentorium is thus pushed forwards; the anterior margins of the temporo-sphenoidal lobes impinge against the great wings of the sphenoid bones; and the olfactory bulbs are apt to suffer damage at these points of junction with the brain near the anterior perforated space, or some of the olfactory nerves may be ruptured in their passage through the cribriform plate of the ethmoid bone.

2. Diseases of the Acoustic Nerves.

a. Auditory Hyperæsthesia or Hyperacusia.—In this condition the sensibility of the auditory sensory mechanism is increased, so that sounds too faint to be detected in the normal state are heard distinctly, while the power of discriminating differences in sounds is unusually acute. Hyperacusia is often observed in hysterical patients, in con-

ditions of ecstacy, and sometimes even in somnambulism. Auditory hyperæsthesia, however, generally declares itself, not by an increased power of discriminating tones, but an increase of the pleasant or painful feelings which accompany sound, and this condition may be called *auditory hyperalgesia*. In disease the painful feelings which accompany sound predominate, and in cases of acute disease, general debility, hysteria, and various mental affections, the slightest sound becomes exceedingly distressing to the patient. Auditory hyperæsthesia is often accompanied by subjective sensations, illusions, and hallucinations. *Tinnitus* is the most common form of subjective sensation, and it may assume the form of whistling, humming, and various other noises. This symptom is generally caused by disease of the external or middle ear, changes in the circulation of the brain, large doses of quinine or salicin, and forms a prominent feature of Ménière's disease. Auditory *illusions* are frequently observed in mental disease, more especially in melancholia and dementia; in the former they assume the form of abusive or threatening words or commands to do acts of violence, and in the latter they take the form of heavenly messages or revelations. Auditory illusions are sometimes unilateral, and in some cases there are alternating illusions of different senses, such as right-sided optic and left-sided auditory illusions. Auditory *hallucinations* sometimes constitute the aura of an epileptic attack. The galvanic current affords valuable aid in the diagnosis of the various conditions of the auditory nerve, but for information on this subject the reader is referred to special treatises.

b. Auditory anæsthesia consists of abnormal diminution or abolition of the sense of hearing. The cause of deafness may be either lesion of the local apparatus of hearing and of the peripheral nerve-endings, of the conducting apparatus, or of the central terminal organ. Functional deafness is observed in hysteria, and after toxic doses of quinine, lead, and other agents.

3. Diseases of the Gustatory Nerves.

The gustatory nerves are derived from the glosso-pharyngeal nerve (Fig. 84) which supplies fibres to the posterior third of the tongue, the palate, and the walls of the pharynx; and from the lingual branches of the fifth nerve, which are distributed to the tip and anterior two-thirds of the tongue. It has been proved by experiment and by an analysis of clinical observations that most, if not all, of the gustatory fibres of the lingual nerve are derived from the chorda tympani; this nerve joins the facial in the Fallopian canal, but on reaching the genicu-

late ganglion the gustatory fibres leave the facial and return by means of the petrosal nerves to the trigeminus, with which they enter the cavity of the skull and brain. Gowers believes that the gustatory fibres of the glosso-pharyngeal are also derived from the fifth nerve.

FIG. 84.

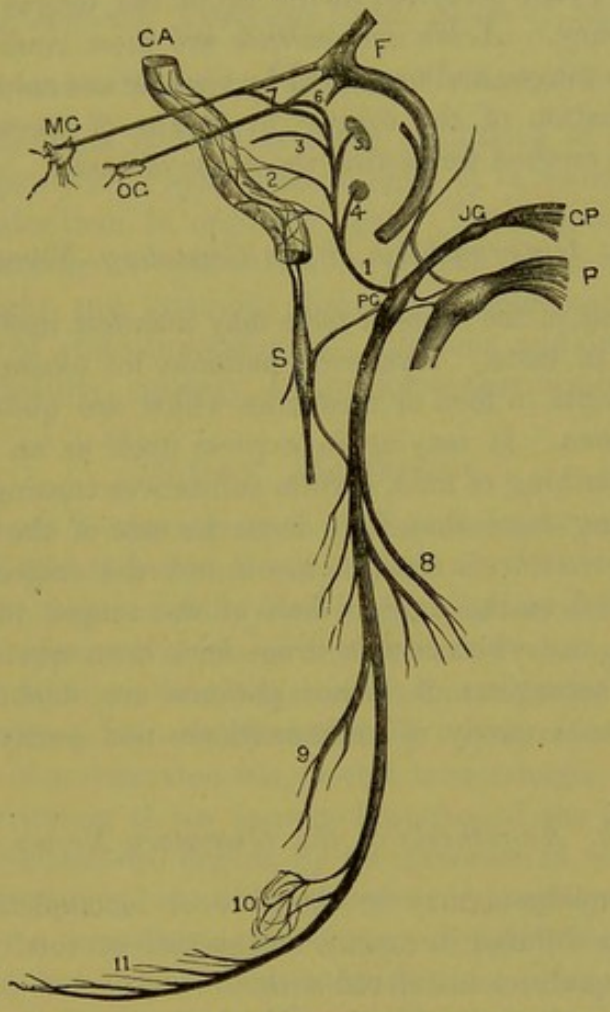


DIAGRAM OF GLOSSO-PHARYNGEAL NERVE, ITS CONNECTIONS AND BRANCHES.
(From HERMANN'S "Physiology.")

GP, Glosso-pharyngeal nerve. JG, Its *jugular ganglion*. PG, Its *petrous ganglion*.
1, Tympanic branch, or nerve of Jacobson, the branches of which are as follows:
2, Filaments to plexus on carotid artery; 3, To Eustachian tube; 4, To *fenestra rotunda*; 5, To *fenestra ovalis*.
6, Twig of union with *small superficial petrosal n.*
7, Twig of union with *great superficial petrosal n.*
8, Pharyngeal branches of glosso-pharyngeal n.
9, Muscular branches to stylo-pharyngeus and constrictors (?).
10, Tonsillitic branches.
11, Terminal lingual branches.
P, Pneumogastric nerve, from the *ganglion* of the *root* of which, branches pass to the petrous ganglion.
S, Superior cervical ganglion with an ascending branch to the *petrous* ganglion.
MG, Meckel's ganglion. OG, Otic ganglion. F, Facial nerve. CA, Carotid artery.

Tests of the Sense of Taste.—The patient should be directed to put his tongue out, with the mouth widely open and the eyes closed, and then

the sapid substance is applied, with a glass rod or small brush, to the part to be tested. The tongue ought not to be withdrawn into the mouth until time is given for the taste to be perceived.

Bitter tastes are most distinctly perceived at the root of the tongue, and they may be tested by a solution of quinine or infusion of quassia. *Sweet tastes* are best perceived at the tip of the tongue and are tested by syrup or honey. *Acids and salines* are most readily perceived at the sides of the tongue and are tested by vinegar and solution of common salt. Galvanization of the tongue by means of special electrodes is also a valuable method for testing the sense of taste.

a. Hyperæsthesia of the Gustatory Nerves.

Hyperæsthesia of the sense of taste may manifest itself as an increase in the delicacy of taste. Hysterical patients, for example, often detect certain ingredients in food or medicines which are quite inappreciable to healthy persons. It may again express itself as an increase in the enjoyment or loathing of food, certain substances causing a more agreeable or disgusting taste than they do in the case of the healthy palate. Amongst the *paræsthesia* must be mentioned the subjective sensations of taste perceived on the anterior half of the tongue in some cases of facial paralysis, and when certain drugs have been taken. The subjective gustatory sensations of insane patients are, doubtless, of centric origin, and consist partly of hallucinations, and partly of illusions of taste.

b. Anæsthesia of the Gustatory Nerves.

Gustatory anæsthesia may be complete or incomplete in its degree, circumscribed or diffused in extent, and partial or total as regards the forms of sapid qualities interfered with.

Gustatory anæsthesia may be caused by catarrh or other local disease, or by lesion of the glosso-pharyngeal, lingual, chorda tympani, facial, or trigeminal nerves. When the glosso-pharyngeal nerve is affected taste is weakened or lost on the root of the tongue, palate, and pharynx on the corresponding side, but no uncomplicated case of this kind is yet recorded. Little is known with regard to disease of the cortical centres of taste. If the lesion is situated on the lingual or chorda tympani nerves, or in certain sections of the facial or trigeminal nerves, the gustatory anæsthesia affects the anterior or lateral half of the tongue along with its border and apex. When loss of taste in the areas of distribution of the lingual and glosso-pharyngeal nerves of one side is associated with hemianæsthesia of the same side of the body, it may be

inferred that the lesion is central, such a combination of symptoms being generally met with in hysteria.

4. Diseases of the Optic Nerves.

a. Functional Affections of the Sense of Sight.

(1) Optic Hyperæsthesia and Hyperalgesia.

Optic hyperæsthesia consists of those conditions in which external objects are distinctly seen in an obscure light, or in which the acuteness of vision is greater than in ordinary sight. But disease usually gives rise to painful feelings or to abnormal psychical visual sensations, such as sparks of light and luminous disks, and these may be comprised under the name of *optic hyperalgesia*. Illusions and hallucinations of sight occur in insanity, epilepsy, and various other cerebral affections.

(2) Optic Anæsthesia.

Optic anæsthesia is characterized by diminution of vision or *amblyopia*, or by abolition of vision or *amaurosis*.

Etiology—The forms of amblyopia and amaurosis which occur in the absence of any apparent lesion of the fundus of the eye on ophthalmoscopic examination are those which interest us at present. Amblyopia or even amaurosis is sometimes caused by exposure to cold, venereal excesses, exhaustion, hæmatemesis, profuse menorrhagia, or other severe loss of blood, irritation of the sensory branches of the trigeminus, and irritation of the abdominal organs by the presence of worms, constipation, tumors, pregnancy, or uterine disease. An epileptic attack may be succeeded by a temporary enfeeblement of sight, which usually disappears rapidly, but may become more or less permanent when the attacks are frequently repeated. Unilateral blindness, lasting only for a few minutes, may occur in paroxysms in the midst of perfect health, and it may possibly be regarded as a vaso-motor epilepsy. Amblyopia may result from toxic agents such as lead, alcohol, tobacco, opium, belladonna, quinine, and santonin, or it may be a sequel of acute diseases, like typhus fever, scarlet fever, and pneumonia.

Symptoms.—The symptoms of functional amblyopia and amaurosis are the same as those which are observed in organic diseases of the optic nerves, except that in the former no changes are found in the fundus on ophthalmoscopic examination.

Vision presents four distinct alterations in amblyopia: (*a*) Diminution in the acuteness of vision; (*b*) Alterations in the field of vision; (*c*)

Disorders in the perception of light; and (*d*) Disorders in the perception of colors.

(*a*) *Diminution in the Acuteness of Vision.*—The patient sees objects through a mist; he has difficulty in distinguishing minute objects, or at times may observe a dark spot in the centre of vision.

Tests of the Acuteness of Vision.—The acuteness of vision is usually tested by asking the patient to read print of a certain size of type, and at definite distances. In Snellen's scale the size of type is numbered according to the distance in feet at which the print can be read by the normal eye in a good light. The acuteness of vision is expressed by a fraction of which the denominator is the number of the test type, and the numerator the distance in feet at which it can be read. The sight of each eye must always be tested separately.

(*b*) *Alterations in the Field of Vision.*—The field of vision may be altered in several ways, but the form usually observed in functional amblyopia begins at the margin of the field and progresses concentrically until only a small central area is left. The first loss of vision may appear in the centre of the field, constituting a *central scotoma*, while at times the whole field of vision is covered with *scotomata*. The blindness may sometimes be surrounded by spectral appearances and then it is named a *scintillating scotoma*. In their simplest form they consist of a blind area which is surrounded more or less completely by a luminous border, the latter widening as the former expands. This luminous arc is subject to a rapid oscillatory movement which has been variously described by different observers. In the more pronounced forms the luminous border assumes a zigzag outline which has been compared to the outlines of a fortification. It is also fringed by gorgeous colors which are in continual trembling movement, or appear to "coruscate," or to emit a "shower of sparks." The phenomenon lasts from a quarter to half an hour and then passes off.

Tests of the Field of Vision.—The most ready test of the field of vision is to direct the patient to fix one eye, the other being closed, on the corresponding eye of the operator, and the latter then moves his hand to the right, left, above, and below, and at a certain distance from the fixed point as a centre. If the field of vision be limited in any particular direction, the observer will have to approach his hand nearer and nearer to the point on which the patient's eye is fixed before it is seen, and thus any serious departure from the normal limit can be readily detected. If greater accuracy be required, the field must be measured by means of the "mapping system" or by the perimeter, for a description of which the reader is referred to ophthalmological works.

(*c*) *Disorders of the Perception of Light.* — There are several varieties of partial anæsthesia in which the ophthalmoscopic appearance may be negative. Sometimes the patient cannot see at night, a condition which is called *hemeralopia;* at other times sight is deficient in daylight, and this condition is called *nyctalopia.*

(*d*) *Disorders of the Perception of Colors* (*Dyschromatopsia, Achromatopsia*).—The perception of colors may be defective when the acuteness of vision is very little impaired, and, conversely, color vision may be little affected when there is considerable limitation of the field of vision. The area of the field of vision varies for each color, green having the smallest and yellow and blue the largest visible areas (Fig. 85).

FIG. 85.

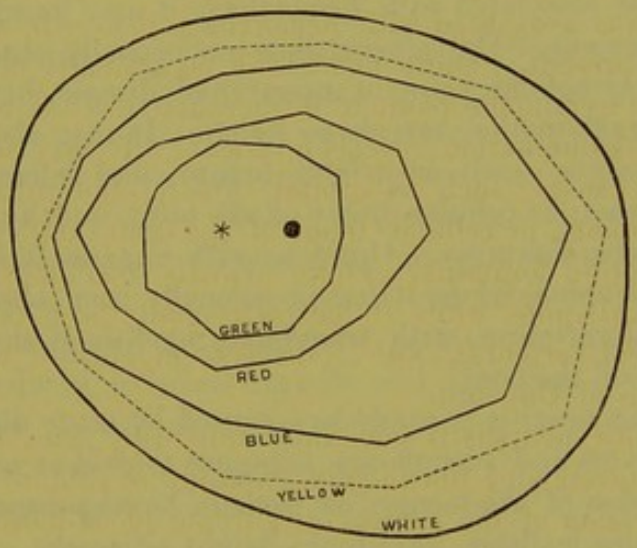


DIAGRAM SHOWING THE FIELDS OF COLOR VISION IN A NORMAL EMMETROPIC EYE ON A DULL DAY. (After GOWERS.)

The fields are each rather smaller than on a bright day. The asterisk indicates the fixing point, the black dot the position of the blind spot. (Usually the blue field is larger than the yellow.)

Tests of Color Vision.—A scale of colors is submitted to the patient and it must be ascertained whether he can identify and name each; or the patient is asked to match a given color from a number of others presented to him.

In amblyopia the order in which the perception of colors is lost is usually that in which the fields are arranged on the retina, the first defect being for green, then red, while yellow and blue are the last to be lost. The condition in which central vision for the perception of one or more colors is much restricted is named *dyschromatopsia*, while total color blindness is named *achromatopsia.*

Color blindness is sometimes a congenital defect, and of this form there are three varieties; namely, (1) Red blindness, (2) Green blind-

ness, and (3) Violet blindness. Dalton suffered from red-blindness; he imagined that diluted ink gave a color much resembling a florid complexion; blood appeared to him not unlike in color to that called bottle-green; and he could not distinguish between the color of a ripe cherry and that of a leaf.

b. Organic Diseases of the Optic Nerves.
Optic Neuritis and Atrophy.

Etiology.—Optic neuritis may be caused by tumors of the brain, hydrocephalus, basal meningitis, meningeal hemorrhages, and thrombosis of the cavernous sinus, but it only rarely results from abscess of the brain, and softening from occlusion of vessels, and in the few cases in which it was associated with hemorrhage it may be suspected that a glioma was present. Optic neuritis is occasionally observed in cases in which the symptoms point to intracranial disease, but in which the post-mortem examination reveals no lesion. It may also be caused by extracranial causes, such as orbital tumors, and inflammation of the bones, periosteum, or cellular tissue of the orbit, and hyperostosis narrowing the optic foramina. Optic neuritis sometimes follows injuries of the cervical spine, while it has occasionally been observed in caries of the cervical vertebræ, with transverse myelitis of the cervical and dorsal regions of the cord.

Double optic neuritis is sometimes caused by acute diseases, such as the specific fevers and pneumonia, exposure to cold or to a very bright light, suppression of the menses, or chronic blood-poisoning from lead, syphilis, diabetes mellitus, or chronic Bright's disease.

Varieties.—The diseases of the optic nerves may divided as follows:

(1) *Congestive* and *inflammatory affections:*
(*a*) Simple congestion of the disk.
(*b*) Congestion with swelling of the disk (optic neuritis).
(*c*) Albuminuric retinitis and neuritis.
(2) *Atrophic affections:*
(*a*) Simple or primary atrophy.
(*b*) Secondary atrophy.

(1) Inflammatory Affections of the Optic Nerve.

(*a*) *Simple congestion* is characterized by an increased redness of the disk, which is the more readily recognized when it is greater in one eye than in the other. The redness invades the physiological cup and may entirely obscure it; the sclerotic ring and the edge of the choroid are rendered indistinct, and the disk loses its sharpness of outline.

(*b*) *Optic neuritis* is characterized by œdema as well as congestion of the disk, which becomes enlarged, swollen, red, and cloudy, the physiological cup is obscured, the sclerotic ring and the edge of the choroid are concealed, and the edges of the disk are badly defined and hazy. As the disease advances the disk becomes more swollen, it assumes a reddish-gray color, and its periphery becomes distinctly striated, partly owing to the swelling and opacity of the nerve fibres and partly to an enormous development of minute vessels. The veins are engorged, tortuous, and often varicose, while the arteries are reduced in size, and appear paler than the veins. When exudation takes place the vessels become veiled and lost to sight at the border of the disk, but reappear partially as they proceed inwards, and disappear again before reaching the lamina cribrosa.

The inflammation may now subside, the swelling gradually diminishes, the edge of the choroid becomes apparent, and the only indication of the previous inflammation which remains may be a narrow zone of atrophy adjacent to the disk and along the edge of the choroid.

(j) *Engorged or Choked Disk.*—If the inflammation continues the disk becomes still more swollen, and its margins becoming steeper, the vessels which pass over the side become concealed by the edge of the swelling, and reappear in a different position in the fundus. The arteries are reduced to small threads and are often invisible, but the veins are visible towards the edge of the tumor and often appear distended and tortuous for a long distance from the disk. Hemorrhages are now frequent and extensive, and generally appear at the edge rather than on the surface of the swelling. Sight becomes, as a rule, rapidly impaired during this stage of strangulation. This form of neuritis has been called the *choked* or *engorged* disk.

(jj) *Subsidence of Optic Neuritis.*—After the strangulation has existed for some time the veins become less distended, the swelling loses its intense red color and becomes pale and less prominent, hemorrhages cease, the extravasated blood is absorbed, leaving pigmented or white spots on the retina, the edges of the choroid and the sclerotic ring become dimly apparent after a time, and then the disk assumes a whitish or grayish color, its edges being generally irregular and surrounded by a zone of choroidal atrophy.

(jjj) *Descending Neuritis or Neuro-retinitis.*—It is not often possible to distinguish during life between neuro-retinitis and the slighter degrees of engorged disk. In neuro-retinitis there is only a slight degree of swelling, the changes are more marked towards the edge than in the centre of the disk, hemorrhages are absent, white spots

may be seen scattered over the disk or along the edges of the vessels, and the disk has a striated appearance from atrophy of the nerve fibres.

(iv) *Retrobulbar Neuritis and Perineuritis.*—In retrobulbar neuritis the primary congestion soon passes on to atrophy with narrowed vessels. It is met with in periostitis of the orbit and in cases where the optic nerves are constricted by thickening of the cranial bones. Optic perineuritis results from chronic inflammation of the sheath of the nerve, which gives rise to thickening and purulent infiltration of the trabeculæ, and it generally ends in optic neuritis.

(v) *Albuminuric Retinitis and Neuritis.*—In diseases of the kidneys the arteries of the retina become diminished in calibre like the arterioles of the body generally. As the disease advances the retinal arteries are reduced to small lines, and when swelling is present they may be invisible beyond the edge of the disk, while the thickening of their walls causes white lines to appear along their edges. The arteries are liable to undergo aneurismal dilatations, and retinal hemorrhages form a marked feature of the affection. Albuminuric retinitis presents several varieties, viz., (*a*) degenerative, (*b*) hemorrhagic, and (*c*) inflammatory retinitis, to which may be added, according to Gowers, (*d*) albuminuric neuritis.

(*a*) *Degenerative Albuminuric Retinitis.*—This form, which is the most common, begins by the appearance of white spots on the retina; they are small and rounded at first, but after a time increase in size and become irregular in outline, while neighboring spots sometimes coalesce to form large white patches. Small white spots are generally to be seen around the macula lutea; they are sometimes so small as to be seen only on careful direct examination, but are at other times large and well marked, and arranged end to end, so as to form radiating streaks irregularly disposed around the macula, and giving to the retina at this part a silvery appearance.

(*b*) *Hemorrhagic Albuminuric Retinitis.*—The chief characteristic of this variety is the large number of hemorrhages which occur, and white spots only appear around the macula lutea at a late stage of the affection.

(*c*) *Inflammatory Albuminuric Retinitis.*—In this variety there is general swelling of the retina, the disk is obscured, the arteries are thready and numerous, the veins are distended and tortuous, with irregular outline, and large hemorrhages often occur, while the white spots are often numerous and well marked. On the subsidence of the inflammation the optic nerve may undergo secondary atrophy.

(*d*) *Albuminuric Neuritis.*—In this affection inflammation of the optic nerve predominates over the retinal changes, the edges of the

disk are indistinct and veiled under a grayish-red swelling, and the arteries are small and often hidden, while even the veins may be concealed in the swelling, and form curves over the sides of the swollen disk. White spots may be seen on the surface of the swollen disk, on the retina, and around the macula lutea, and small hemorrhages may be observed about the fundus, but are rare over the swollen disk. When the inflammation subsides a consecutive atrophy of the optic nerve may be left. Albuminuric neuritis cannot often be distinguished from ordinary optic neuritis.

General Symptoms.—Sight may remain unimpaired in advanced cases of optic neuritis, and when amblyopia is present its degree is by no means proportional to the amount of change observed on ophthalmoscopic examination. Vision is likely to fail sooner in descending neuritis than in cases of engorged disk, while a high degree of unilateral or bilateral amblyopia may precede for some time any changes in the fundus in retrobulbar neuritis.

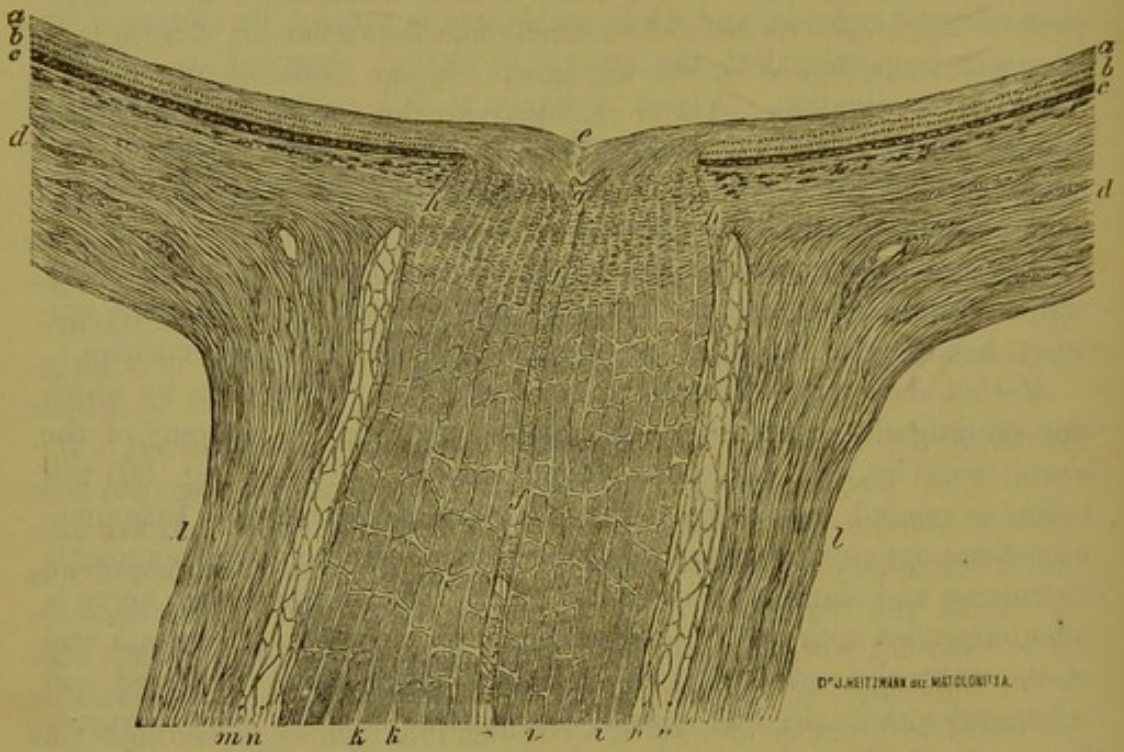
The cerebral symptoms most likely to be associated with optic neuritis are headache, vertigo, vomiting, loss of memory, unilateral epilepsy, hemiplegia, and paralysis of some or all of the ocular muscles.

Morbid Anatomy.—In order to understand the mechanism by which the swelling of the disk is caused in optic neuritis, the anatomy of the nerve must be kept in mind. The annexed diagram (Fig. 86) will suffice to remind the reader of the structure of the nerve. Inflammation of the optic nerve gives rise to a serous infiltration which augments its volume and diminishes its consistence. The sheath of the nerve is often distended with fluid, the connective tissue of the pial sheath and of the trabeculæ surrounding the nerve bundles becomes thickened and infiltrated with nuclei and cells, a considerable number of leucocytes is found surrounding the vessels, and the nerve fibres undergo degenerative changes, while in advanced cases the tissue of the *lamina cribrosa* is distended and its structure altered. The veins are large and tortuous, but the arteries are abnormally small.

Various theories have been advanced to account for the different forms of optic neuritis. Descending neuritis and perineuritis of the nerve are caused by a local disease and need not be further discussed. But the double optic neuritis, or choked disk, which is caused by the presence of an intracranial tumor does not find a ready explanation. It was suggested by Von Graefe that the increase of intracranial pressure which results from the presence of a tumor within the skull caused an obstruction to the flow of blood from the eyes by compressing the cavernous sinus. Many objections have been urged against this theory, and it was finally abandoned when Sesemann showed that the supraorbital vein

anastomoses so freely with the facial veins, that pressure on the cavernous sinus would only produce a very temporary effect. When it was discovered by Schwalbe that the subvaginal space around the optic nerve was continuous with the subdural space around the brain, Schmidt suggested that any increase of intracranial pressure would tend to distend the sheath of the optic nerve with fluid, and consequently would produce strangulation of the nerve fibres in their passage through the sclerotic ring and lamina cribrosa. This theory is by no means free

FIG. 86.



HORIZONTAL SECTION THROUGH THE OPTIC NERVE AT ITS POINT OF INSERTION IN THE GLOBE, AND ITS PASSAGE THROUGH THE MEMBRANES OF THE EYE. (From LANDOIS'S "Physiologie")

a, Internal; *b*, External layers of the retina; *c*, Choroid; *d*, Sclerotic; *e*, Physiological cup; *f*, Central artery of the retina; *g*, Point of its bifurcation; *h*, Lamina cribrosa; *l*, Dural sheath; *m*, subdural space; *n*, Subarachnoidal space; *r*, Arachnoidal sheath; *p*, Pial sheath; *i i*, Bundles of nerve fibres; *k k*, Connective-tissue trabeculæ.

from objections. Optic neuritis may be absent in very large tumors and in chronic hydrocephalus when the increase of intracranial pressure must be very great, while small cortical tumors which can scarcely be supposed to increase the pressure to any appreciable extent, often give rise to the most marked swelling of the disk. Dr. Hughlings-Jackson suggested that intracranial tumors act like foreign bodies, and produce optic neuritis by their irritating effects. Benedikt elaborated this theory still further. He supposed that the irritation of the tumor acts on the

optic nerves through the vaso-motor nerves, and this opinion is now known as the hypothesis of reflex vaso-motor action. In a later publication, Dr. Hughlings-Jackson maintains that the irritation of the tumor causes a discharge from the cortex of the brain, the first result of which is to occasion spasm of the vessels of the optic nerves, which is followed by a secondary paralysis of these, just as occurs in voluntary muscles subject to unilateral spasm from intracranial disease. He believes that the nutritive change which constitutes optic neuritis is caused by this secondary paralysis. I have myself suggested that optic neuritis is a trophic change caused by irritation of the external geniculate bodies which I believe to be the homologues of the spinal ganglia.

(2) Atrophic Affections of the Optic Nerves.

In atrophy of the optic nerve the intraocular extremity of the nerve becomes slowly and progressively transformed into a pure white, or grayish-white disk. There is complete cessation of the capillary circulation of the disk and consequently its healthy rosy tint disappears.

(*a*) *Primary Atrophy.*—This form of atrophy is generally associated with other symptoms of locomotor ataxia, and consequently Charcot called it *tabetic amaurosis*, or *parenchymatous atrophy;* on ophthalmoscopic examination the optic disk is seen to be of a pearly white color (white atrophy), occasionally mixed with a slight tinge of blue or of gray (gray atrophy). The contour of the disk is sharply defined, and it may maintain its normal size and round form for a long time, although its outline is occasionally irregular, and its size reduced. The central artery and vein maintain their normal volume and direction, but the lateral branches of the disk are in great part atrophied. White atrophy is usually bilateral, but it may sometimes remain limited for many years to one eye. The retina is quite normal throughout. The onset of the affection is usually slow, and the patient observes for months or years that his sight is becoming progressively enfeebled. The field of vision becomes concentrically contracted but in a very irregular manner, and a central scotoma may occasionally be associated with the peripheral limitation. Dyschromatopsia or achromatopsia is present at an early period of the atrophy, and the acuteness of vision becomes progressively diminished, but the diminution is not always in direct proportion to the degree of change in the optic nerve, inasmuch as the patient can sometimes read the smallest type when the atrophy is very advanced. The patient may experience various subjective sensations such as sparks or flashes of light (photopsia), or a play of colors (chromatopsia). The mode of locomotion is characteristic; the head is retracted and the chin elevated, the gait is shuffling, the eyes are directed

upwards, and the expression of the countenance is vague because the eyes are not fixed on any object. White atrophy is generally associated with various disorders of the pupil, of which paralytic myosis is the most frequent. The pupillary disorders are independent of the white atrophy, being caused by an extension of the lesion which underlies locomotor ataxia, to the cilio-spinal region of the cord.

(*b*) *Secondary atrophy* of the optic nerve is caused in various ways. The following varieties may be distinguished :

(*c*) *Atrophy by Compression of the Fibres of the Optic Nerve.*—This form may be caused by pressure on the chiasma or on the optic nerve in any part of its course through the base of the brain, optic foramen, or orbit. It is often preceded by a stage in which the disk is congested, but when atrophy is established the ophthalmoscopic appearances of the disk are the same as those of white atrophy. A lesion of one optic tract causes, but only after some years, pallor of the corresponding halves of the disk. The disease is steadily progressive and the prognosis is most unfavorable.

(j) *Atrophy Secondary to Optic Neuritis.*—The main features of this form of atrophy have already been described, when the subsidence of neuritis was under consideration. The disk is at first yellow or dull white, its contour is completely hidden under an exudation, and the vessels are varicose; but as the exudation becomes absorbed the disk becomes whiter and whiter, its capillaries undergo atrophy, and the central vessels themselves become smaller although they preserve their tortuous course. The disk is larger than normal, and its edge is irregular and broken and remains to some extent obscured by exudation, while patches of exudation are often seen around the disk and in the neighborhood of the macula. This form of atrophy is not always followed by blindness, and patients after becoming blind during the acute stage may recover some degree of vision.

(jj) *Atrophy Secondary to Obliteration of Vessels.*—This form of atrophy is caused by embolism or thrombosis of the central artery of the retina. The disk is of a pearly white color; its margin is covered with a white veil which extends to the retina, and the arteries are so small as to be scarcely perceptible and are often surrounded by a whitish and more or less opaque exudation. After the exudation has been absorbed this form of atrophy is distinguished from every other by the small size of the arteries of the disk and retina.

(jjj) *Choroiditic Atrophy.*—This form of atrophy is very similar to that which is consecutive to obliteration of the central artery; the retinal vessels are greatly atrophied, but the disk has a peculiar reddish or yellowish tint and its edges are slightly blurred.

(iv) *Atrophy Secondary to Retinitis Pigmentosa.*—In this variety of atrophy the retinæ become studded by pigmentary spots from migration of the choroidal pigment, and the central vessels are atrophied, while their collateral branches disappear, after a time, altogether. The capillary vessels derived from the ciliary arteries of the optic nerve are, however, not affected to the same extent, and consequently the disk generally preserves a well-marked rosy tint.

(v) *Atrophy by Excavation.*—This optic atrophy is caused by increase of intraocular pressure in such diseases as glaucoma and hydrophthalmia, and it is characterized by the deep excavation of the optic disk. A certain degree of excavation may occur in the other forms of atrophy from wasting of the fibres of the optic nerve and cicatricial contraction of the newly formed connective tissue.

Morbid Anatomy.—The anatomical changes in atrophy of the disk extend through the optic nerves. In primary atrophy the nerve is much reduced in size; it is gray and gelatinous in appearance; the connective-tissue trabeculæ are hypertrophied, and the nerve fibres are progressively destroyed, so that the nerve is ultimately reduced to a cord of connective tissue. In atrophy from pressure the nerve is much reduced in size and the connective tissue is much increased. In the various forms of atrophy the disk presents a superficial depression which does not usually pass beyond the limits of the choroid; the lamina cribrosa is only covered by a thin layer of the débris of the disk, but does not itself undergo displacement; and the ganglionic layer and the nerve fibres of the retina undergo atrophy, but the other layers are unaffected. The degeneration always ascends to the chiasma, and the optic tracts are atrophied, in long-standing cases, as far as the external geniculate bodies.

c. Diseases of the Optic Commissure and Tracts.

The lesions which usually affect the optic tracts and commissure are circumscribed affections of the bones and membranes of the brain or injuries of the base of the skull.

Symptoms.—The characteristic symptom of disease of the optic tract is an enfeeblement or abolition of sight of one lateral half of the retinæ. The blind half of the retina is separated from the sensitive half by a sharply defined vertical line, but central vision is preserved in both eyes. When this condition is described with reference to the field of vision it is called *hemianopsia*, and to the retina, *hemiopia*. If the left optic tract be compressed by a tumor (Fig. 87, K), the two left halves of the retinæ—the outer of the left and the inner of the right—are cut off from the cortex and consequently there is blindness of the

right halves of the fields of vision, a condition named *right lateral hemianopsia*. The affected portions of the retinæ are associated in their actions, and this condition has, therefore, been also named *equilateral or homonymous hemianopsia*. When both the inner or both the outer halves of the retinæ are blind, the condition is called *crossed hemianopsia*. When the lesion is situated over the centre of the commissure (Fig. 87, T) the inner halves of both retinæ are affected. With reference to

FIG. 87.

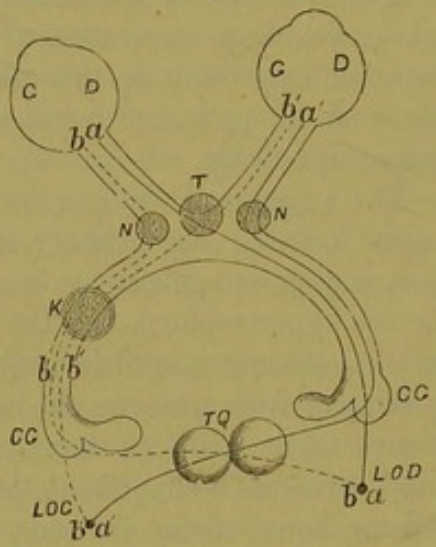


DIAGRAM OF DECUSSATION OF THE OPTIC TRACTS. (After CHARCOT.)

T, Semidecussation in the chiasma; TQ, Decussation of fibres posterior to the external geniculate bodies (CG); *a' b*, Fibres which do not decussate in the chiasma; *b' a'*, Fibres coming from the right eye, and coming together in the left hemisphere (LOG); K, Lesion of the left optic tract producing right lateral hemianopsia; A, Lesion in the left hemisphere (LOG), produces crossed amblyopia (right eye). T, Lesion producing temporal hemianopsia; NN, Lesion producing nasal hemianopsia.

the fields of vision this condition is called *double temporal hemianopsia*. In this defect patients experience difficulty in walking, but they are often able to read the smallest print. *Double nasal hemianopsia* is rare, and can only result from a double lesion (Fig. 87, N N).

V. DISORDERS CAUSED BY DISEASE OF THE CEREBRO-SPINAL SENSORY CONDUCTING PATHS.

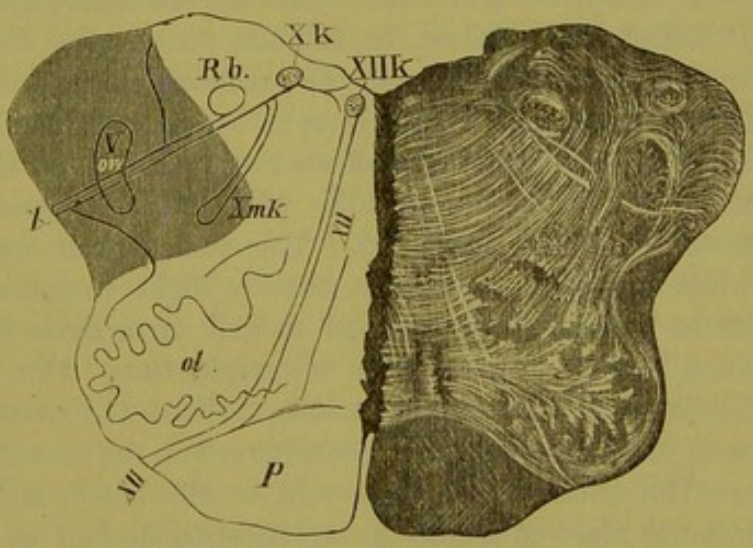
Various sensory disorders are caused by disease of the fibres of the posterior roots after they join the spinal cord, but inasmuch as these fibres are affected most frequently in posterior sclerosis, the resulting disorders will be considered along with tabes dorsalis.

Paranæsthesia.—In transverse lesions of the spinal cord the part of the body which is innervated from that portion of the cord which lies below the level of the lesion is more or less anæsthetic, according to the completeness of the destruction of the cord produced. A hyper-

æsthetic belt is frequently met with surrounding the body on a level with the upper limit of the lesion, caused by irritation of fibres descending from the posterior roots which are situated immediately above the level of the lesion.

Hemiparanæsthesia.—In destructive lesions of one lateral half of the spinal cord there are, on the side of the lesion, motor paralysis and loss of muscular sense below the level of the lesion from destruction of the fibres of the pyramidal tract, cutaneous hyperæsthesia of the parts below the level of the lesion from irritation of the sensory conducting paths of the opposite half of the spinal cord, an anæsthetic belt on a level with the lesion from implication of the posterior roots, and a hyperæsthetic belt above this level from irritation of fibres descending from the posterior roots immediately above the lesion. On the side opposite to the lesion there is anæsthesia of the sensations of touch, pain, tem-

FIG. 88.



SECTION OF THE MEDULLA OBLONGATA ON A LEVEL WITH THE LARGEST DIAMETER OF THE DISEASED FOCUS, WHICH IS REPRESENTED BY THE SHADED PART ON THE LEFT HALF OF THE DIAGRAM. (After SENATOR.)

P, pyramids; *ol*, olivary bodies; *Vom*, ascending root of the fifth nerve; *Xk*, nucleus of the vagus; *Xmk*, motor nucleus of the vagus; *Rb*, fasciculus rotundus; X, the vagus; XII, the hypoglossal nerve.

perature, and tickling of the parts supplied from below the upper limit of the lesion, from destruction of the sensory conducting paths after they have crossed over to the opposite side of the cord, and a zone of hyperæsthesia immediately above the level of the upper limit of the lesion caused by irritation of fibres descending from the posterior roots immediately above the lesion, and some of which probably cross over to the diseased side of the cord. In unilateral lesions of the lumbar or the cervical enlargements a zone of anæsthesia is found in the lower or upper extremities which varies in its position according to the level of the lesion, and the root or roots which are involved in it.

Crossed Hemianæsthesia.—In this affection there is anæsthesia of the face on one side of the body, and of the trunk and extremities on the opposite side. In a case of this kind reported by Senator the lesion consisted of a spot of softening in the restiform body, and implicated the ascending root of the fifth nerve (Fig. 88). It must be remembered that this root begins as low down as the third cervical nerve, so that some degree of anæsthesia of the side of the face may be observed in unilateral lesions of the upper portion of the cervical region of the cord.

Hemianæsthesia.—In this affection there is loss of sensibility of the whole of one-half of the body, face, and extremities, including the accessible mucous membranes as well as the skin. The abolition of sensation is sometimes incomplete, and then cutaneous analgesia or thermo-anæsthesia may be present, while tactile sensibility remains unaffected. At other times the anæsthesia of the skin and mucous membranes is complete, and even muscular sensibility and muscular sense are abolished. The patient, for instance, does not feel deep pressure, strong contraction of the muscles may be produced by the faradic current without causing pain, and when his eyes are closed he is unable to describe the position in which the affected extremities may be placed by passive movements, and is not aware when his attempted voluntary movements are forcibly prevented. The patient can walk without difficulty when his eyes are closed, but by slight pressure upon the affected side he may be easily induced to walk in a circle while under the impression that he is walking in a straight line. One half of the mucous membrane of the tongue, mouth, veil of the palate, and the conjunctiva of the same side are insensitive, but the cornea retains its sensibility. The affected side feels cold to the touch, and the prick of a pin does not bleed so readily as on the opposite half of the body.

The cutaneous reflex actions may be lost on the affected side while the deep reflexes are retained.

The *senses of taste and smell* are abolished on the affected side.

The *sense of hearing* is diminished, and in some cases there may be complete unilateral deafness.

The *sense of sight* is impaired, but not abolished. There is concentric restriction of the field of vision, and the perception of colors is diminished or lost (*dyschromatopsia*), these defects being present in both eyes, but most marked on the side opposite the lesion.

Hemianæsthesia as just described occurs in its most typical form in hysteria and occasionally in epilepsy, chorea, neurasthenia, and other grave neuroses. It also occurs in organic diseases when the lesion is situated in or near the posterior third of the posterior segment of the

internal capsule. Hemianæsthesia is generally present in hemichorea and athetosis, the lesion being situated in these diseases either in the external and posterior part of the optic thalamus or in the posterior part of the lenticular nucleus. In these diseases the posterior part of the internal capsule is only partially injured and the degree to which sensory conduction is impaired varies greatly in different cases. When the lesion is situated on the lenticular nucleus the various forms of cutaneous and muscular sensibility are affected, but the special senses often remain intact. The special senses may also remain unaffected when the posterior part of the internal capsule is compressed by the growth of a tumor in the optic thalamus, probably because the slow growth of the tumor enables the fibres of the optic radiations of Gratiolet to be pressed aside without rupture.

Hysterical hemianæsthesia is associated, as we have just seen, with concentric restriction of the fields of vision of both eyes, and especially with amblyopia of the eye on the same side as the other sensory disorders. In order to account for this fact, Charcot assumed that all the fibres which come from one eye are connected with the cortex of the opposite hemisphere, and he supposed that for the fibres which did not cross in the commissure there was a supplementary crossing in the corpora quadrigemina (Fig. 87, T Q). But that part of the scheme which relates to the supplementary crossing is now, I believe, abandoned by its distinguished author, because a considerable number of cases are now recorded in which homonymous lateral hemianopsia has been caused by disease of one cerebral hemisphere, the lesion being situated in such cases on the pulvinar of the optic thalamus, the white substance of the occipital lobe, or the cortex of the occipital lobe as far forwards as the angular gyrus. From an analysis of recorded cases it would appear that the fibres of the optic tract pass through the geniculate bodies and anterior tubercle of the corpora quadrigemina, and are continued upwards through the pulvinar of the optic thalamus, the posterior part of the internal capsule, and the posterior part of the corona radiata, to reach the cortex of the occipital lobe. In homonymous hemianopsia caused by disease of one optic tract, central vision is retained in both eyes, a fact which proves that the macula lutea of each eye must be connected with the corticles of both hemispheres. As the organs of vision are bilaterally associated in their functions, it is probable that the cortical visual centres are connected by commissural fibres through the corpus callosum.

VI. DISORDERS CAUSED BY DISEASE OF THE CORTICAL SENSORY CENTRES.

1. Cutaneous and Muscular Hemianæsthesia.

Ferrier localizes the centre of tactile sensibility in the hippocampal region, but unilateral lesions of these convolutions are not known to give rise to anæsthesia. Disease of the temporo-sphenoidal lobe sometimes causes loss of tactile sensibility of the opposite side, but it is most probably caused by interference with the sensory fibres of the internal capsule. Cutaneous anæsthesia and loss of muscular sense have occasionally been found associated with hemiplegia in cases of extensive softening of the cortex of the parietal lobe, and it is, therefore, probable that the cortical centre of general sensibility is widely diffused over the so-called motor area of the cortex.

2. Disorders of the Sense of Smell.

a. Hallucinations of Smell.—The case of a woman is reported by Dr. McLane Hamilton, who had for nearly thirty years suffered from occasionally recurring epileptiform attacks which were always ushered in by an aura of a disagreeable odor. The patient died of phthisis, and at the autopsy the pia mater was found adherent over the right uncinate gyrus and adjacent convolutions, while the subjacent gray matter was atrophied and in a state of sclerosis. The olfactory nerves were not involved in the lesion.

b. Anosmia.—Ferrier found that destruction of the *subiculum cornu ammonis* in monkeys caused loss of smell on the side of the injury, but in *hemianæsthesia* from disease of the fibres of the posterior part of the internal capsule the loss of smell is on the side opposite the lesion. Anosmia of the left nostril is sometimes met with in cases of right hemiplegia and aphasia, but the loss of smell is most probably caused by softening of the external root of the olfactory tract and not from disease of the cortex. A case of abscess of the temporo-sphenoidal lobe is reported by Dr. Glynn, in which the most prominent symptom was complete anosmia, but the loss of smell was most probably caused by compression of the olfactory tracts at their points of junction with the brain. On the whole, our clinical information with regard to the localization of the cortical centre of smell is exceedingly scanty and will not bear a critical investigation.

3. Disorders of the Auditory Sense.

a. Hallucinations.—Ferrier places the auditory cortical centre in the first and second convolutions of the temporo-sphenoidal lobe, and irritative lesions of these convolutions give rise occasionally to hallucinations of hearing. A man, under my care, received a blow over the head a little above the left ear, and at the seat of injury, which was opposite the first and second temporo-sphenoidal convolutions, a slight depression in the bone could be detected. After the injury the patient suffered from recurring epileptiform attacks which were ushered in by loud rattling noises, caused most probably by a discharging lesion having its origin in the auditory cortical centre.

b. Psychical Deafness.—In this condition, which was first described by Goltz as occurring in dogs after portions of the cortex of the brain had been washed away, the animal is not deaf, but fails to recognize the significance of the usual calls. He does not, for example, respond to the call of his name, and is not cowed by angry threats, and yet distinct evidence is obtained that he hears noises. This defect was named psychical deafness by Munk. The state in man most nearly approaching to it is *word-deafness*, which will be subsequently described along with the disorders of speech.

c. Absolute Deafness.—Unilateral destructive lesions of the first and second temporo-sphenoidal convolutions do not give rise to complete deafness of one ear, because the sense of hearing is bilaterally associated, and so long as one hemisphere is unaffected the auditory sense remains unimpaired or only slightly weakened. The condition which will be subsequently described as word-deafness is, however, associated with disease of the first and a portion of the second temporo-sphenoidal convolutions. But if the first and second temporo-sphenoidal convolutions contain the auditory centres, bilateral lesions of these ought to give rise to complete deafness. A case is reported by Wernicke in which the patient first suffered from word-deafness and subsequently became completely deaf, and after death the first temporo-sphenoidal convolution was found softened in each hemisphere. A careful dissection of the ears proved the absence of any local disease of the peripheral organ of hearing.

4. Disorders of the Visual Sense.

a. Hallucinations of Sight.—Tumors of the occipital lobe give rise to hallucinations of sight such as colored vision, and the images of animals and of variously dressed men, and when the growth causes epileptiform convulsions the attacks are often preceded by a visual aura.

b. Psychical Blindness.—This condition was, like psychical deafness, first observed by Goltz in dogs after portions of the cortex of the brain were washed away. The dog, after the operation, sees and avoids obstacles, thus recognizing resistance and the other fundamental properties of matter, but he has lost the power of appreciating the special properties of matter to so great an extent that he does not recognize his food as such. A more or less similar condition has been described by Fürstner as occurring in some cases of general paralysis of the insane. The patient, for example, recognizes that a piece of money placed in his hand is a metal, but has lost all knowledge of the special properties which constitute it a coin. *Word-blindness* is a more or less similar condition. Subsequent dissection has proved that in the dogs operated upon by Goltz, it was the posterior lobes of the brain which were chiefly denuded of cortex by his method of procedure, and in Fürstner's cases the cortex of the occipital lobes was always found diseased when a post-mortem was obtained. It will be hereafter seen that in word-blindness the disease is limited mainly to the cortex of the angular gyrus.

c. Homonymous Bilateral Hemianopsia.—Many cases are now recorded in which this defect of sight was caused by softening of the cortex of the left angular gyrus, and the condition is frequently associated with word-blindness.

d. Absolute Blindness.—Disease of the cortex of one hemisphere of the brain does not usually give rise to complete blindness. The case of a boy, however, is reported by Dr. Abercombie, who, after an injury which caused a depressed fracture of the right parietal bone, suffered from hemiplegia of the left side and amaurosis of the left eye, from which he made a quick recovery on the depressed part being removed. The position of the fracture rendered it probable that the angular gyrus was injured. A case is reported by Dr. Glynn, of Liverpool, in which the patient became suddenly and completely blind, and in which a clot was found occluding the posterior cerebral artery of the left side, causing extensive softening of the left occipital and temporo-sphenoidal lobes. The case of a woman is reported by Dr. Shaw, of Brooklyn, who had become suddenly aphasic and perfectly deaf and blind. At the autopsy the angular gyri and superior temporo-sphenoidal convolutions of both hemispheres were found completely atrophied, and no other lesions could be discovered in the brain or peripheral organs. This case would seem to indicate that bilateral disease of the angular gyri will alone suffice to cause complete blindness. Berger has recently recorded two cases of complete blindness with normal or almost normal reaction to light of the pupils and in which extensive softening was found in the occipital lobes.

Nothing is known of disorders of the sense of taste from cortical disease.

Treatment.—Inasmuch as anæsthesia and hyperæsthesia are generally associated with other important phenomena such as motor paralysis, the treatment of these symptoms does not require to be specially discussed in this place. In the treatment of neuralgia the first indication is to remove the exciting cause. An endeavor must be made to remove every source of external irritation such as carious teeth, foreign bodies pressing upon the nerves, cicatrices, tumors, and neuromata. It is manifest that in order to fulfil this indication great scope is afforded the exercise of surgical skill. At other times the exciting cause of a neuralgia is to be found in remote irritation such as when the presence of intestinal worms or uterine disease gives rise to facial neuralgia. Venous congestion of the pelvic organs from constipation and portal obstruction is sometimes the cause of sciatica, and in these cases benefit is likely to be obtained from purgatives and the use of natural saline waters like those of Kissingen, Marienbad, and Harrogate. A turpentine enema has also been strongly recommended in such cases. The early stages of neuralgia which arises in rheumatic and gouty subjects must be treated by absolute rest, diaphoresis, the milder counter-irritants, the vapor or Turkish bath, and salicylate of soda or alkalies with colchicum, and by iodide of potassium either alone or in combination with guaiacum, when the acute symptoms have subsided. When the patient is suffering from anæmia, as often occurs in trigeminal neuralgia, large doses of carbonate of iron, with or without cod-liver oil, have a very beneficial effect. In such cases arsenic either alone or combined with iron is a useful remedy, and it is also useful in ophthalmic neuralgia of malarial origin. In malarial neuralgia, however, large and repeated doses of quinine are the most trustworthy remedy and it also may be employed in the treatment of other forms of neuralgia, and is especially useful in trigeminal neuralgia. In neuralgia of syphilitic origin large doses of iodide of potassium must be given, and this drug will also be useful whenever there is reason to suspect the presence of neuritis. Zinc, nitrate of silver, chloride of gold and sodium, and strychnia, are other drugs which have occasionally been found useful in the treatment of neuralgia. Gelseminum sempervirens, best given in the form of tincture, and croton chloral hydrate, either in one large dose of a scruple or in four grain doses every four hours, have been found useful in the treatment of trigeminal neuralgia, and Dr. Ringer speaks favorably of a liquid extract of tonga prepared by Messrs. Allen & Handbury, given in drachm doses every four hours. In sciatica, rectified oil of turpentine has been found useful. It is best administered in gelatine capsules each of

which contains fifteen drops of the oil, and two or three of these may be given at meal-time. Copaiba has succeeded in the hands of Dr. March, of Rochdale. The local treatment of neuralgia consists of the application of hot fomentations and poultices in the early stages, while flying blisters may be applied along the course of the painful nerve when the acute symptoms have subsided. When the nerve is deep-seated like the sciatic its course may be painted with tincture of iodine, or the actual cautery may be used in chronic and obstinate cases. In such cases a very effectual but very severe remedy consists in the subcutaneous injection over the seat of severest pain of a few minims of a strong solution of nitrate of silver (gr. x to ℥j). An injection, by means of a subcutaneous syringe, into the substance of the nerve of a one per cent. solution of perosmic acid has been favorably reported upon by Eulenburg and others. Ointments containing opium, veratria, aconitia, or equal parts of chloral and camphor, may be rubbed into the skin over the painful nerve, or the course of the nerve may be painted with aconite or belladonna liniment, or rubbed with narcotic and soothing liniments. Chloroform is also found useful as a local application, and its inhalation may be advisable to allay the pain of very violent paroxysms of neuralgia. Chloral and camphor when rubbed together in a mortar form a clear solution which is an excellent local application in neuralgia of superficial nerves.

The constant current has been found very successful in the treatment of obstinate neuralgia. In neuralgia of isolated superficial branches the direction method may be employed and a descending *stabile* current passed through the painful nerve. When the polar method is used, the anode is to be placed on the specially painful points and held stationary there, whilst the cathode rests on the back of the neck, sternum, or on any other indifferent part of the body. When the deeper-seated nerves are affected, the anode may be placed on some indifferent part of the body and the cathode over the point of emergence of the affected nerve. In order to reach the main divisions of the fifth nerve at the base of the cranium, and after their emergence through the foramen of the sphenoid bone, the current may be conducted transversely through the base of the skull at the appropriate spots, the anode being placed on the painful side. Benedict recommends that in severe cases of trigeminal neuralgia galvanic currents should be passed longitudinally and transversely through the skull, and along the sympathetic nerve. Faradization occasionally answers better than the constant current, the moist poles being applied to the painful points and along the nerve trunks. The electric hand is sometimes very soothing and agreeable to the patient.

In the treatment of sciatica the anode should be placed over the sciatic foramen or upon the sacrum, and the cathode upon the specially painful parts. Remak advises that successive portions of the nerve, from six to eight inches in length, should be successively brought under the influence of the current, beginning at the sacrum and passing down to the feet. Another method recommended by Remak, under the name of circular current, consists in the stabile application of the anode upon the painful points and over the trunk of the nerve; broad electrodes and strong currents being used. In severe cases Benedict recommends that one electrode should be introduced into the rectum and the other placed over the sacrum, so that the current may pass through the plexus. Ciniselli recommended a zinc and copper plate connected by a wire, to be applied to the affected limb and worn continuously. In chronic cases I have obtained excellent results by introducing several acupuncture needles over the course of the nerve as it emerges from the sciatic foramen, and passing a feeble galvanic current through them, the positive pole being in contact with the needles and the negative placed over the sacrum. The faradic current gives the best results when the pain has in great measure disappeared and the subsequent muscular feebleness has to be treated, or if more or less anæsthesia be present the faradic brush may be employed.

In order to afford immediate relief to the pain the administration of narcotics forms an indispensable part of treatment. The most generally effectual narcotic is morphia administered by subcutaneous injection. Another useful method of employing morphia is to dust from one-sixth to one-third of a grain every three or four hours over the raw surface left when the cuticle is removed by the application of a blister over the course of the painful nerve. It may also be useful to change sometimes from morphia to stramonium, hyoscyamus, or atropine. In the treatment of epileptiform trigeminal neuralgia Trousseau recommended the use of large doses of opium or morphia, and in some cases he prescribed as much as a drachm of morphia, or two or three of opium in the course of the day; but this treatment ought only to be resorted to when every other method fails. Large doses of bromide of potassium, either alone or in combination with tincture of opium or chloral, act beneficially sometimes in this form of neuralgia. In intractable cases of neuralgia relief for the pain, when all other methods fail, is only to be obtained by means of surgical operations. Resection of the painful nerve, and even amputation of the affected limb, have been resorted to in intractable cases, but such serious operations ought only to be resorted to under the most pressing necessity. Stretching of the affected nerve is a much less serious operation, and several cases of neuralgia

of different nerves are now on record in which the operation has proved successful. Lange procured relief of pain in sciatica by producing forcible flexion of the thigh, and thus stretching the affected nerve over the neck of the femur.

The form of plantar neuralgia described by S. Weir Mitchell has hitherto proved intractable to every kind of treatment. The posterior tibial nerve was stretched in one of my patients by my colleague, Mr. Southam, but the relief which followed was not lasting.

Coccygodynia, in addition to the usual remedies, must sometimes be treated by means of a surgical operation. The usual operations are extirpation of the coccyx, and separation of the bone from all the nerves connected with it by means of a tenotomy-knife introduced subcutaneously.

Disorders of the special senses come under the care of the physician as symptoms of other diseases of the nervous system, and the treatment must be directed against the chief lesion, whatever it may be. The possibility of the sensory affection being of syphilitic origin should never be forgotten. Local electrical treatment is sometimes found useful in those cases in which the acuteness of any of the special senses is diminished.

The treatment of those sensory disorders which are caused by disease of the sensory conducting paths and cortical centres, must vary according to the nature and locality of the lesion. Local treatment is seldom of any use.

CHAPTER III.

SPASMODIC DISORDERS.

The general characteristics of the various forms of spasm have already been considered and we shall now describe in detail (I.) the spasms which are caused by reflex or direct irritation of the reflex spinal mechanisms and which may be named spino-neural spasms, and (II.) the spasms which are caused by irritation of the cortical motor centres or of the fibres of the pyramidal tracts, and which may be named cerebro-spinal spasms. It is not always possible to make a trenchant division between these two forms of spasm. Some forms of writer's cramp, for instance, belong probably to the spino-neural, and others to the cerebro-spinal paralyses, and even some local spasms like masticatory spasm may be caused either by reflex irritation or by irritation of the cortex of the brain, but it will be found convenient to describe all forms of these affections in the same place. Notwithstanding the difficulties which present themselves in carrying out this division of spasm, the importance of the distinction which underlies the classification must be our justification for adopting it.

I. SPINO-NEURAL SPASMS.

The spino-neural spasms may be divided into: 1, local spasms affecting muscles supplied by particular nerves or their branches; 2, general spasms caused by excessive irritability of the spinal reflex mechanisms; and 3, spasms of myopathic origin. Amongst the spino-neural spasms tetanus and tetany are here included, but we are by no means sure that a more extended knowledge of the pathology of these affections will justify their retention in this category.

1. Local Spasms.

a. Spasms of the Muscles of the Eyeball.

The external muscles of the eyeballs are subject to both tonic and clonic spasms.

(1) *Tonic spasm* of the internal rectus is more frequent than spasm of any other muscle of the eyeball, and it is usually associated with

blepharospasm. It gives rise to homonymous diplopia similar to that which occurs in paralysis of the sixth nerve, but in the latter the squint is permanent and the relative positions of the true and false images fixed, while in the former there is a constant oscillation between the two images which alternately approach and recede from each other. All cases of spasmodic strabismus are accompanied by paroxysms of neuralgia, and by photophobia and lachrymation. Tonic spasm of the internal rectus may be caused by long-standing paralysis of the external rectus.

(2) *Tonic spasm of the external rectus*, which is rare, causes divergent squint, and gives rise to diplopia with crossed images, but contrary to what occurs in paralysis the two images never remain fixed, but alternately approach and recede from each other.

(3) *Nystagmus* consists of a clonic spasm of the muscles of both eyeballs, giving rise to continual lateral oscillatory or rotatory movements which are entirely beyond the control of the patient. When the patient looks at a remote object the trembling becomes very pronounced, but the eyes become more and more fixed in proportion as they are directed to near or small objects. Nystagmus may be caused by congenital cataract, corneal opacities, and other local diseases, but it is very generally due to central changes and is a prominent symptom in sclerosis in patches, and in Friedreich's hereditary form of locomotor ataxia, while it may arise in connection with meningitis, hydrocephalus, and other intracranial diseases. It is often present in albinos, and colliers are frequently affected.

b. Masticatory Spasm (Trismus).

Etiology.—Bilateral masticatory spasm is often a symptom of general spasms, such as tetanus, epilepsy, hysteria, and chorea. When it occurs as a separate affection it may be caused by disease of the motor nerves themselves, or be a symptom of basal meningitis, apoplexy, intracranial tumors, and other central diseases. It may also result from reflex irritation either of the sensory branches of the fifth nerve or of some remote part of the body.

Symptoms.—The spasm is generally bilateral and it may be either *tonic* or *clonic*.

In the *tonic* variety the lower is approximated to the upper jaw, and when the teeth are so firmly clenched that they cannot be separated from one another by force the condition is named lock-jaws or *trismus*. The muscles of the jaw are then tense, rigid, often painful, and mastication is impossible. In the *clonic* variety the lower jaw is moved either in a vertical or horizontal direction, the former giving rise to chattering of

the teeth, as in the cold stage of ague, and the latter to grinding and munching of the teeth. The teeth are much worn by the constant grinding against each other, and they may even be broken by the violence of the spasm.

c. Spasm in the Area of Distribution of the Facial Nerves (Histrionic Spasm, Mimic Convulsion, Convulsive Tic).

Etiology.—Convulsive tic sometimes appears to be inherited, and it may occur in neuropathic subjects from emotional causes, or in the absence of any recognizable cause. It is caused frequently by direct or indirect irritation of the facial nerve. The direct causes are often only a slighter degree of those which occasion paralysis of the nerve, such as exposure to cold, tumor of the base of the brain or in the lower end of the pons, aneurism of the vertebral artery, inflamed glands in the neighborhood of the stylo-mastoid foramen, abscess of the parotid gland, caries of the petrous portion of the temporal bone, and otitis. Reflex irritation is often conveyed through the fifth nerve and the causes of irritation are trigeminal neuralgia, carious teeth, and irritation of the eyeball and conjunctiva. The source of irritation is, however, sometimes remote, the spasm having been caused by such diseases as cervico-brachial neuritis, and intestinal or uterine disease.

Symptoms.—Facial spasm may be *clonic* or *tonic*, the former being by far the more frequent.

The *clonic* form is characterized by periodically recurring attacks of spasm, each paroxysm consisting of sudden and violent contractions and relaxations of some of the muscles of the face. The duration of each paroxysm varies from a few seconds to a few minutes, and the interval between them is generally brief, except during sleep, when the spasms entirely cease. The spasm is generally unilateral, and thus the contortions and grimaces of the affected side contrast strangely with the calm expression of the healthy side. The contortions produced are extremely variable and consist mainly of elevation and depression of the occipital and frontal muscles, corrugation of the eyebrows, twitching or winking of the eyelids, elevation of the cheek and of the nostril, and distortion of the angle of the mouth, these symptoms being present either singly or in every imaginable combination. The spasm is sometimes *partial*, being limited to the muscles supplied by single branches of the nerve. The palpebral twigs alone are sometimes affected, and the spasm then manifests itself by a rapid winking called *nictitating spasm*. In some cases there is only slight twitching movements of one of the eyelids, the lower being the one most usually

affected. At other times the spasm gives rise to a paroxysmal closure of the eyelids which may last from a few minutes to a few hours, and may occasionally extend over a period of weeks or months, this form being named *blepharospasm*. This form is accompanied by photophobia, and is generally caused by reflex irritation of the fifth nerve. In some other cases the spasm is limited to the area of distribution of the malar and labial branches, and then it gives rise to a convulsive grin on one or both sides, resembling laughing, and consequently this form is called the *sardonic laugh*, *risus caninus*, or *cynic spasm*. All the muscles supplied by the facial nerve, including the platysma myoides, may be involved, and the spasm sometimes extends to the territories of other nerves, such as the fifth, hypoglossal, spinal accessory, and upper branches of the brachial plexus.

Sensory disorders never form a prominent feature of the disease, but the patient may occasionally suffer from numbness or even a slight degree of pain in the side of the face, and he may also complain of headache and *tinnitus aurium*. Vaso-motor and secretory disorders are never present, the electrical reactions of the affected muscles are normal, and all the voluntary movements of the face can be executed with undiminished power. Certain points may be discovered, pressure on which will arrest the spasm; these, when present, correspond with the pressure point in trigeminal neuralgia.

The *tonic form* of facial spasm was first clearly recognized by Dr. Marshall Hall. In this form the muscular contraction is persistent, the tip of the nose, the angle of the mouth, and the chin are drawn to the affected side, and the furrows and dimples of that side are rendered deeper. The contracted muscles render the tissues too scanty for covering the orifices, so that when the eye is closed a tightness is induced at the angle of the mouth, and when the angle of the mouth is moved further from the eye, as in speaking, it is difficult to keep the eye closed, articulation is rendered indistinct, and the bolus of food tends to collect between the teeth and the affected cheek, causing slight trouble during mastication, but there is no difficulty of deglutition.

d. Spasm of the Muscles Supplied by the Hypoglossal Nerve (Lingual Spasm, Aphthongia).

Etiology.—Lingual spasm is rare as an independent affection, but is a common symptom of hysteria, chorea, epilepsy, and eclampsia, while it often accompanies spasm of the muscles supplied by the fifth nerve. Spasm of the tongue may also result from meningitis, and lesions of its cortical motor centre, and it is frequently met with in bilateral athetosis

and the spasmodic paralyses of infancy, while tremor of the tongue is a symptom of progressive bulbar paralysis, and disseminated sclerosis.

Symptoms.—In lingual spasm the muscles innervated by the hypoglossal nerve are thrown into a state of clonic or tonic convulsions when the patient attempts to speak, the affection being more or less similar to writer's cramp, and the spasms implicating groups of other muscles engaged in effecting special movements. In some cases the tongue becomes fastened to the hard palate by a tonic spasm whenever the patient tries to speak, but in other cases it is attacked with clonic spasms, and the sterno-hyoid, thyro-hyoid, and sterno-thyroid may likewise be implicated in the convulsion.

e. Spasm of the Muscles Supplied by the Vagus and Pharyngeal Plexus.

(1) Spasm of the Soft Palate, Pharynx, and Œsophagus.

(*a*) *Spasm of the soft palate and Eustachian tube* is not well known as a natural disease, but noises in the ears can be induced in healthy persons by the application of the faradic or galvanic current to the end of the Eustachian tube, which are caused most probably by contraction of the tubal muscle. Twitching movements of the palate have occasionally been observed in advanced cases of paralysis agitans.

(*b*) *Spasm of the constrictors of the pharynx* usually accompanies acute pharyngitis and spasmodic stricture of the œsophagus, while it is a prominent symptom of hydrophobia.

(*c*) *Œsophagismus*, or spasmodic stricture of the œsophagus, is frequently met with in hysterical women, and some believe that *globus hystericus* is the subjective correlative spasm of the œsophagus. It may be so severe and persistent in some cases of hysteria as to simulate organic stricture, and the patient may be reduced to the verge of death by inanition. It is sometimes caused by irritation in the peripheral course of the nerves, or in a reflex manner in cases of ulcer of the tube, but it is rare as a result of central disease.

(2) Spasmodic Affections of the Larynx.

(*a*) spasm of the glottis in infants or internal convulsions (laryngismus stridulus).

Etiology.—The predisposing causes of spasm of the glottis in infants are the same as those which tend to produce eclampsia, and, indeed, the two diseases frequently coexist. Spasm of the glottis usually attacks children between four and ten months of age; it is more fre-

quent in boys than in girls; and badly nourished, cachectic, or rachitic children are almost exclusively affected. The exciting causes are exposure to cold, the irritation of teething, or the presence of parasites or other irritating substances in the alimentary canal. It may sometimes be caused by direct irritation of the laryngeal mucous membrane from excessive crying.

Symptoms.—The attack generally begins suddenly without premonitory symptoms, and inasmuch as the action of the adductors predominates over that of their antagonists the glottis is completely closed during the continuance of the spasm. The attack is characterized by a sudden arrest of respiration; the chest becomes fixed; the face is turgid and of a blue color; the countenance expresses great anxiety; the mouth is widely open, as if to make a deep inspiration; the head is drawn backwards; the eyes are staring; the action of the heart is tumultuous and irregular; and the infant appears to be on the verge of suffocation, then the spasm relaxes. The paroxysm lasts from two to twenty seconds, and the end is announced by a series of sonorous inspirations, as if air were drawn through a narrow reed. The last inspiration becomes longer and more sonorous than the rest, and then the respirations assume their natural rhythm. During the paroxysm the spasm involves the extremities, causing flexion of the hand and extension of the feet, and occasionally the infant is seized with general epileptiform convulsions at the end of the attack. The disease may be limited to a single paroxysm, but it usually consists of a series of paroxysms, coming on at irregular intervals for many days or weeks. The mortality of the disease is very great, death occurring suddenly by suffocation during an attack, or from nervous exhaustion from frequently repeated attacks.

(*b*) SPASM OF THE GLOTTIS IN ADULTS.

Spasm of the glottis in adults may be caused by local disease of the larynx, the inhalation of irritating gases, the entrance of morsels of food into the larynx, or irritation of one of the recurrent laryngeal or pneumogastric nerves. It may also be caused by central diseases of the nervous system, like hysteria. Paroxysms of hysteria generally culminate in a deep stridulous inspiration and opisthotonos. On laryngoscopic examination during an attack of spasm the mucous membrane may be found to be healthy or slightly congested, and the vocal cords may be seen separating for an instant, and then becoming spasmodically closed.

(*c*) SPASM OF THE TENSORS OF THE VOCAL CORDS (APHONIA SPASTICA).

This condition does not interfere with the respiratory function, but the voice is rendered feeble, jerky, and intermittent. The patient may speak a few words in a normal voice, but the current of the voice is soon partially interrupted, and the sound becomes like the suppressed voice of a person engaged in some act requiring a straining effort, like the act of defecation.

(*d*) SPASMODIC LARYNGEAL COUGH.

The cough caused by spasm of the laryngeal muscles is a shrill or barking cough of metallic quality, which comes on in paroxysms and lasts for many hours, only ceasing when the patient sleeps. This cough occurs in young girls, but is occasionally met with in boys. The paroxysmal cough of whooping-cough is caused by a direct or reflex irritation of the laryngeal nerves. The attack begins with a deep inspiration, which is followed by a succession of short and frequently repeated coughs with no intervening inspirations. When the chest is contracted to the utmost, and the patient seems on the verge of asphyxia, another deep inspiration is taken, during which the air, rushing through the spasmodically closed glottis, makes a characteristic whistling, crowing, or whooping noise.

(*e*) TABETIC LARYNGEAL CRISES.

The course of locomotor ataxia is liable to be complicated by paroxysms of spasmodic cough, not unlike those of whooping-cough, and which have been named *laryngeal crises*. These attacks may be divided into three varieties according to their degree of intensity: (*a*) the mild; (*b*) the medium; and (*c*) the severe crises.

(*a*) The mild attacks are like paroxysms of whooping-cough, the duration of each varying from a few seconds to about ninety.

(*b*) The medium form is characterized by a more violent cough and a louder and more difficult inspiration, the duration of the attack being from five to ten minutes.

(*c*) The severe form is manifested by symptoms of asphyxia, unconsciousness, and epileptiform convulsions, and the attack may last from half an hour to several hours.

The attacks vary greatly in their periods of recurrence; they are more frequent during the day than during the night, and they may sometimes be repeated as often as fifty times in the twenty-four hours. The attack may be excited by a current of air, sudden exertion, or a

slight catarrh, or it may come on suddenly, and without warning, in the middle of a conversation or during sleep. At other times the patient may feel a scalding sensation, or a feeling as if a foreign body were lodged in the larynx, immediately before the attack.

(*f*) CLONIC SPASM OF THE LARYNGEAL MUSCLES (CHOREA OF THE LARYNX).

In some cases the laryngeal muscles contract and relax, during attempts at phonation in such a way that the glottis is alternately closed and opened in an irregular manner, so that the power of inflecting the voice is diminished. Gerhardt reports the case of a flutist who was not able to play his instrument without a continuous noise being caused in his throat, and he believes that the affection is analogous to professional spasms in the hand. The laryngeal muscles are subject to clonic spasms in chorea, and in bilateral athetosis, while they are occasionally affected by incoördinate movements in ataxia.

f. Spasm in the Region of the External Branch of the Spinal Accessory Nerve (Wry-neck, Caput Obstipum Spasticum).

Etiology.—The relatives of those suffering from wry-neck are often subject to hysteria and other nervous affections; the male sex is rather more frequently affected than the female, and adults than young or old people. The most usual exciting causes are excessive exertion, emotional excitement, exposure to cold, irritation of remote organs, while it sometimes comes on suddenly without assignable cause.

Symptoms.—Spasms of the muscles supplied by this nerve may be divided into, *a*, *tonic* and, *b*, *clonic* wry-neck.

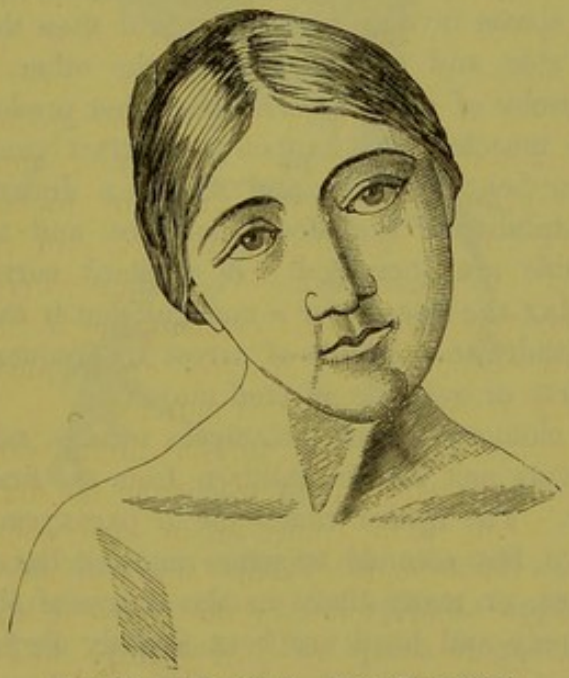
(*a*) The *tonic* form is almost exclusively confined to one of the sterno-cleido-mastoid muscles, but a part of the trapezius is sometimes affected. In this form of the disease the head is rotated so that the occiput is approximated to the shoulder of the affected side; the ear of the same side is drawn towards the clavicle; and the chin is turned upwards and towards the opposite side. In chronic cases a spinal curvature with its convexity to the sound side occurs in the cervical region, and compensatory curves in the dorsal and lumbar regions.

When the trapezius alone is the seat of the spasm the head is drawn strongly backwards and is inclined to the opposite side; there is no turning of the chin; the point of the shoulder is elevated; and on any attempt being made to bend the head forwards the muscle becomes tense and painful (Fig. 89).

(*b*) The *clonic* form of the disease may be either *unilateral* or *bilateral.*

The *unilateral* variety begins with uneasiness in the neck, and it is soon noticed by the patient or his friends that the head is not straight. As the disease advances the patient complains of a dull aching pain in the course or at the insertion of the muscle, and when the spasm is limited to one of the sterno-cleido-mastoid muscles, the head becomes rotated obliquely to one side by a succession of jerks, the occiput being turned towards the shoulder and depressed, and the chin elevated in the opposite direction. The muscles on the side to which the head is drawn are hard, contracted, and frequently hypertrophied. When the trapezius is affected, the head is bent back and the shoulder raised in the manner already described. At the end of a short time, generally a

FIG. 89.



SPASM OF THE TRAPEZIUS. (From DUCHENNE)

few seconds, the muscle relaxes and the head returns to the normal position, but this is soon followed by a second contraction and a second rotation. At first there may be a considerable interval between the contractions, but as the disease advances the interval is shortened, and ultimately the contractions may number twenty or thirty in the minute. In an early stage the patient may arrest the spasm by a voluntary effort, or counteract it by a voluntary contraction of the muscles of the opposite side, but in long-standing cases the voluntary is overpowered by the involuntary contractions, and the head is habitually twisted to one side, except, indeed, when the patient counteracts the deformity by holding his head between both hands, or placing it against a resisting

object. Patients are distressed by wakefulness caused by constant movements of the head against the pillow, but as sleep is approaching they feel the movements becoming gradually less, and the spasm generally ceases entirely during sleep.

The disease is rarely confined to the muscles supplied by the spinal accessory nerve, and it is even probable that the splenii and obliqui capitis are often the first attacked, and they are at least generally implicated. When the scaleni are affected œdema and some degree of anæsthesia of the corresponding arm may be present from compression of the brachial plexus and veins. Facial tic and masticatory spasm may be associated with clonic spasmodic torticollis, and in aggravated cases the spasm extends to the muscles supplied by the cervical and brachial plexuses. The clonic form of the disease is generally unilateral, but occasionally the spasm invades both sides, and then the head is rotated first to the one side, and after a time to the other, according as the action of the muscles of one side or of the other predominates. Occasionally the two muscles may happen to contract simultaneously, and the head is then bent forwards and the chin drawn to the sternum. The faradic irritability of the affected muscles, and the faradic sensibility of that side are increased. A constant current of moderate strength may relax the spasm for a moment, but it soon returns when the current is withdrawn. Points of arrest are frequently found in the course of the nerve or over the affected muscles.

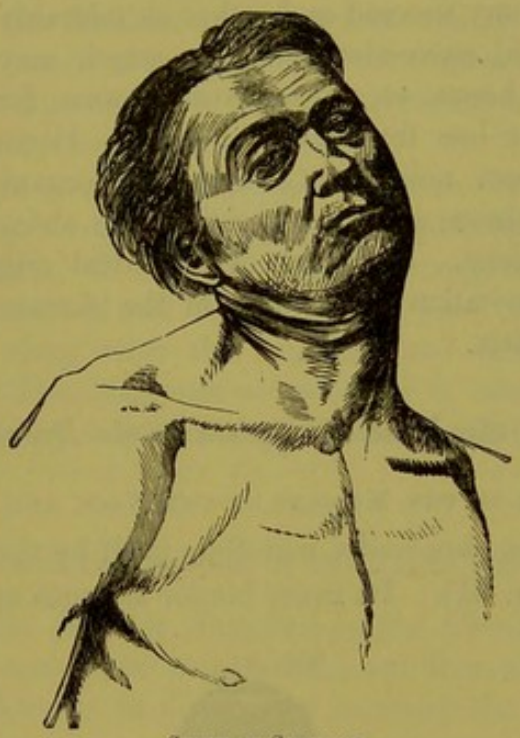
The *bilateral* clonic wry-neck (eclampsia nutans, salaam convulsion) is almost exclusively met with in children from the first period of dentition to puberty. The spasm comes on in paroxysms, each of which lasts only from a few seconds to some minutes, but generally recurs two or three times, or many times in the course of the day. During the attack the body and head are bent slightly forwards, and this is followed by almost instantaneous relaxation, to be succeeded after an interval of a few seconds, by a second bowing of the head, and so on until the paroxysm ceases after from twenty to one hundred alternate contractions and relaxations. The spasm in the neck has frequently associated with it some degree of facial spasm, blepharospasm, strabismus, or a slight convulsive movement of one or other arm, and at other times attacks of general convulsions intervene, so that the case becomes manifestly one of ordinary epilepsy. During the attack the child seems bewildered, but there is no complete loss of consciousness, and as soon as the movements cease the patient may be quite bright and happy, but occasionally the attack is followed by exhaustion and drowsiness.

g. Spasm of the Muscles supplied by the Cervical Plexus.

(1) Spasm of the Muscles of the Neck.

(*a*) *Spasm of the splenius capitis* causes the head to be drawn backwards and towards the affected side (Fig. 90), and the chin to be some-

Fig. 90.



Spasm of Splenius

what depressed and directed towards the affected side, while a hard ridge can be felt at the point where the splenius appears beneath the anterior border of the trapezius. The spasm is generally tonic with remissions and occasional more energetic contractions.

(*b*) *Spasm of the obliquus capitis inferior* causes either intermittent or persistent rotation of the head around its vertical axis without elevation of the chin or depression of the mastoid process.

(*c*) *Spasm of the deep muscles of the neck* is characterized by strong backward rotation of the head when the affection is bilateral, or towards the affected side when it is unilateral.

(2) Spasm of the Diaphragm.

(*a*) *Tonic spasm of the diaphragm* causes so much dypsnœa that the patient is threatened with asphyxia. The lower half of the chest is expanded and immovable, and the epigastrium is made to project strongly, whilst rapid and superficial respirations are effected with the

upper part of the chest. There is severe pain in the epigastrium and along the attachments of the diaphragm, the voice becomes feeble and muffled, there is well-marked cyanosis, and the attack proves fatal if it last beyond a short time.

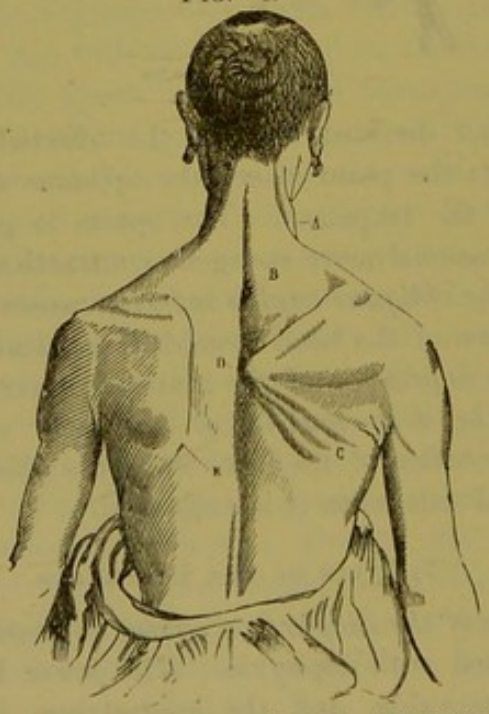
(*b*) *Clonic spasm of the diaphragm, singultus or hiccough*, consists of short energetic contractions of the diaphragm accompanied by an inspiratory sound, which is usually suddenly arrested by closure of the glottis. The contractions may succeed each other at tolerably long intervals or may occur in rapid succession, and the attack may last from a few minutes to many hours, or, in aggravated cases, for weeks, and they may recur more or less frequently for years. Hiccough is caused by reflex irritation from neighboring or remote organs, and it is to be regarded as an ominous sign in cancer of the abdominal viscera and in cachectic conditions. It is often of hysterical origin, and it may be caused by direct irritation of the roots of the phrenic nerves from cervical pachymeningitis.

h. Spasm of the Muscles supplied by the Brachial Plexus.

(1) Spasm of the Muscles of the Neck and Trunk.

(*a*) *Spasm of the rhomboidei* manifests itself by the peculiar position of the scapula (Fig. 91). Its inner border assumes an oblique position

Fig. 91.



Contraction of Rhomboid Muscles. (Duchenne.)

A, Levator scapulæ; B, Retracted rhomboid; C, Fibres of serratus magnus; D, Abnormal position of the inferior angle of the scapula; E, Inferior angle on the healthy side.

from above downwards and from within outwards, the lower angle is drawn upwards and approximated to the vertebral column, the muscle can be felt as a firm swelling between the scapula and spinal column, and it offers a distinct resistance when an attempt is made to raise the arm to a vertical position.

(*b*) *Spasm of the Levator Anguli Scapulæ.*—The upper and inner angle of the scapula is strongly elevated, the head is slightly inclined to the same side, the shoulder is drawn somewhat forwards, the supra-clavicular fossa is increased in depth, and the contracted muscle projects distinctly beneath the anterior border of the trapezius, which can easily be isolated from it by faradization.

(*c*) *Spasm of the serratus magnus, latissimus dorsi, the teres major and minor, the supra- and infraspinati, the subscapularis, or the pectoralis major*, is rare. It may be readily recognized by the position assumed by the scapula and arm, and the kind of interference with their movement, along with the hardness and fulness felt over the affected muscle. The diagnosis is aided by a comparison with the deformity produced by faradization of the corresponding muscles of the opposite side.

(2) Spasm of the Muscles of the Upper Extremity.

(*a*) *Spasm of the Muscles Supplied by the Circumflex Nerve.*—In the tonic form of spasm the arm is held out from the body, and it is also directed backwards in those cases in which the posterior fibres of the deltoid and teres minor are affected. The lower angle of the scapula is pressed backwards towards the vertebral column, as in paralysis of the serratus magnus. In cases of clonic spasm of the deltoid the arm is thrown upwards and moved convulsively in various directions, but other muscles are usually implicated at the same time.

(*b*) *Spasm of the muscles supplied by the musculo-cutaneous nerve* causes some resistance to passive abduction of the arm, and strong flexion of the forearm.

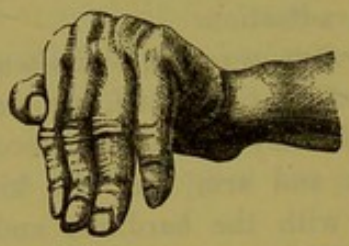
(*c*) *Spasm of the muscles supplied by the musculo-spiral nerve* causes extension of the forearm at the elbow, of the hand at the wrist, of the fingers at the metacarpo-phalangeal articulations, and of the thumb at both the metacarpo-phalangeal and phalangeal articulations, as well as supination of the forearm.

(*d*) *Spasm of the Muscles Supplied by the Median Nerve.*—In spasm of these muscles the forearm is strongly pronated; the hand is bent towards the radial side; the fingers are flexed; and there is apposition of the thumb. Spasm of the muscles of the hand supplied by the

median produces apposition of the thumb, with approximation and slight flexion of the first phalanges of the index and middle fingers.

(*e*) *Spasm of the Muscles Supplied by the Ulnar Nerve.*—In spasm of these muscles the hand is rendered concave; the thumb is adducted; the little finger is strongly flexed and opposed; and the remaining fingers are moderately flexed at the metacarpo-phalangeal and extended at the phalangeal articulations. The position assumed by the hand in spasm of the interossei is shown in Fig. 92.

Fig. 92.



Position of the Hand in Spasm of the Interosseous Muscles. (After Gowers.)

(*f*) *Writer's Cramp and Allied Affections* (*Graphospasmus, Mogigraphia, Professional Hyperkineses*).—Writer's cramp is only one of a large group of affections which have been called *professional hyperkineses*. This name has been given to them because the spasm affects muscles engaged in delicate associated and acquired actions, such as those required for writing, piano-forte playing, sewing, etc.

Etiology.—Writer's cramp is most frequently met with in men, while piano-forte player's spasm occurs most frequently in women. Professional spasms of all kinds are liable to occur in nervous subjects and neuropathic families. The spasm may be caused by exposure to cold, injuries of nerves or muscles, and foreign bodies in the fingers, or it may be caused in a reflex manner by periostitis of the external condyle of the humerus. The main cause, however, of writer's cramp is excessive writing, and consequently it is most frequently observed in secretaries, clerks, and merchants. The other professional cramps are also caused in the same way by the excessive use of particular groups of muscles.

Symptoms.—Writer's cramp may be divided into (1) the spastic, (2) the tremulous, and (3) the paralytic forms.

(1) In the *spastic* form of the disease some of the muscles of the hand are seized with tonic or clonic spasms whenever the patient tries to use the pen and this gives rise to an irregular stroke in the writing. The patient may, indeed, be quite unable to write while holding his pen in the usual position, but he is often able to do so tolerably well by holding it in a new and more or less grotesque manner. After a time

the spasm becomes stronger; the thumb and first finger may be suddenly extended so that the pen drops, or there is a spasmodic action of the opponens pollicis, with abduction and flexion of the index-finger, and the pen is thus rapidly moved away from the paper. At other times there is spasmodic flexion of the first three fingers, which then become pressed tightly against the pen, so that it cannot be moved further onwards; or there may be movements of pronation and supination in the forearm, and the pen is raised from the paper, and moved backwards and forwards in the most irregular manner. The faradic contractility of the affected muscles is sometimes increased, and at other times diminished, the former probably indicating an early, and the latter a late stage of the affection.

(2) In the *tremulous* form of the disease the hand and forearm, or even the whole arm, are the subjects of well-marked tremors on any attempt at writing, so that the pen only makes undulating or angular strokes. The writing is altered in character; the strokes are coarse, imperfect, and unequal, and numerous irregularities and false strokes are to be observed, while in the highest degrees of the affection the writing becomes a mass of undulating and zigzag strokes, and wholly illegible.

(3) In the *paralytic* form the patient experiences great fatigue and weakness of the hand and forearm on attempting to write, but as soon as the pen is laid down the feeling of weakness and exhaustion disappears, to reappear when it is taken up again.

In those who suffer from writer's cramp the movements requisite for sewing, piano-forte playing, embroidery, buttoning-up the clothes, and all actions requiring delicate manipulation are also impaired; and if the patient has learned to write with his left hand, the spasm, to his great disappointment, frequently extends to it also.

The most common sensory disorder is an undefined feeling of straining and fatigue, a sensation of pressure in the affected muscles, a painful drawing of the nerves in the direction of the trunk, or a feeling of coldness in the whole arm.

The pain frequently extends to the shoulder and back, and some of the spinous processes of the cervical and dorsal vertebræ may be tender to pressure.

Other spasmodic disorders are not infrequently associated with writer's cramp, the most frequent of these being strabismus, stammering, and spasms of the face, throat, and other parts of the body, while weakness and tremors of the lower extremities occasionally occur. In addition to the mental depression caused by failure of the hand the patient is often of an anxious disposition, and subject to general nerv-

ousness, which makes the writing worse when executed in the presence of a spectator.

The following are a few of the other professional hyperkineses:

Piano-forte player's spasm is not uncommon in professional players, especially women, and the spasm is in every respect similar to that of writer's cramp.

Telegraphist's cramp has been met in France with the use of Morse's machine, in which the letters are represented by an association of dashes and dots.

Violin-player's spasm occurs either in the form of painful exhaustion and stiffness, or as a convulsive spasm of some of the muscles of the hand, arm, or shoulder. It may attack the right or left hand, and it renders playing impossible.

Tailor's and shoemaker's spasm is of the same kind as the other professional spasms, and as soon as the patient begins to work, tonic or clonic spasms, or functional debility of the muscles of the hand and arm, are experienced.

Spasmodic movements have been described as occurring in smiths, milkers, painters, makers of artificial flowers, harp-players, turners, and watchmakers.

i. Spasms of the Muscles supplied by the Dorsal Nerves.

(1) Spasms of the Respiratory Muscles.

(*a*) *Inspiratory spasm* differs considerably from hiccough, although the latter frequently complicates the former. The essential feature of the affection is that many or all of the muscles participate in the spasm, and that a true inspiration, unbroken by sudden closure of the glottis, takes place. The spasm consists of a more or less rapid succession of deep inspirations, whilst the intervening expirations are performed in the usual noiseless way. The chest is powerfully expanded, the epigastrium is protruded, the auxiliary muscles of respiration are excited to action, the pectoral and sterno-cleido-mastoid muscles are brought into strong relief, the shoulders are raised, the head is drawn backwards, and the respiratory muscles of the face, ala nasi, and eyelids enter into strong contraction. Inspiration is noisy, and often accompanied by eructations of gas from compression of the stomach. The spasm usually occurs in paroxysms of variable duration, the abdomen is generally tympanitic, and there are, as a rule, other symptoms of nervous derangement, especially those characteristic of hysteria.

(*b*) *Attacks of sneezing* (*sternutatio convulsiva*) occur in a paroxysmal and spasmodic form, so that the patient will sometimes sneeze

several hundred times in succession. Ordinary sneezing is a reflex act excited by irritation of the nasal filaments of the fifth nerves. Attacks of sneezing are generally accompanied by a profuse watery secretion from the nose, and, when of long duration, they are productive of great misery.

(*c*) *Attacks of yawning* (*oscedo*, *chasme*) consist of a succession of yawns following one another with greater or less rapidity, and are accompanied by the well-known phenomenon of gaping, flow of saliva, increased flow of tears, and diminution in the acuteness of hearing, with dull tinnitus aurium.

(*d*) *Spasmodic cough* comprises all those paroxysmal attacks of coughing which are accompanied by a loud ringing sound. Such attacks of coughing may last for a variable period, and may also recur frequently for months or years.

(*e*) *Fits of laughing or crying* are a form of expiratory spasm, the former consisting of a succession of loud expirations accompanied by vocal tones, and the latter of long-drawn expirations, often interrupted by sobs and generally by a profuse secretion of tears. These actions are accompanied by well-known mental states, and by characteristic facial expressions. In pathological conditions they may be independent of emotional disturbances, and they then constitute subordinate symptoms of several general neuroses, such as hysteria, or are produced by disease of the central nervous system.

(*f*) *Spasm of the Dorsal and Abdominal Muscles.*—The dorsal muscles are the subjects of a strong tonic spasm in tetanus, and sometimes in hysteria, while they are affected by both tonic and clonic spasm in attacks of epilepsy and eclampsia. They may also be affected by spasm in cases of lateral spinal sclerosis, and various other central affections. The abdominal muscles are also affected by spasm in central nervous affections, and they are the subjects of spasm in biliary calculi and all painful affections of the abdominal organs. In spasm of the muscles of the back the body is strongly arched backwards, a condition which has been named *opisthotonos*, or when the action of the muscles of one side predominates it is arched backwards and to one side, a condition named *pleurosthotonos*.

In spasm of the abdominal muscles the walls of the abdomen become hard, tense, and resisting, and as it is generally accompanied by spasm of the diaphragm, the breathing is of the upper costal type, and becomes accelerated and shallow.

k. Spasm of the Muscles supplied by the Lumbar and Sacral Nerves.

(1) *Spastic contracture of the hip* consists of a tonic spasm of the psoas and iliacus, the quadratus lumborum, and occasionally of one or two of the muscles of the front of the thigh. The extremity is flexed at the hip-joint, the tendon and muscular belly of the ilio-psoas muscle project strongly, the pelvis appears to be raised on the affected side, the limb is shortened, and the patient inclines to the affected side in walking. Any attempt to extend the limb causes acute pain in the tense muscles and in the knee. This affection is caused by disease of the lumbar vertebræ with psoas abscess, and by reflex irritation in disease of the hip-joint.

(2) *Spasm of the quadriceps extensor femoris* gives rise to rigid extension of the leg on the thigh, and is observed in tetany and neuralgia of the knee-joint. Clonic spasm of the extensors of both sides was observed in a case in which no obvious cause could be traced. The spasms recurred at brief but irregular intervals, and gave a peculiar hopping character to the gait.

(3) *Contracture of the adductors* of both thighs approximates the knees together, and is a symptom of lateral sclerosis of the spinal cord. It also occurs in tetany, and is probably caused sometimes by reflex irritation from disease of the hip-joint, or from genital irritation.

l. Spasm of the Muscles supplied by the Sciatic Nerves.

(1) *Spasm of the flexors of the leg* causes the leg to be flexed on the thigh, and in aggravated cases the heel is brought in contact with the buttocks. It occurs in hysteria, diseases of the knee-joint, and spinal diseases.

(2) *Spasm of the anterior muscles of the leg* is of rare occurrence. A painful contracture of the tibialis anticus, peroneus longus, and gastrocnemius is described by Dr. S. Weir Mitchell as occurring in young people after long standing, and leading to deformity of the feet. Spasm of the peroneus longus plays an important part in the production of certain forms of club-foot, and is, according to Duchenne, of two kinds, the first consisting of persistent contraction of the muscle: the second of a functional spasm which occurs only when the leg is brought into use. The latter form is particularly liable to occur in congenital spasmodic affections.

(3) *Spasm of the muscles of the calf* produces talipes equinus, in which the heel is strongly elevated and the toes depressed. The well-

known "cramps" of the calf belong to this variety, and it occurs in sciatica and painful affections of the knee-joint.

(4) *Diffused spasm of the muscles of the lower extremity of one or both sides* occurs in hysteria, as a motor aura in epilepsy, and in spinal diseases.

2. General Spino-neural Spasms.

a. Saltatory Spasm.

Etiology.—In all cases of this disease either a decided neuropathic disposition has been clearly traceable or the patients themselves have been subjected to depressing influences before the attack. The male is as frequently attacked as the female sex, and the ages of those affected range from ten to seventy years.

Symptoms.—In this affection singular spasms of the muscles of the lower extremities arise when the sole of the foot is placed upon the ground, which have the effect of throwing the patient repeatedly in the air. These spasms continue so long as the patient maintains the erect posture, and they cause the patient to hop and jump on the floor, and render him quite unable to stand still for an instant. In some cases the feet are thrown off the ground at every spring, and in others the hopping movements are caused by quickly alternating elevations and depressions of the heel, while in one of two cases reported by Gowers, the springing movements were made from the hip and knee-joints. The movements disappear when the patient sits or lies down, but in most cases they can be made to reappear by pressing on the soles of the feet. The onset of the symptom is, as a rule, more or less sudden, but in a few cases premonitory symptoms such as a sense of weight, tearing pains, or slight tremors, have been observed. The spasms may disappear in a few days or last for many weeks, and they may recur after a perfectly free interval. The distribution of the spasms varies greatly, they are at times limited to the legs, while at other times they extend to the muscles of the back, face, neck, and pupils, but the arms always remain unaffected. Emotional excitement has sometimes been found to aggravate the spasm and at other times to arrest it.

b. Tetanus.

Etiology.—Tetanus may be of *traumatic* or *idiopathic* origin. Traumatic tetanus, which is by far the more important and frequent variety, is caused by punctured, lacerated, or contused wounds, and may even

result from contusions without external abrasion, but is rare after incised wounds. The extent and severity of the wound do not appear to bear any direct relation to the frequency of tetanus, and in some cases the primary wound is healed and forgotten when the symptoms make their appearance. The interval between the injury and the development of tetanus varies; the average duration is from four to fourteen days, but the symptoms may begin in a few hours or be delayed many weeks after the injury. Various causes may coöperate with the external wound in the production of tetanus, the most potent of these being exposure to cold and damp after the patient has undergone great excitement and fatigue like that caused by a great battle. The absence of antiseptic dressings and other improper treatment seem to increase the liability to tetanus.

The most usual cause of idiopathic tetanus is exposure to cold and damp, more especially when the patient is warm and perspiring. It may occur in the course of pleurisy, peritonitis, and other acute diseases. Malaria appears to give rise occasionally to an intermittent tetanus which may be cured by quinine. Strychnine and other toxic agents cause symptoms resembling tetanus.

Symptoms.—Premonitory symptoms are generally observed in tetanus, consisting of shivering or a distinct rigor, sensation of dragging in the neck, stiffness in certain muscles, difficulty of articulation and deglutition, and yawning. In traumatic cases the wound may become sensitive, and the patient complain of shooting pains radiating from it. These symptoms may occur a few hours or even a few days before the characteristic tonic spasms make their appearance.

The spasms, as a rule, begin in the muscles of the jaw. At first the jaws can be separated, and the movements of chewing and swallowing be accomplished, although with difficulty. Soon, however, the jaws become firmly clenched, constituting the condition called *trismus;* swallowing of even a small quantity of fluid is difficult and fatiguing owing to spasm of the œsophagus; while articulation is indistinct, and the voice altered, partly from the difficulty of moving the tongue, and partly from implication of the muscles of the larynx.

Spasm of the facial muscles gives to the countenance a characteristic expression. The angles of the mouth are drawn outwards, being at the same time depressed or elevated, and the lips are often drawn apart and expose the set teeth, so that the face assumes a sneering expression, named the "*risus sardonicus.*" The nostrils are dilated; the eyes are staring and motionless; the pupils are generally contracted; the brow is wrinkled; and all the lines of the face become strongly marked, and give to the patient an aged appearance.

The spasm rapidly extends to the muscles of the back of the neck,

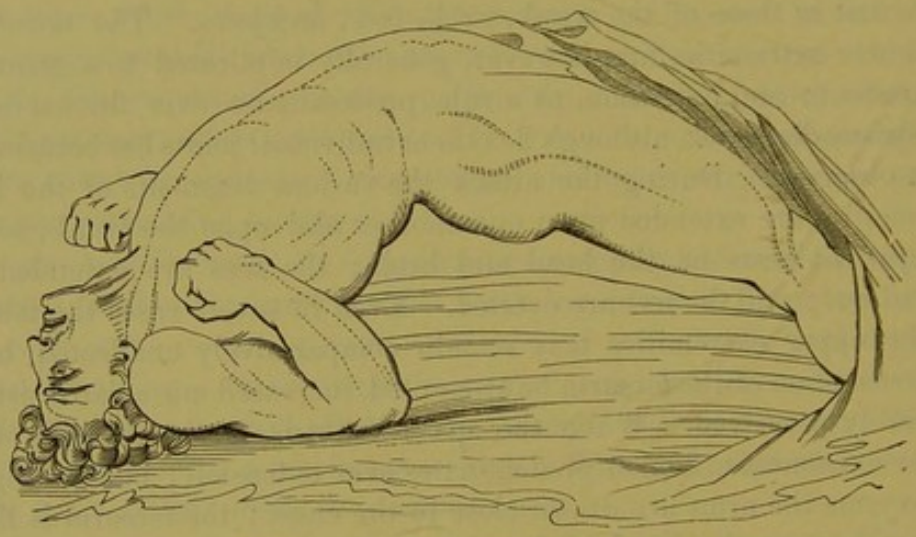
causing retraction of the head; while the erectore spinæ soon become implicated, and the vertebral column is then arched backwards; the chest is projected forwards and rendered very broad; and the body rests on the back of the head and sacrum, constituting the condition called *opisthotonos*. The epigastrium is sunk, and the abdomen flattened, while the hardness assumed by the abdominal muscles is characteristic. On rare occasions the body is said to be bent forwards, the convexity of the arch being directed backwards, a condition named *emprosthotonos*. In a few cases the body is maintained in a rigid attitude without being curved in any direction, a condition named *orthotonos;* and in some rare cases it is curved laterally—*pleurosthotonos*.

The muscles of the extremities are usually not affected to so great an extent as those of the trunk, neck, face, and jaws. The muscles of the lower extremities are, however, generally implicated to a greater or less extent; and extension, as a rule, predominates over flexion during the spasmodic attack, although flexion at individual joints has occasionally been observed. During the attack the various segments of the lower extremities are extended upon one another and upon the trunk, so that the patient rests on the head and heels; the toes are extended and spread out; and the feet are everted. Even in severe cases the muscles of the upper extremities may remain comparatively unaffected, but if passive motion of the forearm be attempted, increased muscular resistance is readily perceived. When the spasm extends to the muscles of the upper extremities, flexion predominates over extension; and during the paroxysms the arms are drawn close to the chest; the forearm is flexed upon the arm; the hand is flexed at the wrist; and the fist is closed, the palm being directed towards the upper arm (Fig. 93).

In some cases the spasm persists continuously from the beginning to the termination of the disease; but, as a rule, the spasmodic rigidity of the muscles occurs in paroxysms with intervals of comparative, but never complete muscular relaxation. Each paroxysm lasts from a few seconds to several minutes, or with slight remissions for hours; while the duration of the free interval varies from ten minutes to hours, but at other times the spasms recur and remit with such frequency that they assume a more or less clonic character. As the disease progresses, the paroxysms of spasm recur with greater frequency, and muscular contraction is sometimes so violent that teeth are broken, long bones fractured and powerful muscles torn across. The paroxysms recur spontaneously, but they are induced by the most trivial external cause, such as a draught of air, a sudden noise, or an attempt to swallow or to administer an injection. Attempts at swallowing may, indeed, provoke an attack so readily that the disease may bear a certain resemblance to

hydrophobia. The spasm occasioned by the attempt may be in the pharynx, gullet, or in the cardiac end of the stomach, as the œsophagus passes through the diaphragm. But wherever it may be situated, all attempts at swallowing are rendered impossible, and fluids introduced are ejected through the nose and mouth, so that the patient can neither be fed in the usual way nor by the stomach pump. During the paroxysm the action of the inspiratory preponderates over that of the expiratory muscles, so that the act of coughing is rendered impossible, and mucus accumulates in the bronchi. In severe paroxysms the chest becomes fixed; the countenance is livid; the eyes are suffused; the patient foams

Fig. 93.



Taken from the original painting by Sir Charles Bell. (From Spence's "Surgery").

at the mouth; and he is tormented with a feeling of dread and suffocation. Arrest of respiration may sometimes be caused by spasm of the glottis; but, as a rule, it is the result of spasm of the thoracic muscles and diaphragm. In the intervals respiration is only slightly changed in frequency, but it is accompanied by a painful sensation of increased resistance, requiring effort. Motor paralysis is a rare symptom of tetanus. Rose observed paralysis of the muscles of one side of the face in two cases, and in one of them the tetanus was caused by a deep wound over the upper jaw, so that the lesion was situated in the area of distribution of the facial nerve. Similar cases have been recorded by others. General muscular weakness, and paralysis of certain groups of muscles are observed as terminal phenomena, and strabismus is, according to Wunderlich, a precursor of death.

The *sensory* disturbances in tetanus are such as are usually produced by intense muscular cramp in the muscles of the calf. Some observers

have noticed an increase of the sensibility to pain independently of the spasms, while at other times the acuteness of the senses of touch and temperature may be diminished. Paræsthesiæ, such as numbness, tickling, and tingling, have occasionally been observed. Pain at the epigastrium, piercing through to the back, is, according to some authors, a pathognomonic symptom of tetanus. It is present during both the tetanic paroxysms and the intervals, and depends most probably upon spasm of the diaphragm.

Psychical disturbance is generally absent in tetanus. The mind is almost always clear from the beginning to the end of the disease, although delirium or coma may supervene a short time before death, often due to the remedies used. Sleeplessness is one of the most troublesome symptoms of acute cases of tetanus, and even in subacute cases sleep is only obtained at broken intervals. The spasms cease during sleep and the narcosis of opium or chloroform. The skin is hot and bathed in perspiration, having a peculiar pungent smell, while the surface may be covered by sudamina as in other cases of profuse sweating. In the majority of cases the temperature ranges from 101° F. to 103° F., and may even rise suddenly to 105° F. in cases which recover, although it is not maintained long at this level except in fatal cases. In many cases there is hyperpyrexia immediately before death, the temperature rising to 108° F. or even 112° F., and it may continue to rise for some hours after death.

The pulse may remain normal during the first stage of tetanus, but there is a considerable increase in its frequency during the tetanic seizure, and in the last stage, especially when there is elevation of temperature, it may beat as often as 180 in a minute. Liston observed in a case of amputation during tetanus the vessels so contracted that not a drop of blood had escaped.

The daily quantity of urine passed in tetanus is usually below the average in health; the reaction is strongly acid; the specific gravity is high; and there is generally an abundant deposit of urates on cooling. Sugar in the urine in tetanus was first discovered by Demme, and its presence has since been detected by others. Senator found that the excretion of nitrogen was not increased in tetanus as compared with the amount excreted by a person fasting. He also states that the creatinine is not increased. There may be retention of urine, caused probably by spasm of the sphincter, while at other times dribbling may occur during the paroxysm. The bladder is, however, never affected to so great an extent as it is in acute spinal meningitis. Spasm of the sphincter ani is often present, as is proved by the difficulty of introducing an enema pipe.

The general health of the patient suffers greatly during the course of the disease. The distorted position of the body, the persistent sleeplessness, the difficulty of respiration, and the impossibility of swallowing combine to render the state of the patient extremely distressing. The bowels are constipated, the tongue is generally coated, a tenacious viscid saliva accumulates in the mouth, and the patient may be excessively hungry and tormented with thirst, yet can swallow neither food nor drink.

The intensity of the disease is variable. The symptoms in slight cases may consist only of trismus and some stiffness of the neck; in other cases they develop rapidly and prove fatal in a few days, or occasionally in a few hours from the commencement. Death may take place during a paroxysm from asphyxia caused by respiratory spasm; from sudden arrest of the heart's action; or from a gradual cessation of the heart's action during a paralytic stage.

In cases of recovery the convulsive attacks become lighter and less frequent, but a considerable degree of muscular rigidity persists long after the paroxysms have disappeared. Recovery takes place in from one to eight weeks, or even longer, and a certain degree of weakness and stiffness may remain in the muscles for a long time.

In the tetanus of strychnia the masticatory muscles are rarely attacked first, and may possibly escape altogether; the symptoms are well marked at the commencement, and reach their full development in a few minutes; opisthotonos is a very early symptom. There are usually intervals of complete intermission; and death occurs commonly in three hours, or else recovery is very rapid.

c. Tetany (Tetanilla).

Etiology.—Tetany occurs most frequently between the ages of seventeen and thirty years, but it is not unfrequent in children from the fourth to the sixth years of age. Amongst the predisposing causes of the affection may be mentioned disordered menstruation, the puerperal state, and pregnancy, and persons are particularly liable to be attacked at the age of puberty. Tetany has sometimes been observed as one of the sequelæ of typhoid and other fevers, and either obstinate constipation or exhausting diarrhœa appears to predispose to the disease, while it is probable that many cases are of rheumatic origin. Of the existing causes the most frequent are exposure to cold, fright or other emotional disturbance, while it may occasionally occur as an epidemic amongst girls at school.

Symptoms.—The affection generally begins by tingling and feelings of heat and cold in the hands, or by decided pains in the forearm and hand or in the calves of the legs. Soon afterwards the fingers feel stiff, and after a time this feeling increases to the decided spasm which constitutes the chief feature of the disease. The spasm occurs in paroxysms, each of which begins with rigidity of the hands and fingers; the thumb is strongly adducted; the fingers are approximated and flexed at the metacarpo-phalangeal and often extended at the phalangeal joints; and the palm is made hollow by the approximation of its outer and inner borders, so that the hand assumes, as Trousseau remarks, the peculiar conical form which the accoucheur gives to it when about to undertake the operation of turning. At other times the second, third, and fourth fingers are strongly flexed at the phalangeal as well as the metacarpo-phalangeal articulations, while the index-finger is only slightly flexed and all the fingers are firmly applied to the thumb, while the hand is usually strongly flexed at the wrist and directed to the ulnar border, although occasionally it is hyper-extended. The different positions assumed by the fingers and thumb may, indeed, be imitated by a powerful faradic excitation of the ulnar nerve, or by a simultaneous excitation of the ulnar and median nerves. When the spasm extends upwards the upper arms are strongly adducted, and the forearms are semi-flexed and are thus crossed upon the epigastrium. The spasms frequently extend to the lower extremities; the toes are then drawn towards one another and strongly flexed; the sole is hollowed out in the same manner as the hand; the dorsum of the foot is strongly arched; the heel is drawn strongly upwards by the contracted muscles of the calf; and the leg and thigh are in a state of rigid extension.

From the extremities the spasm extends to other portions of the body, but it is only very rarely that a large portion of the muscles of the body is simultaneously attacked, although in aggravated cases the spasm may invade the abdominal muscles and the bladder, as well as the muscles of the face, eyeballs, tongue, jaws, neck, and larynx. Extreme and dangerous symptoms may occasionally be caused by spasm of the muscles of respiration, including the diaphragm.

During the continuance of the spasm the affected muscles feel hard, tense, resisting to passive movements, and although the degree of contraction undergoes considerable variations during the attack, yet the muscles never become entirely relaxed. Each paroxysm may last for a few minutes or a quarter of an hour only, but in some cases it may continue for some hours; its violence then gradually abates, but a painful feeling of fatigue and a certain amount of rigidity of the muscles are generally felt for some time after the active spasm has ceased.

After a variable period of time the paroxysms recur, and although the interval between them may, in some cases, extend over some days or even weeks, yet it is, as a rule, only a few hours in duration, and the attack then recurs several times in the same day. In severe cases the attacks may follow one another so rapidly that the patient only remains free from spasm for a few minutes at a time. Erb found a great increase of the electrical excitability in the nerves of the extremities which supplied the affected muscles, while the galvanic irritability of the nerve was not only increased, but a tetanic contraction was also obtained by a feeble current during cathodal closure and even with anodal opening.

The attacks are preceded and accompanied by tingling, formication, numbness, and other abnormal feelings, while, in addition to the pain felt in the tense muscles, a tearing sensation is experienced in the course of some of the nerve trunks. The sense of touch is impaired, and the patient fails to appreciate the size and hardness of objects grasped, which feel as if they were wrapped in some thick material, and when the lower extremities are affected the subject feels as if he were walking on carpet. The paroxysms of tonic spasm are said by Trousseau to be preceded and accompanied by loss of muscular power; "movements of extension," he says, "are not the only ones abolished by the convulsive contraction of the muscles; those of flexion are equally so. The fingers, for instance, when half flexed, no longer obey the will, and the patient cannot close them further," and Buzzard has made a similar observation. The joints, especially the small joints of the hand, may be red, swollen, and painful, and during the acme of the attack the body may be bathed in perspiration, so that the patient may seem to be affected with acute rheumatism, and this appearance has, indeed, led many pathologists to believe that the disease is of rheumatic origin. Disorder of the general health, although frequently present as predisposing to tetany, does not appear to be an essential part of the disease; but in severe cases a certain degree of fever may be present, and the patient may complain of headache, giddiness, and humming noises in the ears. The mental faculties are always unimpaired, except in those severe and exceptional cases in which the respiratory troubles become so aggravated that the patient is threatened with asphyxia.

The symptoms of tetany generally appear suddenly on exposure to one of the exciting causes of the affection. Such paroxysm is generally of short duration, but the attack is liable to recur for a period of weeks or even months. The disease almost always terminates favorably, although a case is mentioned by Trousseau which terminated fatally from respiratory spasm.

3. Myopathic Spasms.

a. Local Myopathic Spasms.

A large number of spasms are caused by local disease of the affected muscles. The stiffness which accompanies muscular rheumatism, and the temporary spasm of the muscles of the neck which is caused by exposure to cold, are examples of a local affection of the muscles, although it is possible that even in these affections the intra-muscular nerve-endings may take a chief part in the production of the spasm.

b. General Myopathic Spasms (Thomsen's Disease).

Etiology.—This disease, which was first described by Thomsen, who is himself a sufferer, is often inherited, and may appear in several members of the same family, as well as in successive generations. In all cases in which a decided hereditary tendency to the disease was manifested the symptoms appeared in early infancy, but in other cases the characteristic spasm did not appear until from the fifth to the tenth year of age. The exciting causes are any slight injury, such as a fall from a carriage, which is accompanied by a severe fright.

Symptoms.—The most characteristic symptom of this affection is a peculiar stiffness and rigidity of the muscles on voluntary movement, which may increase to a persistent tonic spasm which prevents all intended actions. When the patient performs a voluntary movement the muscles contract slowly at first, but when the contractions are once begun they persist for a long time and only terminate very gradually, so that patients cannot readily let go articles they have once seized. The spasms are increased by cold, during the incubation stage of fevers, after severe and exhausting muscular exertion, and by emotional disturbances. The stiffness is also increased by rest, and the patient experiences great difficulty in beginning any voluntary action, but when, by a great effort, the intended movement is once begun, it is effected with increasing freedom, and when the body becomes warm by exercise the actions of the patient are scarcely to be distinguished from those of healthy persons. The muscles are well developed or even hypertrophied, the electrical reactions are normal or only slightly diminished, but a tetanic contraction is more readily obtained than in health. The muscles of the extremities are always affected with the spasm; in many cases the erector spinæ are implicated, so that the patient suffers from spasmodic lordosis; the movements of the tongue are often interfered with, giving rise to difficulties of articulation; the muscles of the neck

are sometimes also affected, so that on rotation of the head it is liable to be fixed for some time in a distorted position: and in some cases the patient experiences difficulty in opening his mouth owing to spasm of the masticatory and lower facial muscles, while in a case reported by Ballet and Marie the patient observed at one time of his life that when he looked upwards his eyes became fixed and he experienced considerable difficulty in bringing them to the horizontal position. In one reported case the phenomena of this disease coexisted with all the characteristic symptoms of pseudo-hypertrophic paralysis. There are no sensory disorders in this disease, but these patients are liable to suffer from attacks of giddiness. Passive movements of the limbs do not provoke muscular tension, and the tendon reactions are normal. The patients are irritable, suspicious, and reserved, while, as a rule, they are morbidly sensitive with regard to their condition.

CHAPTER IV.

SPASMODIC DISORDERS (*continued*).

II. CEREBRO-SPINAL SPASMS.

Inflammation in or near the pyramidal tracts in their course through either the brain or spinal cord gives rise to spasmodic jerkings of the limbs, but in such cases paralysis is soon established. The paralysis caused by disease of these tracts is attended by muscular tension and contractions, but these symptoms will be subsequently described under the name of the spasmodic paralyses. In post-hemiplegic chorea and athetosis, clonic spasm is present unaccompanied by much paralysis, but both these affections are preceded by an attack of hemiplegia, and they will be more conveniently described along with the spasmodic paralyses. There now remain for consideration the spasms caused by: 1, organic disease; and 2, by functional disease of the cortex of the brain.

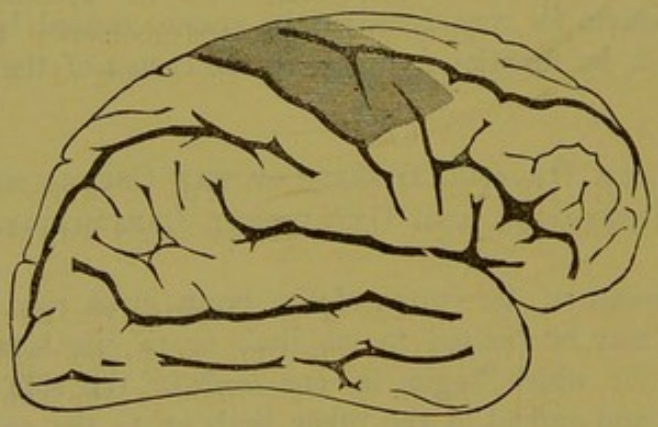
1. Spasms from Organic Disease of the Cortex of the Brain (Monospasms and Unilateral Convulsions).

Organic disease of the cortex of the brain gives rise to attacks of spasm which may be limited to one limb or to one side of the head (monospasm), or which begin in one limb or the side of the head (protospasm), and extend to the other limb or to the one side of the head, and to half the body or to both sides of the body. The characteristics of these spasms are that they are gradual in their appearance and deliberate in their progress, and that they are not attended by loss of consciousness, or the spasm begins before the patient becomes unconscious, so that he is afterwards able to describe a motor aura. Another characteristic of these convulsions is that an attack is followed by a transitory or permanent paralysis of the affected muscles. The clinical varieties of unilateral convulsions may be divided into—(1) crural, (2) brachial, (3) facial, (4) oculo-motor, and (5) masticatory monospasm or protospasm.

(1) *Crural Monospasm or Protospasm.*

In crural monospasm the spasm is limited to the leg, while in protospasm it begins in the leg, often in the toes, and ascending to the muscles of the trunk the arm is next attacked, and the face last. The spasm extends to a greater or less extent to both sides when it affects muscles which are bilaterally associated. In a case of crural protospasm which was recently under my observation the spasm, which began in the toes of the left foot, affected the abdominal and thoracic muscles on both sides, but was wholly limited to one side in the extremities, while the muscles of the face were not much affected. In the same case the spasm of the arm began in the muscles of the shoulder, and descended gradually to those of the hand. The tendon reactions were exaggerated on the side affected with spasm, and a certain degree of permanent hemiplegia existed, the paralysis being most marked in the left leg, which was the part in which the spasm always began. Several cases of crural monospasm and protospasm have been reported in which a post-mortem examination was obtained, and the lesion has always been found in or near the upper part of the ascending parietal convolution, the superior

FIG. 94.



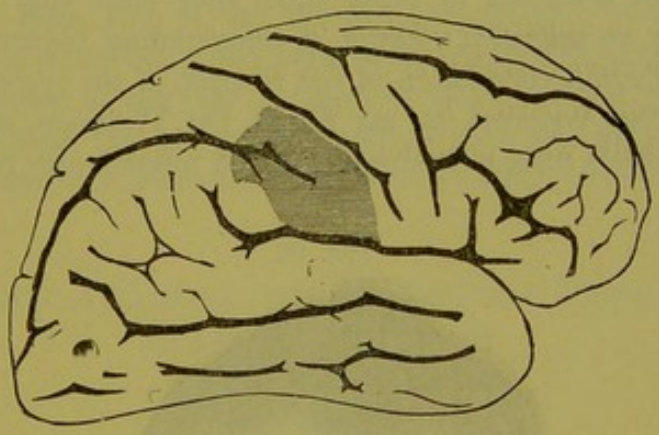
parietal lobule, and the paracentral lobule. Bourneville describes a case of the hemiplegia of infancy, in which the convulsions began by tremors or twitching in the left or paralyzed leg. The cortex of the right hemisphere was found atrophied in front of the fissure of Rolando, the superior half of the ascending frontal, the posterior extremities of the first and second frontal (Fig. 94), and the whole extent of the paracentral lobule.

(2) *Brachial Monospasm or Protospasm.*

In brachial monospasm the convulsions are limited to the arm, while in protospasm they begin in the arm, the face is next attacked, and the

leg last. The spasms often begin in the small muscles of the hand and gradually ascend to those of the shoulder, this being the opposite order to that followed by the spasms when the convulsions begin first in the lower extremity. A case of brachial protospasm has been recorded by Dr. Dreschfeld in which the attack began "by sudden clenching of the fist, flexing of the wrist, and pronation of the forearm on the left side, the corresponding angle of the mouth being at the same time drawn downwards." At the autopsy the dura mater was found adherent to the brain on the right side over the greater part of the ascending parietal convolution and the supra-marginal lobule (Fig. 95). Several

FIG. 95.



other cases of brachial protospasm with post-mortem examinations are on record, and the lesion has always been found in or near the ascending parietal and frontal convolutions. In a case recorded by Dr. Hughlings-Jackson the spasm began in the shoulder and went down the arm, and in this case a tumor was found in the posterior extremity of the first frontal where it joins the ascending frontal convolution. When, however, the spasm begins in the hand the lesion is situated near the lower end of the fissure of Rolando.

(3) *Facial Monospasm or Protospasm.*

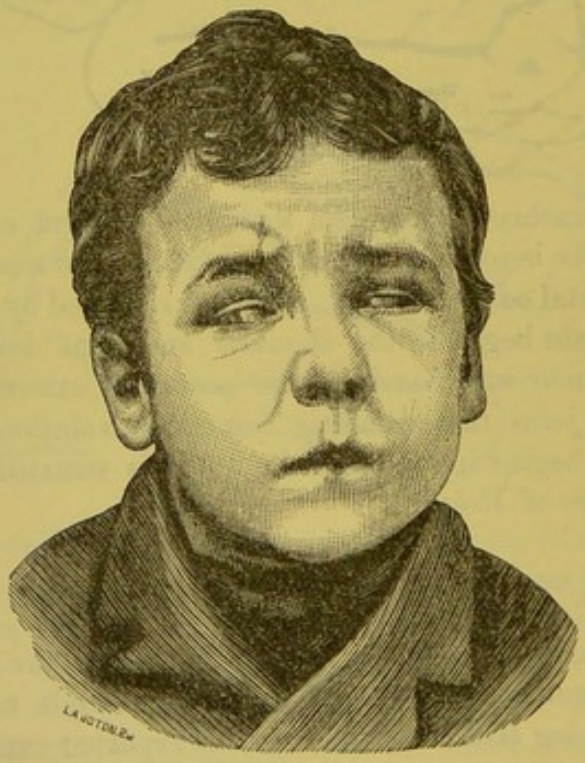
In facial monospasm the convulsion is limited to the muscles of the face, while in protospasm it begins in them, extends next to the arm, and attacks the leg last, if at all. In the reported cases of facial protospasm, with post-mortem examination, the lesion has always been found in the cortex in the inferior part of the ascending frontal and parietal convolutions upon a level with the posterior part of the third frontal convolution. The case of a French soldier, for example, is reported by Hitzig, who, two months after a bullet wound on the right

side of the head, suffered from clonic spasms followed by paralysis of the left side of the face and tongue. An abscess was found in the cortex of the right hemisphere, situated in the inferior part of the ascending frontal on a level with the third frontal convolution.

(4) *Oculo-motor Monospasm or Protospasm.*

Many cases of unilateral epilepsy begin by a conjugate deviation of the eyes and rotation of the head and neck, the eyes and head being directed towards the side affected by spasm. In some cases the patients are able to give an accurate description of the head being forcibly twisted to one side, but in other cases they state that the attack is ushered in by an apparent rotation of surrounding objects, this feeling being the subjective correlation of the rotation of the eyeballs and head. A case has been reported by my friend Dr. Thomson, of Oldham, in which the eyeballs were persistently rotated towards the right (Fig. 96)

FIG. 96.



by a spiculum of bone which projected towards the cortex of the brain near the angular gyrus of the left side from a depressed portion of the skull over the infero-postero-parietal area. On removal of this spiculum

of bone by means of an operation the rotation of the eyeballs almost immediately ceased.

(5) *Masticatory Monospasm or Protospasm.*

Masticatory spasm from cerebral disease is not very common, but several cases are now recorded. Lépine has reported a case of apoplexy in which the masticatory muscles were in a state of persistent spasm up to the time of death. At the autopsy the chief lesion was found to consist of a hemorrhagic focus which occupied the claustrum, external capsule, and lenticular muscles of the right hemisphere, and had separated from the white substance a portion of the gray matter of the Island of Reil, and of the inferior part of the ascending frontal convolution the latter being the part in which Ferrier places the centre for the movements of the jaws. Cases in which trismus was caused by disease in or near the cortex of the brain have also been recorded by Petrina, Seeligmüller, Senator, and Oulmont and Charcot.

2. Spasmodic Affections from Functional Disease of the Cortex of the Brain.

a. Epilepsy.

Etiology.—A hereditary taint may be distinctly traced in rather more than one-third of all cases of epilepsy, but a neurotic constitution is met with in a much larger number of cases. The disease appears to be transmitted rather more frequently through the mother than the father, and the female sex is rather more frequently attacked than the male sex. With regard to age Dr. Gowers found that out of 1450 cases analyzed by him the disease began in 29 per cent. under ten years of age; in 46 per cent. between ten and twenty years; in 15.7 per cent. between twenty and thirty years; in 6 per cent. between thirty and forty years; in 2 per cent. between forty and fifty years; and in 1⅓ per cent. above fifty years. Anæmia, chlorosis, scrofula, rickets, chronic alcoholism, and chronic lead-poisoning, appear to beget a certain instability of the nervous system which predisposes to the production of epilepsy.

The *exciting* causes of epilepsy are profound emotional disturbance, intestinal irritation from the presence of worms, overloading of the stomach, the passage of gall-stones, over-exertion, sexual excess, exposure to cold, acute febrile diseases, sunstroke, injuries to the head, and injuries to the principal nerve trunks.

Symptoms.—The *premonitory* symptoms of an epileptic attack may be divided into remote and immediate warnings, the latter forming the *auræ epilepticæ*. The remote warnings may extend over hours or days before the attack; they usually consist of such symptoms as headache, dizziness, confusion of thought, or some mental change, the patient becoming depressed or morose, or excited, lively, and irritable.

Epileptic auræ, or the immediate warning of the paroxysms, consist of the subsequent account which the patient is able to give of the feelings he experienced immediately before the attack. The aura may consist of (*a*) motor, (*b*) sensory, (*c*) vaso-motor and secretory, or (*d*) psychical phenomena.

(*a*) *Motor Auræ.*—It is not always easy to distinguish between a motor and a sensory aura, because the patient, instead of describing a spasm in objective language, often describes the sensations which accompany the spasm in subjective language. When, for instance, the convulsion begins in the hand, the patient, instead of describing the thumb as being drawn into the palm, says that he felt a dragging sensation in the thumb, or a feeling of creeping or numbness in the hand which gradually passed up the arm. And when the convulsion begins in the lower extremity the aura usually begins as a creeping sensation in the big toe which passes up the leg and may extend to the arm before consciousness is lost. Conjugate deviation of the eyes and rotation of the head and neck, which frequently usher in an epileptic attack, are often regarded by the patient as an apparent rotation of surrounding objects. At times patients see objects recede from them and appear smaller, or approach them and become larger, and these sensations are probably caused by variations in the tension of the muscles of accommodation. The motor aura often begins in the side of the face, and it is then described as a feeling of "the face being drawn," or it may begin in the side of the tongue, and be described as a feeling of something crawling over it. In many cases the aura consists of a sudden inability to speak, or a transient aphasia. At other times it consists of a sensation of shivering or of trembling in the muscles of the back, while at other times there may be a general tremor or a jerking of particular limbs, and Romberg mentions "a sudden relaxation and loss of mobility" as being sometimes the forerunner of an attack. In some cases the aura consists of complicated coördinated movements, such as running forwards or backwards, or turning round.

(*b*) *Sensory Auræ.*—The sensory aura sometimes consists of a feeling of general heat or cold, while at other times it is distinctly localized. Besides various sensations such as tingling, numbness, or pain in the extremities, it may consist of a localized pain in any part of the head, or of a general feeling of pressure in the head. Patients complain of

dizziness and vertigo before an attack, more frequently perhaps than any other form of aura, but these symptoms are most probably but subjective accompaniments of the derangement of the adjustments of the body to surrounding objects in space, which is caused by commencing motor discharges.

The *auræ of the special sensations* may consist of undeveloped or *crude* sensations, or of more elaborate sensory representations. The crude sensations consist of sparks, flashes of light, and various colors; hissing, ringing, or explosive noises; metallic or other abnormal sensations of taste, or unpleasant smells. In some cases the attack is ushered in by sudden blindness of one or both eyes, or by a sensation of unusual stillness. The elaborate sensory representations consist of spectral faces, visions of beautiful places or more commonly of disagreeable objects and scenes, vocal utterances or strains of music, and other complicated perceptions.

Visceral sensations sometimes constitute epileptic auræ, and probably one of the most common of all sensory warnings are sensations which are referred to the pit of the stomach. The epigastric aura sometimes consists of pain, coldness, or burning at the pit of the stomach and sometimes it is spoken of as a vague and indescribable sensation, which ascends to the throat and causes a feeling of choking, or rises to the head, and then immediate loss of consciousness ensues. In some cases a feeling of suffocation constitutes the warning, and in others there may be nausea with rumbling in the bowels, retching, a feeling of intense hunger, bronchial asthma, or palpitation with or without angina.

(*c*) *Vaso-motor auræ* are often described as a sensation of coldness or numbness in the fingers and toes, and the affected part becomes pale and cold to the touch. At other times spots in various parts of the body assume a red color, and become the seat of feelings of transient heat. Of the *secretory auræ*, an excessive flow of saliva is probably the most commonly observed.

(*d*) *Psychical auræ* are frequently experienced in the form of a feeling of intense horror or alarm, and the patient may look startled, guilty, or frightened. In other cases the patient feels as if he were suddenly in a strange country, and in many cases the aura is what Dr. Hughlings-Jackson has named "a dreamy" or "voluminous" mental state similar to that experienced by persons in the act of drowning, during which all the past events of life are said to crowd upon the memory.

It is very important to study the order in which the phenomena of the aura succeed one another. The motor aura generally begins in small muscles, which are organized for special actions, and other muscles become involved in the spasm in a determinate manner, but the succes-

sions of these motor phenomena will be best studied in cases of unilateral epilepsy. A crude sensation of sight, such as a red light, is often followed by a developed perception, such as the image of a man; a confused noise may be succeeded by strains of music; and a subjective sensation of smell may be followed by efforts to prevent the effluvia from gaining admission to the olfactory chambers, showing that the sensation had become objective. A visual is not unfrequently associated with an aural sensation, sometimes the one and sometimes the other taking precedence. In some cases the epigastric aura is followed by an emotion of fear or of anguish, and the patient may have a facial expression corresponding to the emotion.

The *epileptic paroxysm* may be divided for the purposes of description into (*a*) *epilepsia mitior* or *le petit mal*, (*b*) *epilepsia gravior* or *le haut mal*, and (*c*) *epileptiform seizures*.

(*a*) *Epilepsia mitior* or *le petit mal* consists of paroxysms of momentary confusion of thought, or transitory unconsciousness. The patient, for instance, may be attacked while speaking; he becomes suddenly unconscious, there is a pause, probably in the middle of a sentence, but in a few seconds speech is resumed at the point where it was interrupted and the sentence is finished. At times the attack may consist of a feeling of fainting with slight confusion of mind, and, indeed, any of the numerous auræ just described may, along with slight confusion of mind, constitute a minor attack of epilepsy. Most of these attacks are accompanied by minor degrees of muscular spasm. At the outset of the seizure the countenance becomes ghastly pale; the pupils contract; the eyes are fixed and staring or squint; and the mouth is slightly drawn; or there may be partial rotation of the head and eyes, chewing movements, or rolling about of the tongue. An involuntary discharge of urine or feces during the attack frequently occurs. Sometimes the patient utters a shriek and reels, or walks hurriedly round the room and then recovers. At other times the patient is unconscious for a considerable time, but goes on with the work in which he was engaged as if he were conscious. Minor attacks are very liable to be followed by "epileptic mania."

(*b*) *Epilepsia gravior* or *le haut mal* may for the purposes of description be divided into three stages.

(j) The *first stage* is ushered in by three prominent symptoms which occur simultaneously; namely, loss of consciousness, sudden falling, and great pallor of the face, while a fourth symptom is often present in the form of a loud and piercing cry. The loss of consciousness is sudden and complete, and every form of sensibility and mental operation is completely abolished. The patient often falls, as if struck by lightning,

either forwards on his face, backwards on his occiput, or laterally, and so instantaneously that he has no time to select a place or attitude, and may consequently fall into fire, water, or from a height. At other times the patient has sufficient warning of the impending attack to enable him to sit or lie down. Pallor of the face is probably always present at the beginning of the attack, although the symptom is sometimes so transient that it may pass unobserved. The epileptic cry which the patient often utters immediately before or during the fall is loud and piercing and alarms, according to Romberg, both man and animals. It is probably produced by spasm of the expiratory muscles with closed glottis. When the patient falls to the ground he remains for a period of from two to forty seconds in a rigid condition caused by a tonic, although unequal, contraction, of all the muscles of the body. Various distortions are thus produced; there is conjugate deviation of the eyes with rotation of the head and neck; the pupils are dilated and insensible to light; the countenance is variously altered; the jaws are firmly closed and the tongue may be severely bitten; there is opisthotonos, and the different segments of the lower extremities are extended upon one another and upon the trunk, the foot being rotated inwards and the toes widely separated; while the segments of the upper extremities are usually flexed upon one another, the thumb being bent upon the palm, the fingers closed, and the hand pronated. The respiratory muscles are in a state of tonic spasm and the breathing is arrested. The pallor of the face is soon replaced by a dull red or dusky hue; the veins of the head and neck become greatly distended; the carotids throb violently, and the action of the heart is forcible, but the pulse at the wrist is small or imperceptible.

(jj) The *second stage* begins after a variable period of from two to forty seconds, and then the tonic gives place to clonic spasms which are usually more pronounced on one side of the body. The muscles of the face, tongue, pharynx, and larynx are usually first affected by clonic spasm, and those of the extremities and trunk are afterwards invaded. The patient now presents a hideous appearance; the head is alternately drawn laterally, or forward and backward; the eyes are convulsively rotated in various directions, but upward and outward rotation predominates, so that the pupils are hidden and only the whites of the eyes are visible under the blinking, half-closed lids; the face is variously distorted; the jaws are violently convulsed and the tongue being severely bitten, the blood oozes through the clenched teeth as a sanguineous froth; the trunk and limbs are variously thrown about; and the contents of the bladder, rectum, or vesiculæ seminales may be evacuated.

The venous hyperæmia reaches its maximum just as the spasms are

beginning to abate in severity, and the skin is bathed in sweat which sometimes has a fetid odor. The heart beats tumultuously, the carotids throb; and the pulse, if it can be felt, is fuller and more labored than during the period of tetanic contractions. The pupils are alternately contracted and dilated and are said to be slightly sensitive to light. This stage may last from a few seconds to five or ten minutes, the average duration being from two to three minutes.

(jjj) The *third stage* is characterized by a gradual return to consciousness and voluntary power. The convulsions either cease suddenly or wear off gradually, the period of transition being marked by partial jerkings of some muscular groups, or by a diffused tremor of the body. The patient soon attempts to change his position; he opens his eyes and looks around him with a bewildered expression, and may even attempt to speak. The respiratory movements are now more natural in rhythm, although still somewhat irregular; the pupils are contracted; the pulse is quieter and fuller than in the previous stage; the conjunctivæ are injected and petechiæ are often observed on the eyelids, forehead, and temples; the muscles are relaxed and powerless, and the patient is exhausted and disposed to sleep. The attack is often followed by vomiting, and a large quantity of pale urine is often passed.

(iv) The *fourth or after stage* is very variable in the duration, severity, and nature of its symptoms in different cases. In some cases the patient recovers in half an hour and resumes his usual occupation, but, as a rule, recovery is delayed for a much longer period. In many cases the general muscular relaxation is interrupted by clonic spasms, or fibrillary contractions, and the patient suffers from lassitude and stupor, from which he is aroused with difficulty, and, if awakened, he is peevish and irritable. The average duration of the stupor is about an hour when the attack occurs during the day, but when it occurs in the evening or during the night it passes insensibly into the ordinary nocturnal sleep.

Complications.—Various mental disturbances are by far the most important of the complications of epilepsy. The patient sometimes exhibits marked mental derangement immediately before as well as after the paroxysm, and a maniacal condition sometimes constitutes the principal feature of the attack. After an attack of epileptic vertigo a person may continue for some seconds, minutes, or even hours in a dull, half stupid condition. He may mutter a few incoherent words or some lewd expressions, no matter how foreign to his habits; he may unbutton his clothes and expose his person, urinate in a public assembly, exhibit himself naked to his domestics, or even walk in public naked unless prevented, and on recovery he has only the vaguest recollection of what has occurred. These, however, are a few of the minor actions

sions is shown by the fact that successive infants of one family are liable to be attacked with convulsions in the absence of any definite cause. The children of parents with neuropathic constitutions are more liable to be attacked than the children of healthy parents. All debilitating causes increase the tendency to convulsions, and children suffering from rickets are specially liable to be attacked: of sixty-five infants attacked with convulsions, Dr. Gee found that no less than fifty-six of the number were suffering from rickets.

Eclampsia has been divided into several varieties according to the exciting cause of the convulsions. These are (*a*) idiopathic, (*b*) reflex, (*c*) febrile, (*d*) asphyxia, (*e*) uræmic, (*f*) puerperal, and (*g*) toxic convulsions.

(*a*) In *idiopathic* convulsions children are so predisposed to these seizures that an attack supervenes from the slightest exciting cause or even in the absence of any such cause.

(*b*) *Reflex* convulsions are caused by irritation of the extremities of peripheral nerves. Amongst the more usual causes of irritation are pricking by pins, wounds and burns on the surface of the body, retention of urine, the presence of a calculus in the kidney, foreign bodies in the external auditory meatus, and irritation of the digestive canal from the presence of worms or undigested food, and of the gums during painful dentition.

(*c*) *Febrile* convulsions often usher in severe acute diseases such as pneumonia and the eruptive fevers. They appear to correspond to rigors in the adult.

(*d*) Convulsions due to *asphyxia* occur in the course of diseases of the respiratory organs.

(*e*) *Uræmic* convulsions in children generally result from scarlatinal nephritis, and they may arise from most forms of Bright's disease in the adult.

(*f*) *Puerperal* eclampsia is, as a rule, merely a form of uræmic convulsion.

(*g*) *Toxic* convulsions are caused by such agents as prussic acid, nicotine, picrotoxine, œnanthe, crocata, carbonic oxide, and carburetted hydrogen.

Symptoms.—An attack of eclampsia cannot be distinguished from a true epileptic seizure, and it is therefore unnecessary to give a minute description of it. Infantile convulsions have been divided into *internal* and *external*, the muscles of the glottis and the respiratory muscles being chiefly affected by spasm in the former and the muscles of external relation in the latter.

The symptoms caused by spasm of the glottis have already been

described, and we shall consequently limit our further remarks to the external convulsions of children. An attack of eclampsia may occur either with or without premonitory symptoms; but when present, these usually consist of sleeplessness and restlessness, or drowsiness for a day or two before the attack; while immediately before it the pulse is often hard and wiry, the countenance assumes a frightened expression, or the child starts up frightened from a fitful and uneasy sleep. The convulsion usually begins by conjugate deviation of the eyes, and slight jerking contractions of the muscles of the angles of the mouth. The natural look of the infant is now exchanged for a fixed stare, followed soon afterwards by an upward rotation of the eyeballs, the latter being in its turn followed by a fixed stare, and that again by an upward rotation of the globes. The eyeballs are often rotated to the right or left as well as upwards, and the two are generally moved unequally, so that a considerable degree of strabismus may occur. The pupils are sometimes dilated, sometimes contracted, and when they are completely concealed by the superior lids, the whites of the eyes being alone visible, the countenance assumes a frightful and characteristic aspect.

Clonic spasms of the facial muscles produce a series of grimaces and contortions, in which the labial commissures are drawn outwards, and at each successive jerk a peculiar sucking noise is made by the passage of air through the mouth, the lips being covered by a frothy, and often slightly sanguinolent mucus. The superior lip is sometimes drawn upwards, so as to expose the teeth, and the countenance then assumes an almost savage expression. The inferior jaw is sometimes agitated by clonic spasms, while at other times there is trismus, interrupted from time to time by grinding of the teeth. The head is usually strongly retracted, and sometimes rotated to one side. The thumb is flexed into the palm, and the fingers are flexed over the thumb; the forearm is bent upon the arm and is constantly agitated by slight movements of semiflexion and semiextension; the hand is alternately pronated and supinated; and the segments of the superior extremities are contorted into every imaginable shape. The inferior extremities are affected in a similar manner, although to a less degree than the superior. The muscles of the trunk occasionally participate in the clonic convulsions, but as a rule the trunk is maintained rigid by tonic contraction of its muscles. The contractions of the muscles of one-half the body may predominate over those of the opposite side, and then the child is arched laterally in such a way that he may be projected out of bed by the convulsion. The spasmodic contractions of the diaphragm and of the muscles of the larynx produce a peculiar and characteristic noise when air is drawn into the chest during inspiration. Involuntary evacuations

occasionally take place during convulsions. Deglutition is rarely impossible, although attempts to get the infant to swallow during the convulsion are attended with danger.

There is complete loss of consciousness during the attack, but reflex excitability is partially retained. When the convulsion is prolonged the face becomes of a violet color and bathed in perspiration; the head is hot and the extremities cold; the skin is moist; the pulse is frequent and difficult to count, owing to jerking of the tendons; and the respirations are accelerated, but stertorous only in aggravated cases.

The ocular muscles and those of facial expression are usually the first to be affected with clonic spasm, and then the muscles of the fingers and forearm. In the more severe convulsions the muscles of the shoulders are affected, but those of the back and lower extremities are only implicated in aggravated cases. The great tonic contractions which form the first stage of the epileptic attack fail altogether in eclampsia. The duration of an attack of eclampsia is very variable. The convulsions cease in some cases in a few minutes, while in others they recur for hours or days, with only short intervals of calm. The *terminal convulsions* of asphyxia are generally partial, incomplete, and alternate with coma. The *initial* convulsions of fever are intense and generalized, but are usually limited to a single attack. *Uræmic* convulsions are characterized by their violence, the frequent repetition of the paroxysm, and the profound coma which alternates with or succeeds the spasm. Convulsions caused by meningeal hemorrhage or other organic lesion generally assume a unilateral character and are followed by paralysis with contractures, choreiform movements, aphasia, or idiocy. Essential convulsions sometimes terminate fatally, either after a single violent seizure, or after a series of seizures which occur in rapid succession, death being caused by spasm of the glottis or by coma.

c. Hysterical Spasmodic Affections.

Etiology.—Hereditary predisposition exerts a powerful influence in the production of hysteria, the transmission being sometimes direct from the mother to the daughter and at other times indirect through inheritance of a general neuropathic constitution. Hysteria occurs with preponderating frequency in the female sex, and the first symptoms usually make their appearance about puberty, but it also occurs in the male sex and is by no means unfrequently met with in children of both sexes. All causes which lower the nutrition of the nervous system, such as hemorrhages, insufficient nourishment, impaired digestion, and anæmia, predispose to hysteria. It is also frequently induced by the

depressing passions, such as fear, jealousy, and remorse, as well as by anxiety, especially when combined with overwork. Hysteria may be acquired, by those who are predisposed to it, by imitation, and young susceptible girls are not unfrequently seized after being witnesses of an attack in another. An actual epidemic of hysteria may occur in public institutions, and the convulsive diseases of the middle ages appear to have spread in this manner. All uterine derangements and ovarian diseases are apt to be attended by hysteria, and it may also be caused by local irritation of the other viscera, such as intestinal irritation from the presence of worms. A slight contusion or other insignificant injury may cause hysteria in those who inherit a strong predisposition to the disease, but a severe injury, such as a gunshot wound of a large nerve trunk, may induce in the strongest men an emotional condition very similar to hysteria in the female.

Symptoms.—The spasmodic affections of hysteria may be divided into (1) general hysterical attacks, and (2) local spasms.

(1) Hysterical Attacks.

(*a*) *Simple Hysterical Attack.*—The attack is preceded by the sensation of globus along with a feeling of suffocation, a painful dragging in the extremities, pain and giddiness in the head, singing in the ears, or darkening of the field of vision. It is often preceded by a fit of crying or laughing, or a combination of both; the patient suddenly screams or makes a spluttering noise, and falls down in a state of apparent unconsciousness. The head and extremities become affected with general rhythmical clonic convulsions, the breathing is accelerated and exaggerated, irregular, or temporarily arrested. The loss of consciousness is more apparent than real. The hysterical patient generally hears what is said by those around her, and she has almost always time to find a suitable place upon which to fall; she often throws herself on a couch or reclines on a sofa, and not unfrequently appears to bestow some degree of attention upon the propriety and gracefulness of her attitude.

Another peculiarity of the hysterical attack is that the facial expressions and attitudes assumed are not devoid of meaning, but are repetitions of those occurring in health under varying emotions. Sometimes the expression is that of great terror, at other times there is a frown as if of anger, and at still other times it becomes imploring or beseeching.

Hysterical attacks rarely last more than a few minutes, but they may recur in quick succession, so that they seem to form an almost continuous paroxysm, extending over a considerable period. The hysterical seizure frequently ends in a fit of crying and sobbing, there is no sub-

sequent coma, and on recovery the patient generally passes a large quantity of clear and limpid urine of low specific gravity. Hysterical attacks always occur when some one is present to witness them, and never during sleep or when the patient is alone.

(*b*) *Cataleptic attacks* are liable to occur in hysterical patients; they are of variable duration, disappearing sometimes in the course of a few hours, and being prolonged at other times, with slight intermissions, for a period of months. Cataleptic rigidity is sometimes limited to particular limbs; but, as a rule, the whole body is implicated, and then all voluntary movements are suspended and reflex action is diminished. All the forms of general sensibility are usually lost, but one or more of the special senses, especially hearing, may be retained.

(*c*) *Hysteria in Boys.*—Boys, at the approach of puberty, not unfrequently suffer from hysterical symptoms resembling those observed in the female sex. Sometimes the symptoms may assume the form of globus along with attacks of causeless weeping and sobbing; at other times there may be partial spasm of the glottis, a barking cough, attacks of dyspnœa or some local sensory or motor disturbance. Psychical phenomena often predominate. In a case which came under my observation, the boy was sometimes found creeping on his hands and knees and barking like a dog; another time he jumped like a frog from the floor on to the table. The depraved form of hysteria named *chorea major* is often met with in boys. In this variety of the disease the patients run, dance, jump, or climb with much greater readiness and dexterity than similar actions could be performed in health, or they may sing or recite poetry, even in a foreign language.

(2) Local Spasms.

In hysteria both tonic and clonic spasms may occur in every muscle and group of muscles of the head, trunk, and extremities. Every one of the spasms already described as occurring in the area of distribution of the peripheral motor nerves may appear in hysteria, but it is unnecessary to describe these in detail. The facial muscles are incessantly active in many hysterical patients, so that the countenance has a restless and unsettled expression, and, indeed, the "facies hysterica" constitutes one of the main characteristics by means of which the practised physician is enabled to diagnosticate the disease. It is "characterized," according to Dr. Todd, "by a remarkable depth and prominent fulness, with more or less thickness of the upper lip. There is also a fulness and obviously drooping condition of the upper eyelids. This drooping conformation of eyelids is at once a mark of beauty, and of that from

which many beautiful women suffer very much, namely, the hysterical state of constitution."

Spasmodic closure of the glottis may produce alarming dyspnœa, and the patients are liable to attacks of convulsive laughter and weeping, which often arise apparently in the absence of any emotional disturbance. During hysterical attacks loud screams are commonly emitted, and in that form of hysteria named chorea major the patients often imitate the cries of animals by mewing, barking, or howling. Hysterical patients often suffer from a temporary acceleration and exaggeration of breathing without there being any feeling of embarrassed respiration, and at other times they suffer from temporary spasmodic pauses in the respiratory rhythm. Hiccough and yawning are frequent and sometimes very distressing symptoms.

The pharyngeal muscles are sometimes spasmodically contracted, so that swallowing becomes difficult or impossible. Spasm of the tongue is not unfrequently associated with that of the pharyngeal muscles, and at every attempt to move the organ it becomes distorted in various directions, so that articulation and swallowing become greatly impeded. The sensation of choking in the throat, named *globus hystericus*, is supposed by some to be caused by a spasm of the œsophagus, while at other times the spasm may be so persistent as to resemble organic stricture.

The stomach is liable to undergo spasmodic contractions, which give rise to persistent and distressing vomiting. The patient vomits almost immediately after food is taken, and the latter is usually consequently ejected in an undigested condition. Some of the food is, however, probably retained, as the nutrition of the patient rarely suffers in proportion to the apparent violence and persistency of the vomiting.

Irregular peristaltic movements occur in various parts of the intestines, and these may be so energetic that they can be felt through the abdominal wall, and the patient may become convinced that a movable body is present in the abdomen. Spasm of certain portions of the intestines may be so persistent as to cause temporary stricture, and the bowels above the constricted portion become so distended with gas, as to give rise to what has been called a "phantom tumor;" or a real obstruction of the bowels may sometimes be caused by accumulation of feces behind the constricted portion. Eructations, borborygmi, and griping pains may also be caused by irregular peristaltic movements of various portions of the digestive canal.

Spasmodic retention of urine, generally combined with increased inclination to micturate, occurs in many hysterical patients, and this condition is sometimes, but not always, associated with a painful condition of the genitals.

Vaginismus, caused by spasm of the constrictor vaginæ, sometimes renders coitus difficult or impossible; it is generally associated with hyperæsthesia of the vaginal orifice, and the spasm is induced by reflex action.

Hysterical patients are also liable to suffer from attacks of palpitation, and during these the pulse is small and hard and the skin pale and cold, owing to spasm of the small arteries of the body. Patients suffering from hysteria are also subject to fainting fits, and these are most probably caused by sudden anæmia of the brain from spasm of its vessels.

d. Hystero-epilepsy.

In hystero-epilepsy the patient suffers in the intervals between the attacks from various hysterical symptoms, the most usual of which are complete or incomplete hysterical hemianæsthesia associated with ovarian hyperæsthesia. The paroxysm is always preceded by an aura, consisting of a sensation which proceeds from the region of the hyperæsthetic ovary and ascends successively to the epigastrium, throat, and head, and on reaching the last the patient utters a loud shriek and falls to the ground. All the muscles of the body now become the subjects of tonic spasm; the head is retracted, and the body and limbs are arched backwards and rigid; the respirations are stertorous and infrequent; and foam, sometimes bloody from the tongue being bitten, generally issues from the mouth. The tonic stage is followed by a few clonic convulsions, but these soon cease, and a state characterized by general muscular relaxation, stertorous breathing, and coma, terminates that portion of the attack which resembles the epileptic paroxysm.

The *second* stage, or what the French call the "*phase des grand mouvements*," now makes its appearance. The body is arched backwards in a state of opisthotonos, or bent forwards or laterally; while at other times it is maintained in a rigid position, with the lower extremities extended and the upper stretched out. This stage, according to Richer, "consists of movements which may be executed with a certain rhythm, or in the most complete disorder, and often with excessive violence. In addition to screaming, there is a disposition to bite and to tear, and sometimes the patient becomes furious. At other times the attack of the grand movements is in no way frightful and consists almost exclusively of salutations, jumping, and somersaults, which recall to mind the performance of acrobats, and justify the name of "attaques de clownisme" that has been given to it.

The *third* stage, or stage of emotional attitudes (*phase des attitudes passionelles*), now appears, and during its continuance the patient

assumes in rapid succession attitudes and gestures expressive of various emotions. The first attitude assumed by the patient is usually a threatening one; she raises herself in a half-sitting posture; the brows frown; the fists are clenched; and the face presents an angry and menacing expression. This attitude, however, soon gives place to an expression of abject fear, which in its turn makes room for a look of intense happiness and beatitude. But the expression of beatitude is also fleeting in character, and is succeeded by one of intense voluptuousness, followed by gestures which lead to this stage being called the *phase of lubricity*. Terror now seizes the patient; she sees rats and other odious animals that evoke from her passionate exclamations of dread and disgust, and this is followed by a stage in which she appears to be laboring under the idea that she committed a great offence, and sues for mercy. The patient now hears strains of music, she looks pleased and may join in humming the tune, but her singing is soon followed by weeping, broken by reproaches addressed to her parents as the cause of her misery. This last phase constitutes the phase of recovery, but hallucinations may persist for some time. The patient can always describe afterwards the hallucinations to which she was subject, and each of the attitudes is found to have corresponded to an hallucination.

An attack of hystero-epilepsy may be provoked at any time by various manipulations, such as suddenly gripping the skin of the breast on both sides, on a level with the fifth rib, and midway between the anterior and posterior boundaries of the axillæ. An attack is also readily induced by pinching a fold of the skin of the sub-inguinal region, or by slight pressure on the region of the hyperæsthetic ovary. Sudden and firm pressure exerted on the affected ovary, however, instantly arrests the paroxysm at any stage. When such compression of the ovary is made, the patient's mouth opens widely, the tongue is spasmodically protruded, and the convulsions cease. Attacks of hystero-epilepsy are not often seen in this country in the typical form described by French authors.

Hystero-epileptic attacks may occur in *series* like epileptic attacks, and each group of attacks may consist of from several hundreds to over a thousand separate seizures, while group after group of these attacks may follow each other day by day for weeks. This *status hystericus* is, however, unlike the *status epilepticus* in the facts that the tongue is not bitten, that there is no elevation of temperature, and that it is never fatal.

e. Catalepsy.

Etiology.—Catalepsy often occurs as one of the many manifestations of hysteria, while at other times it is caused by chronic cerebral disease, such as softening, tubercular meningitis, and tumors. In some cases the cataleptic condition appears to be premonitory of epilepsy, while in other cases attacks of catalepsy recur at certain intervals, and thus appear to take the place of epileptic seizures. Some cases, however, cannot be traced to any external cause, and then catalepsy may be called *idiopathic* or *essential*, cases of this kind being observed in families who inherit a decided neurotic disposition. The disease is most frequently observed about the age of puberty, but it has been met with as early as eight years of age, or even earlier, and occasionally in advanced life. The two sexes appear to be equally liable to the idiopathic variety, but the hysterical is almost exclusively observed in the female. The exciting causes of idiopathic catalepsy are gastric and intestinal irritation, and great emotional disturbance. Malarial infection is said to have caused typical attacks of catalepsy.

Symptoms.—The cataleptic attack is sometimes preceded by premonitory symptoms, such as headache, vertigo, trembling of individual muscles, and an undefined sense of discomfort. As a rule, the attack begins abruptly; the movements of the patient are suddenly arrested, it may be while he is speaking or performing some action; the face becomes deadly pale; the respirations are slow and tranquil; the pulse is soft; and, although consciousness is lost, the attitude of the patient at the time of the seizure is retained. The muscles in action at the beginning of the attack appear to be the first to become rigid, but in most cases the spasm rapidly extends to all the voluntary muscles, although occasionally it is partial or unilateral. The affected muscles feel firm, and offer resistance to passive movements of the limbs, which is sometimes so great as to amount to tetanic rigidity, but when once this resistance is overcome the limbs, head, and neck, or features may be placed in constrained positions, which they retain for a comparatively long period. After the first resistance of the muscles has been overcome the limbs possess a flexibility and pliability, which has been compared to that of soft wax, and the condition has consequently been named *flexibilitas cerea*. The resistance of the muscles enables the limbs to be moulded in any position compatible with the rigidity of bones and inextensibility of ligaments, and the constrained attitudes in which the limbs may be placed are maintained without change during the whole course of the attack. But even during the cataleptic condi-

tion the muscular stiffness does not persist in its full intensity for a lengthened period. After some minutes the stiffness diminishes somewhat, so that the arm, for instance, when raised horizontally falls lower by its own weight, and the limb undergoes a slight trembling, indicating the approaching exhaustion of the muscles.

Consciousness is usually abolished, but not in all cases. A certain amount of consciousness may be retained in the early stage of the attack or be present throughout, so that strong peripheral irritation may cause pain which will be remembered by the patient. Reflex irritability is sometimes lost; at other times certain reflex actions, such as closure of the eyelids on touching the conjunctiva, are retained. The electric contractility of the muscles, according to Benedikt, is somewhat diminished during the attack, but soon becomes normal, and in a case observed by Rosenthal the electrical reaction of the nerves to both currents was perfectly normal, in another it was increased.

The organic functions are not usually seriously interfered with. The respiration may be normal, but is generally slow and shallow. The pulse is slow, soft, and compressible. The temperature is generally lowered, and at times the surface of the body becomes icy cold. When the surface of the body is cold, and the pulse at the wrist and respiration are almost imperceptible, the condition may be mistaken for real death.

The attack of catalepsy is sometimes very brief, lasting only a few minutes, at other times several hours or days. Attacks described as being very protracted are in reality made up of a succession of these, separated from one another by intervals in which the patients recover either wholly or partially. The seizures sometimes disappear quite suddenly, and the patients at once recover full consciousness and immediately resume the actions which had been interrupted. As a rule, however, recovery is gradual, patients at first being stupefied as if awaking from a profound sleep, a certain amount of muscular stiffness remaining for some time, which renders motion difficult and slow.

In simple catalepsy no mental disorder is observed in the intervals between the attacks, but when it is merely a symptom of profound nervous disease the intervals may be characterized by the occurrence of hysterical convulsions, delirium, maniacal attacks, and hallucinations, or the catalepsy may be associated with ecstasy and somnambulism.

The course of catalepsy is usually chronic, extending over many years. Some individuals suffer only from a small number of attacks separated by intervals of many years. Others, again, have frequent periodical attacks. In hysterical catalepsy the slightest external influence may suffice to provoke a paroxysm. Cataleptic attacks are hardly ever fatal of themselves.

f. Trance.

In trance the patient lies for days together without eating or drinking, there is apparent insensibility, and the pulse and respiration are imperceptible, but the limbs remain flexible and the tonic rigidity of catalepsy is absent. The state of complete insensibility is not, however, continuous, inasmuch as there occur periods during which the patient may see, hear, and remember all that goes on about him, and may partake of small quantities of food. The condition of the patient is not, indeed, unlike that of a hibernating animal. In the state of trance the patient usually lies in a warm room, well covered with clothing, so that little heat is lost by radiation; the mental functions are in abeyance, indicating that the molecular changes which are the correlatives of mental actions have ceased, and all muscular movements are suspended with the exception of the cardiac contractions, and slight respiratory movements, and under such circumstances the amount of waste must be small. The physician must, of course, be on his guard against deception in cases of trance.

g. Ecstasy.

This condition is closely allied to trance, the patient being insensible to outward impressions in both. In ecstasy the mind is absorbed with some fixed idea, generally of a religious character, and the patient becomes oblivious of surrounding events and objects. The limbs are motionless, and often fixed in maintaining a particular attitude, the breathing is slow and feeble, the pulse is almost imperceptible, the eyes are often bright and animated, and the countenance has an expression of rapture.

h. Somnambulism and Hypnotism.

In *somnambulism* the patients appear to be wholly unconscious, yet they walk, climb, and avoid obstacles, and may manifest greater strength, agility, and precision of muscular adjustments than during waking hours.

Hypnotism or *mesmerism* is, as Maudsley remarks, a kind of artificially induced somnambulism. The subject, who is probably always of a neurotic temperament, is induced to look steadily at the operator, the latter attracting his attention by making a few gentle "passes" with his hand. Mr. Braid, of Manchester, directed the person to look upon a disk or some bright object held in front of and a little above the level of the eyes. After a short time there is a slight tremor of the eyeballs

of the subject, his pupils dilate, and he falls into the mesmeric condition. In this state the mental functions are abolished, and all the actions of the subject are afterwards determined by the suggestions of the operator. Under the influence of these suggestions, the subject may sing, recite poetry, and perform the most absurd and outrageous actions. He may be made to eat a raw cabbage, amidst all the outward signs of enjoyment, to appease a suggested hunger; he may spit out pure water given him to drink with all the signs of disgust, on the suggestion that it is bitter and nauseous, or drink infusion of wormwood with apparent relish on being told that it is an agreeable beverage; or he may be made to sneeze violently on being asked to take a pinch of snuff from an empty box. Hysterical patients may be thrown into a condition of trance or of catalepsy, or one half of the body may be thrown into trance and the other half into catalepsy, by being made to look upon a bright light.

Morbid Anatomy and Physiology.—Morbid anatomy has not yet thrown much light upon the nature of local spasms. These spasms are most liable to occur in muscles which are engaged in special actions, such as the facial muscles, the rotators of the head and neck, and the small muscles of the hand. These local spasms are most probably caused by irritation of some part of the reflex loop. The irritation is probably sometimes direct, but it is undoubted that it is frequently of reflex origin. The reflex nature of these spasms is well illustrated in some cases of facial tic. When motor-arresting points are obtained in such cases they are observed not in the course of the facial nerve, but over certain points of the trigeminal nerve. The nature of these spasms will, however, be best illustrated by a detailed reference to the pathology of the professional hyperkineses. The combinations of muscular contractions concerned in writing are exceedingly complex, but some of these combinations are more special than others. The operation of writing is divisible into the acts of (1) prehension of the pen, (2) moving the pen, (3) poising the hand and forearm. Of these acts the first and the third are the most special, and they are certainly the most liable to be affected in writer's cramp. The act of prehension is mainly effected by the muscles of the ball of the thumb and the interossei, and spasm, weakness, and incoördination of the movements of these muscles are the more frequent symptoms of the disease. Poising of the hand and forearm is effected chiefly by the pronators and supinators of the forearm, and as the hand is always held in a position in which gravity aids the pronators, the most delicate part in preserving the attitude is thrown upon the supinators, and a sudden jerking of the latter muscles causing the hand to roll outwards is not an unfrequent

symptom of the disease. Professional cramps are doubtless caused by a functional or molecular lesion of some part of the nervous mechanism which regulates the actions of the muscles executing the movements which are disordered. It is very likely that professional cramps are sometimes caused in the same way as cramp of the calf by irritation of the intramuscular nerve endings, or the end plates in the muscular fibres. It is possible that at other times the seat of the lesion is the mechanism of cells and fibres in the anterior gray horns of the spinal cord which regulates the movements concerned in writing, while in still other cases the lesion is probably situated in the cortical centre, or in the course of the centrifugal conducting paths which connect it with the spinal cord. We should say that the chief lesion is situated in the ganglion cells of the spinal cord, or in the fibres which connect them with the muscles in all those cases in which the electrical reactions of the muscles are diminished, and that it is situated either in the cortex or in the cerebral conducting path above the spinal level in all those cases in which the electrical reactions are increased, especially if this increase is maintained when the disease is of long standing.

Saltatory spasm is here placed amongst the spino-peripheral spasms, because a case recorded by Bamberger appears to indicate that the excess of reflex action in such cases originated in the skin. The spasms are, however, regarded by Frey as being caused by undue tension and stretching of the muscles, and it is, therefore, possible that some of of the recorded cases are caused by exaggerated tendon-reactions. A few of the recorded cases appear to have been of hysterical origin, while in others the presence of contractures shows that the affection was caused by some form of lateral sclerosis. It is, indeed, very probable that very different affections have been grouped together under this name.

Tetanus is here placed amongst the spino-peripheral spasms because there can be little doubt that increased irritability of the gray matter of the spinal cord plays a very important part in the production of the symptoms of idiopathic and traumatic tetanus as well as of the tetanic spasms which are caused by strychnine and other poisons. Lockhart Clarke found great hyperæmia of the spinal cord and its membranes as well as foci of softening in the gray and white substance of the cord in cases that died from tetanus, and these observations have been confirmed by numerous other investigators. Similar changes have also been found in the medulla oblongata. It is, however, possible that the changes which have been discovered were caused by the great congestion of the cord and membranes which must have resulted from the violent attacks of spasm. Some pathologists believe that tetanic seizures are caused by an increased irritability of the gray matter of the spinal

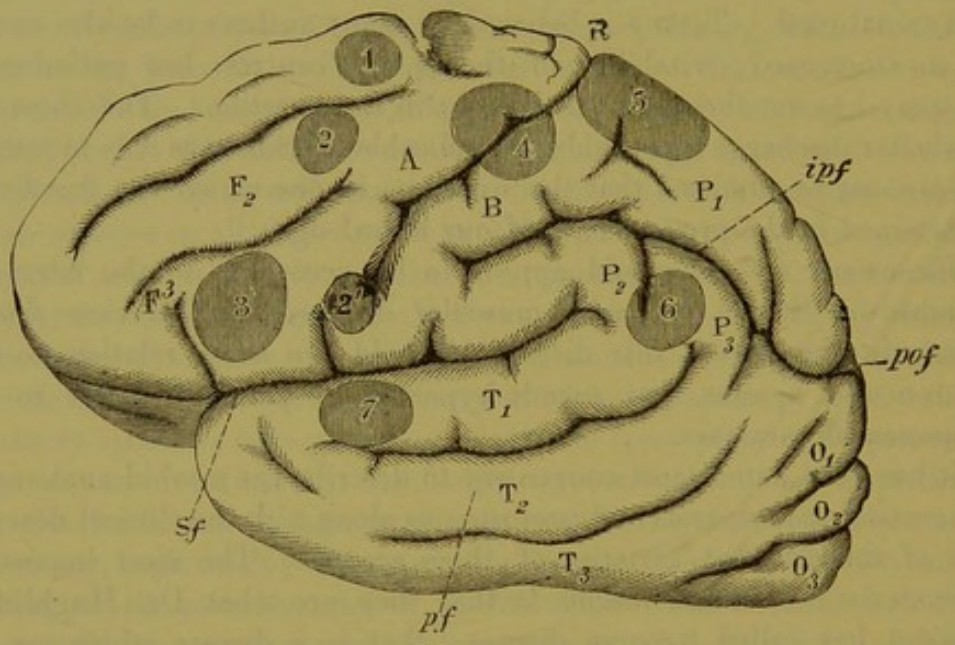
cord and of the medulla oblongata. This increased irritability of the spinal centres not only raises the irritability of the reflex arcs, but also gives rise to spontaneous discharges of energy, and both of these conditions are doubtless present in tetanus. I am, however, inclined to believe with Dr. Hughlings-Jackson that discharges of nerve energy from the cortex of the cerebellum are the main cause of the paroxysms of spasm in tetanus, although these discharges may to some extent be determined by the instability of the centres in the medulla oblongata and spinal cord. Tetany is believed by many authors to be also caused by an increased irritability of the spinal centres, but pathological anatomy has not thrown much light upon the question. The theory of cerebellar discharge is probably as applicable to tetanus as it is to tetany, but it must be admitted that the question is one which can hardly be determined in the present state of our knowledge.

Thomsen's disease would appear to be, according to the latest researches of Erb, a primarily muscular disease. If this view should turn out to be correct, this disease will hold the same relation to the spino-neural spasms that pseudo-hypertrophic paralysis bears to the spino-neural paralyses.

It has been found most convenient to describe the morbid anatomy of the cortical monospasms and protospasms along with the clinical description of the different varieties of these spasms. The most important characteristic of these lesions is that they are what Dr. Hughlings-Jackson has called "coarse disease," that is, a disease which can be recognized by the naked eye. It now remains for us to connect the morbid changes with the symptoms. When an irritative lesion is situated in or near the motor area of the cortex of the brain the surrounding parts are more freely supplied with blood and the gray substance, after absorbing a large stock of nourishment, discharges its accumulated energy at periodical intervals, thus giving rise to paroxysms of convulsions of the muscles whose actions are regulated from that part of the cortex. Every excessive discharge of energy is followed by exhaustion of the motor centres near the irritative lesion, and consequently each convulsive paroxysm is followed by a transient paralysis of the affected muscles. The lesion may destroy a portion of the cortex while maintaining the neighboring portions in a state of irritation, and consequently cortical monospasms are often accompanied by a permanent paralysis of some groups of muscles, and the convulsive paroxysm always begins in the neighborhood of the paralyzed muscles. The annexed diagram (Fig. 97) will readily explain the order in which the spasm progresses in the chief varieties of unilateral convulsion. If the lesion is situated near the longitudinal fissure between the fissure of Rolando the spasm

will begin in the leg, and as the wave of molecular disturbance extends, the arms are next involved and the face last. If the lesion is situated near the frontal convolution the spasm will begin in the muscles of the face, those of the hand are next involved, and those of the leg last. If the lesion is situated about the sulcus of Rolando, the spasms will begin in the muscles of the arm and hand, those of the face are involved

FIG. 97.



EXTERNAL CONVEX SURFACE OF THE HUMAN BRAIN.

Fissures: R, Fissure of Rolando; *Sf*, Fissure of Sylvius; *pf*, Parallel fissure; *ipf*, Interparietal fissure; *pof*, External parieto-occipital fissure. *Convolutions and Lobules:* A, Ascending frontal; B, Ascending parietal convolutions; F_1, F_2, F_3, First, second, and third frontal convolutions; P_1, Superior parietal lobule; P_2, Supra-marginal gyrus; P_3, Angular gyrus; O_1, O_2, O_3, First, second, and third occipital convolutions; T_1, T_2, T_3, First, second, and third temporo-sphenoidal convolutions. *Motor Centres:* 1, Movements for rotation of head and neck; 2, Movements of the upper facial muscles; 2', Movements of the lower facial muscles; 3, Movements of the tongue and jaws; 4, Movements of superior extremity; 5, Movements of inferior extremity; 6, Movements of the ocular muscles; 7, Movements in relation with the sense of hearing.

next, and those of the leg last. When the lesion is situated wholly in the motor area consciousness is either not much affected, or it is lost at a comparatively late period of the spasm, but when it is situated in the prefrontal area of the cortex, consciousness is lost before the commencement of the spasm or soon after it begins. And when the lesion is situated in the occipital or temporo-sphenoidal lobes, the spasm is preceded by some disturbance of sight or of hearing respectively.

In idiopathic epilepsy no constant anatomical changes have been found in the nervous system, but such changes as have been discovered, like atrophy of one of the hippocampi majores, have generally affected

the cortex of the brain. The lesion of idiopathic epilepsy is, therefore, regarded as a *molecular* disturbance.

Dr. Todd was the first to suggest that epilepsy was caused by an explosion of nerve force, but this theory has assumed a definite shape in the hands of Dr. Hughlings-Jackson, who advanced the opinion that all convulsions which are attended by loss of consciousness are caused by discharging lesions of the cortex of the brain. This hypothesis has received a considerable amount of experimental verification. Hitzig was able to determine epileptic paroxysms by various injuries to the cortex of the brain, and Ferrier obtained a similar result by passing a faradic current of moderate intensity through the cortex, the electrodes being widely separated, so that a large portion of the cortex was included in the circuit. According to this theory, the convulsions are caused by a large discharge of nervous energy from the cortex of the brain along centrifugal nerve paths, and the loss of consciousness is caused by the temporary exhaustion which succeeds to an excessive nervous discharge. But we have seen that the paroxysm of unilateral epilepsy is always followed by a temporary paralysis of the convulsed limbs, and it may be asked why it is that the paroxysm of general convulsions is not likewise followed by a temporary paralysis? The reply is that it is so followed. After an epileptic attack there is complete muscular relaxation, but as the patient is at the same time in an unconscious condition the degree of paralysis which is present cannot be estimated, and even after consciousness is regained general muscular feebleness often remains for a time, which although not called paralysis is really paralytic in nature. The unseemly and apparently immoral actions performed, and the atrocious crimes often committed by patients after minor attacks of epilepsy, may be explained on the supposition that the inhibitory influence of the highest centres is temporarily suspended, thus permitting the centres which preside over automatic actions and animal instincts to spring into greater activity.

Many of the spasmodic phenomena of hysteria are best explained on the supposition that the irritability of the cortex of the brain is increased, whilst others are best explained by supposing that the irritability of the cortex diminishes. Hysterical attacks are caused by a diffused discharge of energy from the cortex of the brain, while local spasms are sometimes caused by a local discharge from one of the motor centres, and at other times, especially when they assume the form of persistent contracture, by paralysis of one of these centres, removing the inhibitory action of the brain, and thus permitting the unrestrained action of inferior ganglia. Very little beyond conjecture is known with regard to the pathology of catalepsy, trance, ecstasy,

somnambulism, and hypnotism, but there can be but little doubt that these curious conditions are caused by a molecular disturbance of the cells and fibres of the cortex of the brain, or of the conducting paths which lead from the surface of the body to the cortex. Hysterical hemianæsthesia, for example, is best explained by supposing that the conduction of the centripetal fibres in their ascent through the internal capsule is temporarily arrested. Suppose that a complete bilateral hemianæsthesia exists, what would be the condition of the patient? There would be complete loss of every form of cutaneous and muscular sensibility as well as of sensation in the bones and joints, there would be loss of taste on both sides of the tongue, and of smell in both nostrils, and instead of there being amblyopia and partial deafness on one side as in hemianæsthesia, there would be complete blindness and deafness on both sides, inasmuch as, according to the hypothesis, the sensory centres in both hemispheres either have ceased to act, or the impressions made upon the peripheral sense organs fail to be conducted to them. But impressions made on the periphery would, however, reach the cortex of the brain through the optic thalamus, and the subject of bilateral hemianæsthesia, although effectually cut off from the external world so far as the anatomical substratum of consciousness is concerned, would perform various complicated actions in response to peripheral impressions, but without being attended by consciousness. The state of the patient would, indeed, be very similar to that observed in somnambulism, the mesmeric state, and various post-epileptic and allied conditions.

Treatment.—The first aim of treatment is to withdraw the exciting cause of the spasm, and with this view all sources of direct irritation such as tumors in the vicinity of the nerve, of reflex irritation such as conjunctivitis in blepharospasm, carious teeth in facial spasm, and irritating substances in the atmosphere in sneezing, and of remote irritation such as uterine disease in facial spasm or spasmodic torticollis, must be removed. The source of irritation may be found in impure blood, and if the patient be rheumatic salicylate of soda or iodide of potassium, and if scrofulous, carbonate of iron and cod-liver oil should be administered. When the local spasm is a mere expression of a more general nervous disease such as hysteria or chorea, the treatment appropriate for the central disease must be adopted. The most likely internal remedies for such cases are arsenic, bromide of zinc, and bromide of potassium. The local application of chloroform and ether spray, and the subcutaneous injection of morphia, will be found useful in many cases to allay the distress caused by the spasm. The best local treatment, however, is afforded by the application of the constant cur-

rent. A descending current of moderate intensity may be passed along the affected nerve, and when points of arrest are obtained the anode should be placed over one of these. The antagonists of the affected muscles may be strengthened by the use of the faradic current, but the interrupted current should never be applied directly to muscles which are the subjects of spasm. Some cases of spasm are benefited by the application of a strong galvanic current over the vertebral column, the part of the cord from which the affected nerve is derived being included in the circuit. Continuous pressure over the affected nerve by means of a compress or suitable mechanical appliance at a point at which it emerges from a bony canal or becomes superficial has been found useful in the treatment of spasm. In some cases the pressure is more successful when applied over one of the "spasm-arresting points" of the fifth nerve in facial spasm, in the length of the affected muscles in other cases.

Various surgical operations have been resorted to for the cure of local spasms. Subcutaneous division of muscles and of tendons is most likely to be of use in the tonic form of spasm. Division of the spinal accessory nerve has been successful in several cases of spasmodic torticollis, but section of the facial nerve causes paralysis of such a large number of important muscles that it is hardly a justifiable operation. Stretching of the nerve has, however, been attended with very favorable results in several cases. In spasms of groups of muscles of the extremities various mechanical contrivances may be employed in order to force and maintain the limb in a normal position. In spasmodic torticollis the head may likewise be maintained in the normal position by means of a Sayre's jacket with jury-mast. Another method is to surround the head and the trunk each by a plaster-of-Paris bandage, and to connect the two by a strong India-rubber band passing obliquely from one side to the other in such a position as to counteract the spasm of the affected muscle. Professional cramps must be treated on the same general principles as other forms of local spasm. In slight and recent cases rest for a period of from two to three months will alone suffice for a cure, but this period must be prolonged to from six to twelve months in severe cases.

The most successful treatment of writer's cramp appears to be that practised by Wolff, which is a combination of gymnastics and massage. The gymnastic exercises consist of both active and passive movements. In the active form of exercise the patient is instructed to execute, three or four times a day, a series of vigorous movements with the affected extremity, the hand being opened and closed in quick succession. The number of these movements, and consequently the duration of each exercise, is progressively increased until a duration of about half an

hour is attained for each sitting. In the passive movements the operator makes forcible traction three or four times a day upon each of the affected muscles separately in the direction of its length. This appears to be the most delicate part of the treatment, inasmuch as if too little strength is employed the cure is delayed, and if too much the disorder is aggravated. When the spasm is notably diminished, which usually occurs in a short time, the patient is encouraged to take slow and graduated exercises in writing. The operator practises daily massage of the affected extremity, particular stress being laid upon percussion over the affected muscles with the ulnar border of the hand. When no amelioration of the symptoms is produced in four or five sittings, Wolff believes that the treatment may be abandoned as not likely to prove useful. The treatment by massage is applicable to spasms of other muscles, and several cases of spasmodic torticollis in which I prescribed it, were much benefited.

Mechanical means have been resorted to in severe cases. The simplest method of this kind is to insert the pen into a cork or thick piece of wood, or to fasten it by means of a ring to the first or middle finger. Many patients are relieved by applying a narrow bandage or a strip of court-plaster firmly round the wrist.

Tenotomy of the affected muscles has been performed, but the results obtained have not been very encouraging. The treatment of other *professional spasms* must be conducted on essentially similar principles. Very little is known with regard to the treatment of saltatory spasm, but the agents most worthy of trial are morphine, bromide of potassium, calabar bean, ergotine, conium, and atropine.

In tetanus the great aim of treatment is to diminish the irritability of the gray matter of the spinal cord. The agents which have been used for this purpose are chloral hydrate, bromide of potassium, calabar bean or cannabis indica, nicotine, chloroform, and morphine. The cold bath and cold douche may be used when there is a sudden elevation of temperature, and under these circumstances the patient may be placed in a bath of about 90° F., and the temperature should be gradually reduced to 60° F. by the addition of cold water. In ordinary cases a warm bath is soothing to the patient. The patient should be protected from every source of irritation, such as noises, strong lights, changes in temperature, and sudden contact. His strength should be well supported, and when he is unable to swallow liquids, owing to the spasm, food should be introduced by means of a tube passed through the nose after the patient has been brought under the influence of chloroform.

In tetany the causes of the disease must be carefully inquired into

and removed when possible. The diet should be full and generous, and when the spasm is allayed change of air is indicated. During a severe paroxysm it may be necessary to use chloroform inhalations, opiates, or belladonna, and bromide of potassium, either alone or in combination with chloral, may be a useful adjunct to treatment. The galvanic current to the contracted muscles has often proved useful.

In unilateral convulsions the treatment must be directed against the primary disease, and the possibility of syphilis should never be forgotten. In the treatment of functional spasmodic affections the greatest care must be taken to discover and remove any source of peripheral irritation, and in hysteria great attention must be paid to the treatment of ovarian and uterine disorders.

In the treatment of epilepsy, it has been found that bromide of potassium is the most useful drug, and to do good it must be given in doses ranging from ten to forty grains three times daily. Large doses of this salt are apt to produce an eruption of acne, which soon subsides when the drug is discontinued, and it may possibly be prevented by combining arsenic with the bromide. The bromide of sodium or of ammonium may be used instead of the potassium salt, as being less depressing in its effects. Chloral hydrate is sometimes a useful adjunct to the bromide of potassium. The salts of zinc have also proved useful in the treatment of epilepsy, and they appear to be more efficient with patients under twenty years of age than with those of maturer years. The oxide may be given in doses of from two to five grains three times daily, or the sulphate may be administered at first in doses of three grains, and progressively increased to scruple doses three times daily. The bromide of zinc has been given in gradually increasing doses until a scruple is taken three times a day. The oxide of zinc may be combined with extract of belladonna or hyoscyamus, or with the powdered root of valerian. Belladonna, or atropine and digitalis have been found useful in the treatment of epilepsy, either alone or in combination with one of the bromide salts.

The treatment of eclampsia must vary according to the cause of the convulsion, but during the attack the inhalation of chloroform is the most generally useful method of treatment for arresting the spasm.

The general treatment of hysteria is too wide a subject to be discussed fully in this place. Bromide of potassium is found very useful in the treatment of hysterical spasms, but the administration of iron and other tonics, with attention to the health of the patient, is much more generally useful than any special remedies. The most important part of the treatment, however, consists of the moral management of the patient, and hysterical patients are treated with much greater

success in the wards of a hospital than in their own homes. In aggravated cases, therefore, the patient should be removed from her home and relations, and placed for a period of some months either in a public institution or under the care of complete strangers. The method of seclusion from her friends was first, so far as I know, strongly insisted upon by Dr. R. Brudenel Carter, and it forms a principal factor in the treatment of hysterical neurasthenia, which has been elaborated by Dr. S. Weir Mitchell, and which has already been described. "The attacks," says Dr. Carter, "will, in all probability, occur during a meal, or when there are strangers present, or at some most inconvenient time and place, and it may, on this account, be necessary to have the patient removed to her bedroom. In such case she should be carried there as quickly as possible, placed upon the floor, and immediately left quite alone, the door being shut, and no one being suffered to open it on any pretext whatever until the patient does so herself. But if the room in which the attack takes place can be spared for a few hours, it should be closed and shut up in the same manner, and in either case especial care must be taken not to give utterance to a single expression, either of sympathy or alarm. After the lapse of a longer or shorter time, often at a meal, and sometimes not until the next morning, she will present herself as usual, and will perhaps offer some apology or express some regret for her illness. This should be graciously received, and then every attempt on her part to return to the subject must be carefully and industriously foiled, no inquiries being made about her health, and all complaints being interrupted by the introduction of ordinary conversational topics." Treatment by isolation is only to be recommended in very aggravated cases, and after other measures have failed. In ordinary hysterical attacks the patient takes care not to injure herself, but it is often necessary to prevent respiration being impeded by clothing. In my experience it is often desirable to prevent a hysterical woman from tossing about violently in all directions. The best method of preventing the patient's struggles is to place an attendant on each side of her, and to direct each to grasp one wrist with one hand, and to hold the shoulder firmly down to the bed or floor with the other, and if necessary the legs must be held down by a third attendant. It is remarkable how soon a hysterical patient ceases to struggle when she finds that she is held as in a vice. So long as the patient can shake herself free from her attendants, so long is the struggle likely to be maintained, but when once she feels that she is thoroughly restrained, and that her efforts do not find outward expression, she generally ceases to struggle. The most usual remedy for arresting the attack is to dash cold water on the face and neck, and the plan suggested by Dr. Hare, of forcibly

holding the mouth and nose of the patient so as to prevent her from breathing, is still more effectual. An emetic of sulphate of zinc is also very successful in arresting the attack.

Local spasms in hysteria are best removed by general treatment and moral management, although local treatment and special remedies are occasionally found useful. The most useful local remedies are the cold douche and faradic current.

In the treatment of catalepsy and allied conditions the best results have been obtained by the use of iron and other tonics, ergot, the cold douche, and the faradic current. In protracted cases artificial feeding by the stomach pump and nutritious enemata must be employed.

CHAPTER V.

ATROPHIC PARALYSES.

It has already been shown that the various forms of paralysis may be divided into spino-peripheral or atrophic, and cerebro-spinal or spasmodic paralyses. The spino-peripheral paralyses with which we have to do at present may be divided into (I.) simple neural paralyses caused by local disease or injury of individual motor or mixed nerves; (II.) multiple neural paralyses caused by disease affecting many of the peripheral nerve branches and often assuming a progressive character; (III.) reflex paralyses caused by irritation of afferent nerve fibres; (IV.) spinal atrophic paralyses caused by disease of the ganglion cells of the anterior gray horns of the spinal cord, and to these may be added (V.) certain forms of myopathic paralysis.

I. SIMPLE NEURAL PARALYSES.

1. Paralysis of the Oculo-motor Nerves.

The oculo-motor nerves are the oculo-motorius, trochlearis, and abducens, and the subjoined diagram (Fig. 98) will suffice to remind the reader of their course and distribution.

a. Disorders of the Movements of the Eyelids.

(1) The *levator palpebræ superioris* is but seldom affected by tonic spasm as an isolated affection, and hardly ever by clonic spasm. Tonic spasm of this muscle causes the lid to be drawn upwards, and there is consequent inability to close the eye, either by voluntary effort or during sleep. The orbicular muscle is supplied by the facial nerve, and spasm of it will be subsequently described.

(2) *Paralysis of the levator palpebræ superioris, or ptosis*, may occur as a separate affection, but it is usually associated with paralysis of the superior rectus, both muscles being supplied by the superior branch of the oculo-motor nerve. The upper lid hangs motionless, the palpebral aperture is greatly narrowed, the lid remains motionless when the eye is directed upwards, and the horizontal wrinkles of the upper lid are effaced.

(3) *Disorder of the Associated and Mechanical Movements of the Muscles of the Eyelids.*—When the eyes are directed upwards in health there is an associated contraction of the elevators of the lids and the upper eyelids are retracted, and when the eyes are directed downwards there is a simultaneous downward movement of the upper eyelids, and

FIG 98.

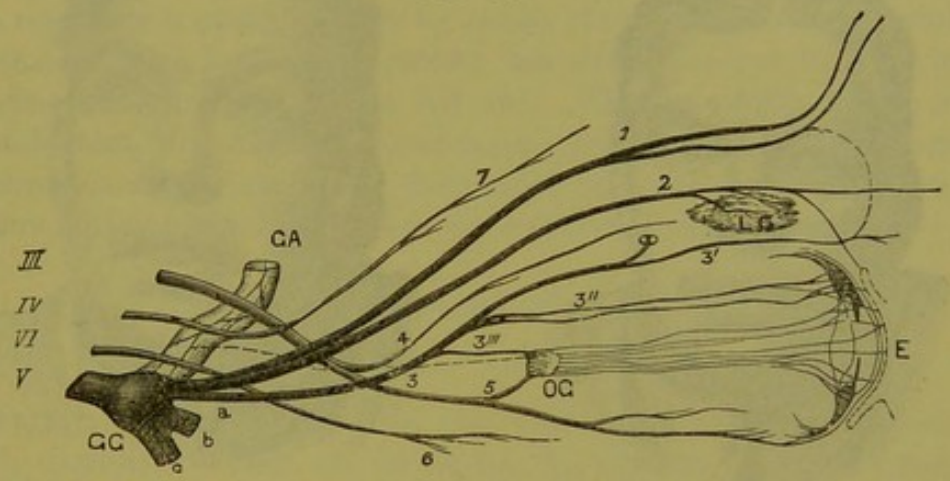


DIAGRAM OF THE FIRST OR OPHTHALMIC DIVISION OF THE FIFTH, SHOWING ALSO THE THIRD, FOURTH, AND SIXTH CRANIAL NERVES. (From HERMANN'S "Physiology.")

V, Sensory root of fifth nerve.
GG, Gasserian ganglion on larger root of the fifth nerve.
a, Ophthalmic division of the fifth nerve.
1, Frontal nerve. 2, Lachrymal nerve. 3, Nasal nerve.
LG, Lachrymal gland.
3', Infra-trochlear branch of *nasal nerve*.
3'', *Long ciliary* branches of *nasal nerve*.
3''', Branch of *nasal nerve* to ophthalmic ganglion.
OG, Ophthalmic ganglion.
b, Second division of the fifth cut across. *c*, Third division of fifth cut across.
III, Third nerve (motorius oculi).
4, Upper division of third nerve. 5, Lower division of third nerve, near point where it gives the *short root* to the ophthalmic ganglion.
IV, Fourth nerve (n. trochlearis).
7, Its fibres passing to the *superior oblique*.
VI, Sixth nerve (n. abducens).
6, Its fibres passing to *external rectus*.
CA, Carotid artery.
E, Vertical section through anterior part of eyeball; conjunctiva indicated by dotted line.

to a less extent of the lower lids also. The downward movement of the upper eyelid is most probably caused by an inhibitory cerebral influence being sent to the elevator of the eyelid and superior rectus muscles simultaneously with the impulse to contraction sent to the inferior rectus, while the depression of the lower eyelid is probably caused by pressure of the cornea against it. It has been shown by Dr. Gowers that these movements are disordered in partial paralysis of the third

nerve. In a case of the kind under my care the patient in looking forwards manifests a slight external squint of the right eye (Fig. 99), while the palpebral aperture on that side is a little smaller than the other

Fig. 99.

Fig. 100.

one. When the patient looks up (Fig. 100) the left or healthy eye is rotated upwards, and there is a simultaneous upward movement of both eyelids on that side, but, as usual, the eye has gained on the lower lid,

Fig. 101.

so that a considerable portion of the sclerotic between the lower margin of the cornea and the edge of the lid is uncovered. The right eye, however, has scarcely moved, and the lids have also remained stationary.

But the most striking deformity occurs when the patient looks down. The left upper eyelid moves downwards in association with the downward rotation of the eyeball, but the right eyelid and eyeball remain comparatively stationary, and the patient presents the singular appearance of looking with the left eye at an object lying at her feet, while the right eye seems to be looking at an object almost on a level with the eye (Fig. 101). This disorder in the movements of the eyelids of the right side is caused neither by spasm of the levator nor by paralysis of the orbicular, because the patient can close that eye with as much readiness and firmness as the left one. The immobility of the right eyelids must, therefore, be caused mainly by an arrest of the associated contraction and relaxation of the levator palpebræ, which respectively occurs during upward and downward rotation of the eyeball.

b. Paralysis of the External Muscles of the Eyeball.

General Etiology.

Paralysis of one or more of the ocular muscles may be caused by cold, exposure of the eye to strong impressions of light, excessive smoking, alcoholic excess, blows on the eye, penetrating wounds of the orbit, fracture of the skull, and compression of the nerves by tumors. But the most frequent and important causes are syphilitic deposits and the degeneration which is the anatomical substratum of locomotor ataxia. Ocular paralysis is also caused by diphtheria, and may occasionally be a sequel of other acute diseases.

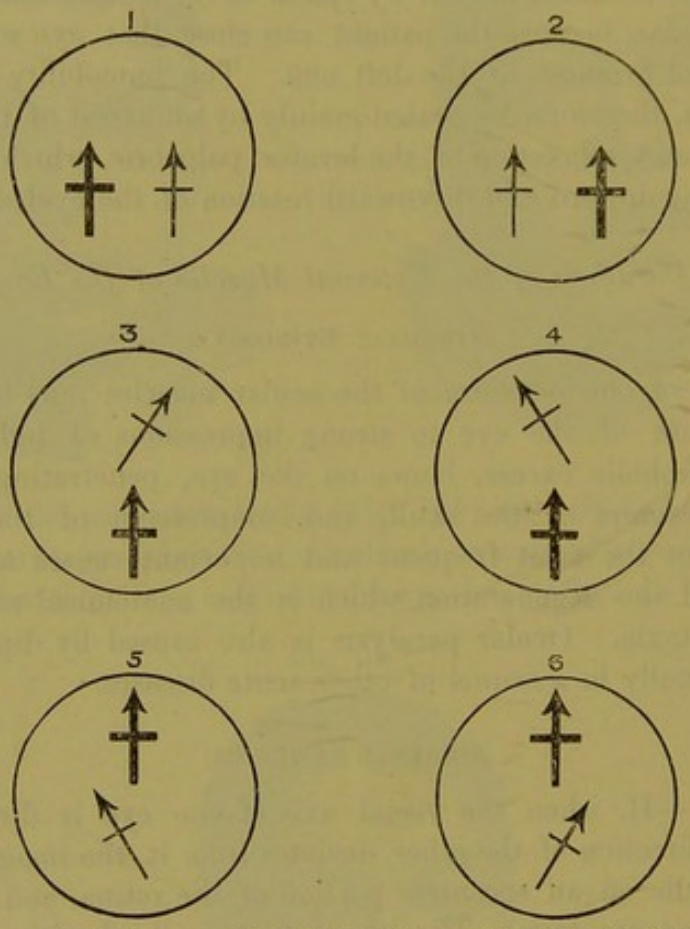
General Symptoms.

Diplopia.—If, when the visual axis of one eye is directed to an object, the direction of the other deviates from it, the image in the distorted eye falls on an eccentric portion of the retina, and two objects instead of one are seen. The image seen on the healthy eye falls on the macula, and is therefore distinct, but the image seen on the distorted eye forms upon a more or less peripheric region of the retina, and is therefore faint and more or less confused; the former is consequently called the *true*, and the latter the *false* image.

Neutralization of the False Image.—When the paralysis is of old date the patient learns to perceive objects only with the healthy eye and the diplopia disappears, whilst the distorted eye, from long-continued disuse, suffers consecutive amblyopia. The diplopia may, however, be made to reappear by placing before the healthy eye a colored glass, which differentiates the true image and permits that of the distorted eye to be perceived.

Relation of the Images to One Another.—The false image always occupies, with reference to the axis of vision, an opposite position to the distortion of the eye. Assuming the left eye to be affected, the false image is displaced horizontally to the patient's right in outward squint (Fig. 102, 1), horizontally to the patient's left in inward squint (Fig. 102, 2); the false is above the true in downward (Fig. 102,

FIG. 102.



In the above diagram the thick cross represents the *true image*, the thin cross the *false image*. The left eye is supposed to be affected in all of them. (After BRISTOW.)

3 and 4), and below it in upward squint (Fig. 102, 5 and 6). In outward or inward squint the images are vertical and parallel with one another, but in all other forms of squint the oblique muscles rotate the distorted eyeball and the false becomes tilted with reference to the true image (Fig. 102, 3, 4, 5, and 6), the false image being tilted in the opposite direction to the rotation of the globe. The distance between the images becomes greater as the object is moved in the direction of the action of the paralyzed muscles.

Vertigo.—Diplopia causes considerable embarrassment to the patient whilst walking, which disappears in great part when the distorted eye

is closed. In ascending a stair, for instance, he sees two steps for every one, and not knowing upon which to place his foot becomes confused and stumbles. Perception of distance is defective, and the patient becomes confused on endeavoring to grasp objects. This constant confusion causes great fatigue, vertigo, and sometimes vomiting.

Compensating Attitudes.—The patient instinctively neutralizes the perception of double images by placing the head in such an attitude that the paralyzed muscle does not require to act, and then the visual axes can be converged upon objects.

Secondary Deviation of the Sound Eye.—This condition has already been described.

False Projection.—When a person looks at an object with one eye only, a judgment of its position in space is formed by the sense of effort made to fix the object. If a patient with paralysis of the external rectus of the left eye be asked to touch an object coming before him from the left or paralyzed side, he misses it by carrying his finger too far to the left, or to the side of the object corresponding to that of the paralyzed muscle. The difficulty of immediately recognizing the position of objects in space, called *false projection*, is caused by the fact that it is necessary to make an increased effort with the affected eye, so that the amount of rotation is overestimated, and consequently an erroneous judgment of the position of the body in space is formed.

Secondary contraction of the antagonistic muscles often occurs; it increases the extent of the duration, and augments the distance of the double images from one another.

Symptoms of Special Forms of Paralysis.

(1) *Complete paralysis of the oculo-motorius* gives rise to paralytic exophthalmos, ptosis, divergent strabismus, dilatation and immobility of the pupil, and impairment of the power of accommodation. Every effort to move the eye in any direction causes it to rotate outwards and downwards, and it gradually becomes fixed in this position by the secondary contraction of the external rectus and superior oblique. Double images appear over almost the whole field of vision; the false image is to the right when the left eye is affected, the two images are on a level when the object is in the horizontal position, and the false appears below the true image when the object is below, and above it when the object is above the horizontal line. Fixation is only possible in an outward and downward direction, the compensating attitude of the head is a very oblique position backwards and towards the healthy side, and secondary deviation of the eye takes place in all directions except in that towards the affected eye.

(2) *Incomplete Paralysis of the Oculo-motorius.*—(*a*) *Paralysis* of *the superior rectus* gives rise to *strabismus dorsum vergens*, in which the affected eye is rotated downwards and a little outwards. The false is seen above the true image and tilted to the patient's right when the left eye is affected (Fig. 102, 3); the vertical distance between the images increases according as the eyes are directed upwards and outwards, while the false image disappears at the horizontal line, provided there be no secondary contraction of the inferior rectus. When the eyes are directed downwards objects are seen single, and the head is thrown back in order to counteract the paralysis.

(*b*) *Paralysis of the internal rectus* gives rise to *strabismus divergens*, and the eye cannot be rotated inwards beyond the middle line. The images are vertical, parallel, and on the same plane; the lateral distance between them increases as the object is moved towards the sound side, and the false image is to the patient's right when the left eye is affected (Fig. 102, 1). There is secondary deviation of the sound eye outwards, and in fixing an object the head is turned towards the healthy side.

(*c*) *Paralysis of the inferior rectus* gives rise to *strabismus sursum vergens*, in which the affected eye is directed upwards and slightly outwards. The false is below the true image and tilted to the patient's left when the left eye is affected (Fig. 102, 5). The false image disappears when objects are held above the horizontal line, but double vision reappears whenever the line of vision is lowered, and consequently this form of paralysis is very troublesome in walking and in all kinds of handiwork.

(*d*) *Paralysis of the inferior oblique* gives rise to a squint in which the eye is turned slightly downwards and inwards. The false image is above the true and tilted to the patient's right when the left eye is affected (Fig. 102, 4). Double images appear when the eyes are directed upwards and disappear at the horizontal line, while the images become more and more separated, both vertically and laterally, and the tilting of the false image becomes more pronounced according as the object is carried upwards and outwards. In the compensatory attitude the head is thrown backwards and the chin turned a little towards the healthy side.

(3) *Paralysis of the trochlear nerve* gives rise to a moderate squint in which the eye is slightly rotated upwards and inwards. Double images appear when the eyes are directed downwards, and become more and more separated both vertically and laterally according as the object is carried downwards and outwards. The images are vertically superimposed, the false being the lower and the more remote of the two, and

tilted to the patient's right when the left eye is affected (Fig. 102, 6). The secondary deviation is usually straight downwards. There is false projection of the field of vision downwards and a little outwards, and the head is inclined forwards and turned towards the healthy side. The feeling of giddiness is often well marked.

(4) *Paralysis of the abducens nerve* gives rise to *strabismus convergens*, and the eye cannot be rotated outwards beyond the middle line. Double images are seen when the eyes are turned horizontally to the paralyzed side, the distance between them increases according as the object is moved to that side; the images are vertical and parallel, and the false one is to the left of the patient when the left eye is affected (Fig. 102, 2). Secondary deviation occurs to the inner side, there is false projection of the field of vision towards the outer side, and the head is turned towards the affected side. The feeling of giddiness is severe, and may be accompanied by nausea and vomiting.

(5) *Paralysis of the ciliary muscle* (*cycloplegia*) renders the patient, if not myopic, unable to focus small objects or read small print, and he forms an incorrect estimate of the size and distance of small objects. It is often complicated by mydriasis, although it may occur as an independent affection.

2. Masticatory Paralysis.

Etiology.—Paralysis of the trigeminus is rare as a result of lesions of the nerve in its extracranial course, but generally results from intracranial lesions such as periostitis, exostoses, caries, extravasations, and tumors, which compress the nerve at the base of the skull. It is also caused by lesions of the pons and occasionally by lesions of the cortex of the brain.

Symptoms.—When the paralysis is unilateral there is difficulty or impossibility of masticating food on that side, and the lateral movements towards the sound side are rendered impossible by paralysis of the pterygoids. The affected muscles are often felt to be wasted, and they remain flaccid during mastication, while those of the opposite side feel rigid at each contraction. The paralyzed muscles may occasionally manifest the "reaction of degeneration." The tensor veli palati is supplied by the fifth nerve, but paralysis of this muscle has not been observed along with masticatory paralysis. The tensor tympani is also supplied by the fifth nerve, and it is probable that disorders of hearing may accompany masticatory paralysis in the absence of any lesion of the auditory nerve, consisting of noises in the ears, and a diminished power of appreciating deep tones. The paralyzed muscles may become atrophied, and they then, as a rule, manifest the reaction of degeneration.

When the affection is bilateral the patient suffers great fatigue during mastication, and he is forced to eat only fluid and pulpy nourishment. When the paralysis is complete the lower jaw falls down with its own weight; at other times the jaw is fixed by secondary contraction of the paralyzed muscles. Bilateral masticatory paralysis is generally met with as a part of labio-glosso-laryngeal paralysis.

Sensory disorders usually accompany unilateral masticatory paralysis, and the sensory branches of the nerve are often implicated in the absence of any paralysis of the motor branch. When all the branches of the nerve are affected, one side of the face, part of the ear, the skin of the temple and forepart of the head, conjunctiva, cornea, nasal and oral mucous membranes, tongue, gums, and part of the pharynx are all rendered more or less completely insensitive on the affected side, but violent eccentric pains may be felt in the anæsthetic area (anæsthesia dolorosa). When the patient puts a cup to his lips it gives him the impression of being broken, as he can only feel with one-half of the lip.

On the affected side the skin of the face is cold and often of a bluish color; the gums are spongy; the mucous membrane of the mouth and nostril may ulcerate and bleed; and neuroparalytic ophthalmia is apt to occur. Irritation of the nasal mucous membrane by ammonia or snuff, on the affected side does not excite sneezing, and the sense of smell on that side is diminished owing partly to dryness of the Schneiderian membrane and partly to the nutritive changes which take place in it. The sense of taste in the anterior two-thirds of the anæsthetic side of the tongue is lost in those cases only in which the lesion is situated at the base of the skull, or in the lingual branch of the nerve. When the anæsthesia is of peripheral origin, reflex actions are abolished. The extent of the anæsthesia will of course vary according to the seat of the disease, and it may sometimes be limited to the area of distribution of a single branch of the nerve.

Diagnosis.—The chief difficulty in diagnosis is to discover the locality of the primary lesion, and for the determination of this point the following rules modified from Romberg may be of use:

(*a*) The more the anæsthesia is confined to slight filaments of the nerve the more peripheral will the seat of the lesion be.

(*b*) If the loss of sensation affect a portion of the face together with the corresponding facial cavity, one of the divisions of the nerve is affected before or immediately after its passage through the cranium.

(*c*) When the entire area of the fifth is more or less anæsthetic, and there are nutritive disorders in the affected parts, the Gasserian ganglion or the nerve in its immediate vicinity is the seat of the disease.

(*d*) If the anæsthesia of the fifth nerve is complicated with disordered function of adjoining nerves, it may be assumed that the disease is seated at the base of the brain.

(*e*) If sensation is lost in the face on one side, and in half of the body and limbs on the other side, the lesion is probably situated in the lateral part of the upper end of the medulla.

(*f*) If sensation is lost in the face, half of the body, and the limbs on the same side, the lesion is situated in the opposite hemisphere of the brain, most probably in or near the posterior third of the posterior segment of the internal capsule.

3. Paralysis in the Area of Distribution of the Seventh Nerve (Mimetic Paralysis, Hemiplegia and Diplegia Facialis, Prosopalgia, Bell's Paralysis).

Paralysis of the facial nerve, which from the long course of the nerve and its exposed position is frequently observed, may be (*a*) unilateral or (*b*) bilateral. The branches and connections of the nerve are shown in Fig. 103.

a. Unilateral Facial Paralysis (Hemiplegia Facialis).

Etiology.—Exposure of one side of the face to cold is one of the most frequent causes of facial paralysis, it is then called rheumatic paralysis although most probably resulting from a slight neuritis. Facial paralysis, although appearing at all ages, is most frequent between twenty and forty years of age, and sex does not seem to exert any influence on its production, and each side of the face is about equally liable to be affected. It frequently results from injury such as a severe blow on the ear, gunshot and other wounds, and fractures of the temporal bone, while in association with paralysis of the auditory nerve it is one of the most common signs of fracture of the base of the skull. It also occurs after extirpation of the parotid gland and other surgical operations about the face and ear, and may be caused in newborn infants by pressure of the forceps. Disease of the parotid gland or of neighboring parts may cause facial paralysis either by pressure on the nerve or by extension of the morbid process to it. Various diseases of the ear may cause facial paralysis, and of all the causes suppurative *otitis interna*, followed by destructive changes in the temporal bone, is probably the most frequent. Bony tumors, and neoplastic formations of all kinds in the internal ear may lead to compression and destruction of the nerve. Primary disease of the facial nerve is occasionally observed as a complication or sequel of acute febrile diseases such as diphtheria or variola,

but is much more frequent as a secondary result of these affections from disease of the temporal bone. Syphilitic periostitis, meningitis, and exostoses, or gummata at the base of the skull, brain, temporal bone, or in the sheath of the nerve itself, may produce paralysis by compressing the nerve. Some persons appear to be predisposed to facial paralysis. Eulenburg mentions the case of a young man who suffered twice from right-sided, and thrice from left-sided facial paralysis. Facial paralysis may occur as a rare symptom of some diseases of the spinal cord when the morbid process extends upwards to the medulla, and it may occur occasionally in tabes dorsalis, and as a rare complication in tetanus.

Symptoms.—Premonitory symptoms consisting of pain, noises in the ear, deafness, and abnormal sensations of taste on the side which is subsequently attacked with paralysis may be experienced for some days before the loss of motor power declares itself. The symptoms of complete unilateral facial paralysis are very characteristic. The affected side loses its wrinkles and furrows and appears smooth, flaccid, and expressionless; while it appears puffy and falls to a lower level than the healthy side. The patient cannot wrinkle his forehead or elevate his eyebrow on the paralyzed side; he is also unable to close his eye and on attempting to do so the eyeball rolls upwards and inwards or occasionally upwards and outwards. The power of winking is lost and the eye remains open during sleep (lagophthalmos), and being no longer protected from the contact of foreign particles it often becomes irritated and inflamed. Owing to paralysis of Horner's muscle, the tears cannot enter the lachrymal canal, and, therefore, flow over the cheek. The nostril on the paralyzed side falls in during inspiration instead of expanding as it does in health, the tip of the nose is sometimes drawn to the healthy side, and the naso-labial fold is obliterated. The mouth is drawn obliquely over to the healthy side, and the distortion becomes more pronounced during all mimetic movements, such as crying, laughing, and speaking. Paralysis of the buccinator causes the cheek to puff out in speaking, and during other expiratory actions; the pronunciation of the labial consonants is impaired; attempts at blowing or whistling fail because the air escapes through the paralyzed fissure of the lips; the saliva dribbles from the affected side; and food is apt to accumulate between the inner surface of the cheek and the teeth. The external muscles of the ear are also paralyzed, but since these muscles are not usually under voluntary control, impairment of movement in them is not readily detected. Signs of paralysis of the platysma, the posterior belly of the digastric, and the stylo-hyoid muscles can sometimes be discovered. If the lesion of the nerve is situated above the geniculate

ganglion, the levator palati and azygos uvulæ become paralyzed, but the symptoms of this condition will be fully described when the various forms of paralysis of the soft palate are under consideration. If the nerve is diseased above the point where the branch to the stapedius is

FIG. 103.

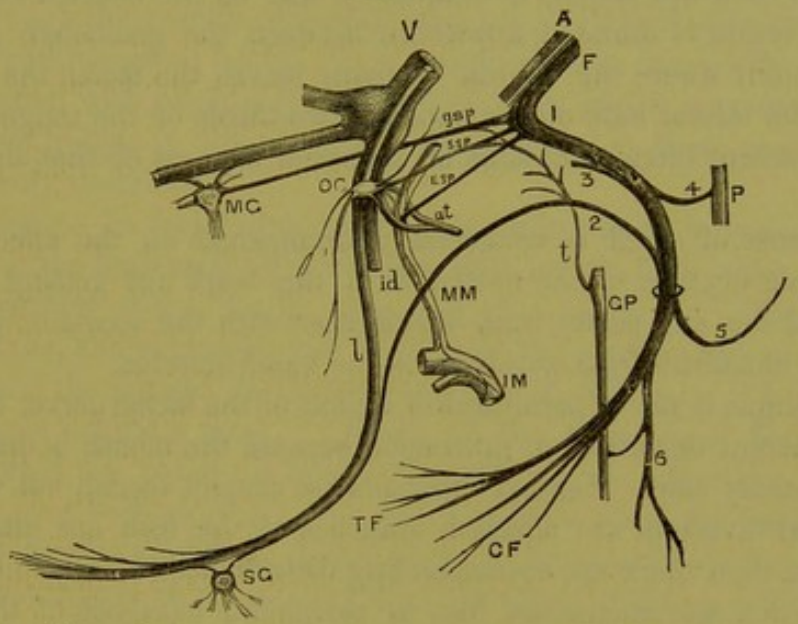


DIAGRAM OF THE FACIAL NERVE, ITS CONNECTIONS AND BRANCHES. (From HERMANN'S "Physiology.")

F, The facial nerve. A, Auditory nerve.

1, The geniculate ganglion.

gsp, Great superficial petrosal nerve connecting the *facial* and Meckel's ganglion.

ssp, Small superficial petrosal nerve connecting the facial with the *Otic* ganglion and with the tympanic branch of the glosso-pharyngeal.

esp, External superficial petrosal connecting the facial with the plexus on the middle meningeal artery.

2, Chorda tympani, joining lingual nerve.

3, Nerve to stapedius muscle.

4, Communicating branch with the *ganglion* of the *root* of the vagus.

5, Posterior auricular nerve.

6, Branch to the stylo-hyoid and digastric muscles.

TF, Temporo-facial division } to muscles of expression.
CF, Cervico-facial division }

V, Fifth nerve. *at*, Auriculo-temporal branch.

id, Inferior dental nerve. *l*, Lingual nerve.

MG, Meckel's ganglion.

OG, Otic ganglion.

SG, Submaxillary ganglion.

IM, Internal maxillary artery.

MM, Middle meningeal artery.

P, Pneumogastric nerve.

GP, Glosso-pharyngeal nerve.

t, Its tympanic branch (nerve of Jacobson).

given off (Fig. 103) that muscle becomes paralyzed, and the membrana tympani is rendered unduly tense by the unantagonized action of the tensor tympani, a condition which causes an abnormal acuteness for

hearing musical tones, especially deep notes, and which is often accompanied by a subjective sound of high pitch.

The auditory and facial nerves are simultaneously affected when the lesion is situated at the base of the brain, in the meatus auditorius internus, or in the middle ear and the adjoining parts of the temporal bone, and then the patient is completely deaf on the affected side.

If the lesion is situated anywhere between the geniculate ganglion and the point where the chorda tympani leaves the facial, the sense of taste in the lateral half of the anterior two-thirds of the tongue is lost, and the patient often complains of abnormal dryness of that side of the mouth.

The sense of smell is occasionally diminished on the affected side partly from dryness of the nostril from the tears not getting into the lachrymal sac, and partly from interference with the access of air to the olfactory chambers from paralysis of the nasal muscles.

The tongue is never paralyzed in lesions of the facial nerve, but there is an apparent deviation or protrusion because the mouth is drawn over to the healthy side. Sensory disorders are usually absent, but when the peripheral divisions are affected, branches of the fifth are often implicated and then there are corresponding disturbances of sensibility.

The reflex movements are lost in peripheral paralysis of the facial. There are no manifest secretory disorders of the face, but it has been found that subcutaneous injection of pilocarpine does not cause sweating of the paralyzed side in cases of peripheral lesions of the nerve. The electrical reactions of the paralyzed nerve and muscles remain normal in slight and transitory cases, but in all aggravated forms they manifest the "reaction of degeneration."

Spontaneous movements may occur in the muscles during recovery, and at times the twitching may be so marked that the disease looks like facial tic. At other times associated movements occur in the paralyzed muscles. When, for instance, an attempt is made to close the eye, the angle of the mouth is drawn outwards and upwards, and conversely, when an attempt is made to draw the angle of the mouth to one side, the eyelids contract; the zygomatici contract when an attempt is made to elevate the eyebrow. The paralyzed eyelid moves or falls to a lower level than the other during downward rotation of the eyeballs. Reflex contraction may occur in the paralyzed muscles, either through the fifth nerve by touching the skin or eyelashes, or through the optic nerves by making a rapid movement towards the eyes.

b. Bilateral Facial Paralysis (Diplegia Facialis).

Etiology.—Bilateral facial paralysis often results from simultaneous lesion of both facial nerves, such as compression of them by a tumor where they lie close together at the base of the skull or in the course of the fibres through the medulla oblongata and pons, bilateral otitis interna, or caries of both temporal bones. It is, however, more frequently observed in connection with progressive bulbar paralysis, and it may occasionally result from lesions in the cerebral hemispheres, but these cases will be subsequently described.

Symptoms.—In facial diplegia the immobility which is present on the one side in the unilateral affection now appears on both sides, but the oblique position of the mouth, nose, and chin is absent. The face is smooth, fixed, and expressionless, even when the emotions are powerfully excited, and the patient, in the apt language of Romberg, "laughs and cries as from behind a mask." When all the branches of both nerves are implicated the patient is unable to wink or close either eye, and the tears flow over both cheeks. The saliva dribbles when the head is bent forwards; taste is abolished in the anterior two-thirds of the tongue; and the mouth may be abnormally dry; paralysis of the stapedii muscles may occasion disorders of hearing, and paralysis of the soft palate gives to the voice a nasal quality and allows fluid to escape through the nose during attempts at deglutition, which is rendered still more difficult by paralysis of the stylo-hyoid and digastric muscles. The nostrils fall in during inspiration, a condition which gives rise to considerable discomfort and difficulty in breathing, and articulation is impaired, as is manifested by the patient being unable to pronounce the vowels *o* and *u*, and the labial consonants.

Diagnosis.—The discrimination of facial paralysis from other diseases presents no difficulty except in the slighter forms of the affection, and the want of symmetry between the two sides may be detected by paying careful attention to the play of the features under varying emotions, or by getting the patient to execute complicated movements, such as whistling, showing the teeth, and pronouncing difficult words. The principal signs by which the disease is recognized in infants are distortion of the face or crying, difficulty of sucking, and lagophthalmos during sleep. The most difficult part of the diagnosis is to determine the exact position of the lesion. We must, in the first place, decide whether the lesion is situated in the fibres which connect the facial nuclei in the pons with the muscles—peripheral paralysis, or in

the fibres which connect the facial cortical centres with the nuclei in the pons—central or cerebral facial paralysis.

Peripheral facial paralysis is characterized by the presence of paralysis of both the upper and lower branches of the nerve, lagophthalmos during sleep, atrophy with the reaction of degeneration of the paralyzed muscles, external wounds or disease in the vicinity of the nerve, and disease of other nerves which lie near to it at the base of the brain or in its course, while there is an absence of hemiplegia and other cerebral symptoms.

Cerebral facial paralysis is characterized by the presence of paralysis of the lower branches of the nerve combined with hemiplegia and other cerebral symptoms, but the power of closing the eye and reflex actions are retained, and the muscles neither undergo atrophy nor manifest the reaction of degeneration. The following rules will enable the reader to determine the exact position of the lesion in cases of peripheral paralysis of the nerve:

(1) If the lesion be situated external to the Fallopian canal, the muscles of the face are alone paralyzed.

(2) If the lesion be situated in the Fallopian canal, but below the point at which the chorda tympani leaves the facial, the muscles of the external ear are paralyzed in addition to those of the face.

(3) If the lesion be situated between the point at which the chorda tympani is given off and the point of origin of the small branch to the stapedius, there are, in addition to the symptoms already mentioned, abolition of taste on the lateral half of the anterior two-thirds of the tongue, and diminution of the salivary secretion on the affected side.

(4) If the lesion be situated between the point of origin of the nerve to the stapedius and the geniculate ganglion, the same symptoms are present, along with abnormal acuteness of hearing.

(5) If the geniculate ganglion itself is diseased all the previous signs are present, and in addition paralysis of the soft palate and distortion of the uvula.

(6) If the lesion be situated in the nerve above the geniculate ganglion, all the previous signs are present except the disorder of the sense of taste, but the auditory nerve is frequently implicated, and then there is dulness of hearing on the affected side.

(7) If the lesion be situated in the pons on a level with the facial nerve, paralysis of the facial muscles is accompanied by one or more of the following symptoms, namely: Hemiplegia of the opposite side, paralysis of the sixth, auditory, and branches of the fifth nerves on

the same side, and a staggering gait with tendency to fall towards the side affected with facial paralysis.

The diagnosis of the seat of the lesion in cerebral facial paralysis will be subsequently considered.

4. Paralysis of the Muscles Supplied by the Hypoglossal Nerve (Glossoplegia).

Etiology.—Paralysis of half of the tongue occurs in hemiplegia, and of both sides in the various forms of bulbar paralysis. Injury of the upper part of the vertebral column, such as fracture of the atlas, may implicate the hypoglossal nerve, and unilateral paralysis with atrophy of the tongue may occur in the advanced stages of tabes dorsalis and in secondary descending sclerosis when the ganglion cells of the hypoglossal nuclei are implicated. The nerve may be injured in its peripheral course by tumors and other lesions at the base of the brain, or by extracranial growths and wounds.

Symptoms.—In unilateral paralysis very little is observed when the tongue is in a state of repose, but when it is protruded the tip is seen to deviate to the paralyzed side in consequence of the predominance of the action of the healthy genioglossus, which directs the tip of the tongue to the opposite side. The various movements of the tongue can only be imperfectly or not at all performed on the affected side.

When the paralysis is bilateral and complete, the tongue lies immovable on the floor of the cavity of the mouth; it is relaxed, often atrophied, with its surface wrinkled, and frequently presenting slight fibrillary contractions on the dorsum. When the patient lies on his back the paralyzed tongue falls backwards in the cavity of the mouth, and by partially closing the glottis it contributes to the production of stertorous breathing in cases of apoplexy, and may even cause asphyxia. If the paralysis is incomplete, the tongue can be protruded, but the patient is unable to execute complicated movements, such as raising the tip towards the roof of the mouth, directing the tip towards the nose after protrusion, or rolling it into a tubular form. In bilateral paralysis mastication becomes seriously interfered with, because the food can no longer be rolled about in the mouth and placed between the teeth, whilst deglutition is impeded because the bolus cannot be properly collected on the dorsum of the tongue and pushed backwards into the pharynx, and consequently food and fluid regurgitate into the mouth, and the patient is annoyed by the constant accumulation of saliva. Articulation becomes indistinct, difficulty being first experienced in

singing and in pronouncing the letters *s*, *sh*, *l*, *e*, *i*, and, at a later period, *k*, *g*, *r*, etc., while if the paralysis be bilateral, complete, and associated with atrophy, vocal speech becomes quite inarticulate and unintelligible, this condition being named *anartheca*.

FIG. 104.

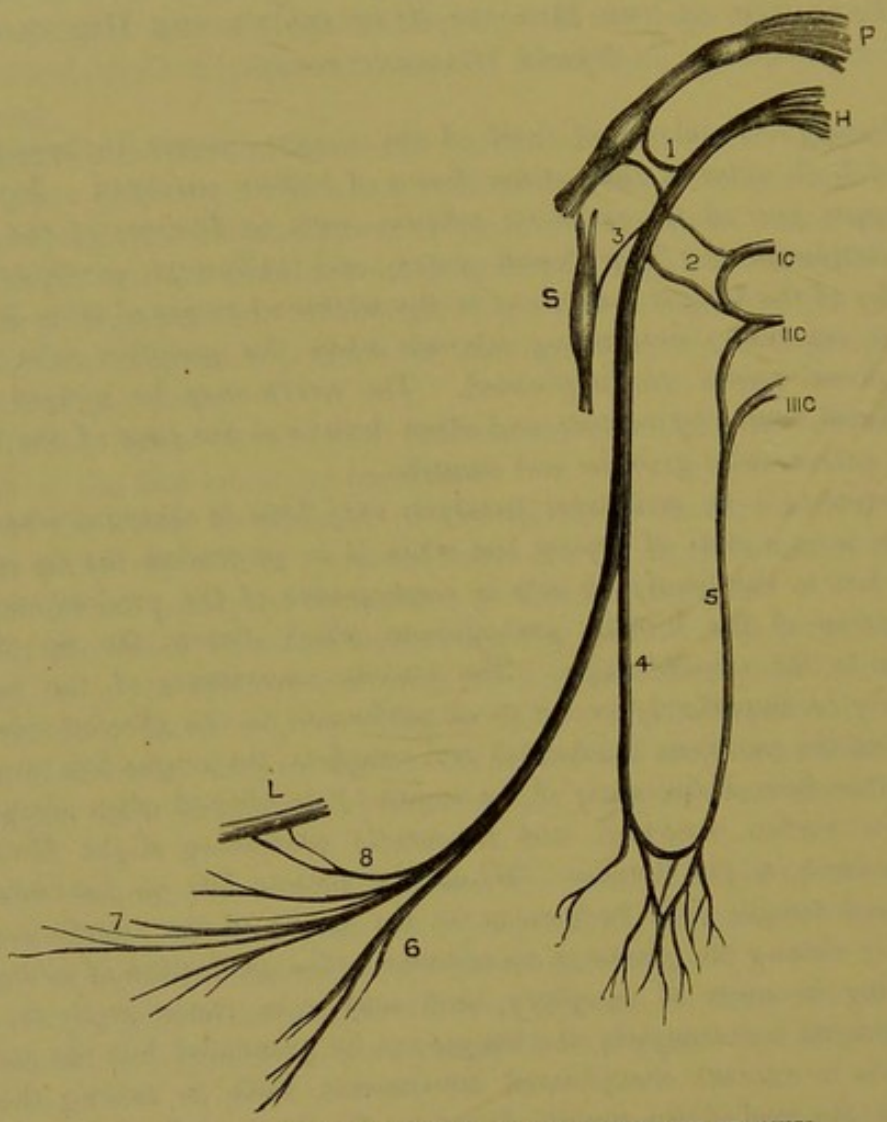


DIAGRAM OF THE HYPOGLOSSAL NERVE, ITS CONNECTIONS AND BRANCHES.

H, Hypoglossal nerve. P, Pneumogastric nerve.
S, Superior cervical ganglion of the sympathetic. L, Lingual nerve.
IC, IIC, IIIC, The three upper cervical nerves.
1, Communicating branches from hypoglossal to ganglion of the trunk of the vagus.
2, Connecting filaments with the loop of first and second cervical nerves.
3, Branch to the sympathetic.
4, Descendens noni.
5, Branch from second and third cervical nerves (communicantes noni).
6, Branch to thyro-hyoid.
7, Terminal muscular branches.
8, Communicating branch to lingual branch of the fifth.

Diagnosis and Prognosis.—The best method of testing the condition of the lingual muscles, according to Hutchinson, is to take each side of the tongue between the finger and thumb of each hand, and then to request the patient to put his tongue out. If half is paralyzed, that side remains soft and flaccid, while the healthy side becomes firm and stiff. Paralysis of the tongue is, however, readily detected by asking the patient to protrude it or to perform complicated movements with it, such as turning the tip towards the nose after protrusion, and rolling it into a tube.

The situation of the primary lesion must be determined from the concomitant symptoms. Unilateral paralysis of the organ associated with hemiplegia is caused by a cerebral lesion, while bilateral paralysis in association with paralysis of the lips and soft palate is caused by a bulbar lesion. The *prognosis* will depend upon primary lesion.

5. Paralysis of the Pneumogastric Nerve.

The distribution of the pneumogastric nerve is represented in Fig. 105.

a. Paralysis of the Soft Palate.

The soft palate is supplied by branches from the pharyngeal plexus, which is composed not only of branches from the vagus, but also of the seventh, the glosso-pharyngeal, and the sympathetic nerves. The levator palati and the azygos uvulæ are supplied from the seventh nerve, but the part which the branches of the other nerves take in the innervation of the palate has not yet been determined, and it will, therefore, be more convenient to group together all the motor and sensory disorders to which the palate is liable.

Etiology.—Paralysis of the soft palate occurs in connection with bulbar paralysis, and in the course of many diseases of the spinal cord, while partial paralysis of it occurs in certain cases of peripheral lesions of the seventh nerve.

Symptoms.—The symptoms differ according as the paralysis is partial or complete.

(1) *Paralysis of the levator palati* causes the velum on the affected side to hang loosely downwards, and to occupy a lower position during rest than the sound side. Tickling does not cause the uvula to be arched upwards, but it is rendered tense transversely by the action of the tensor palati, while its posterior edge is drawn slightly downwards by the action of the palati pharyngeus.

FIG. 105.

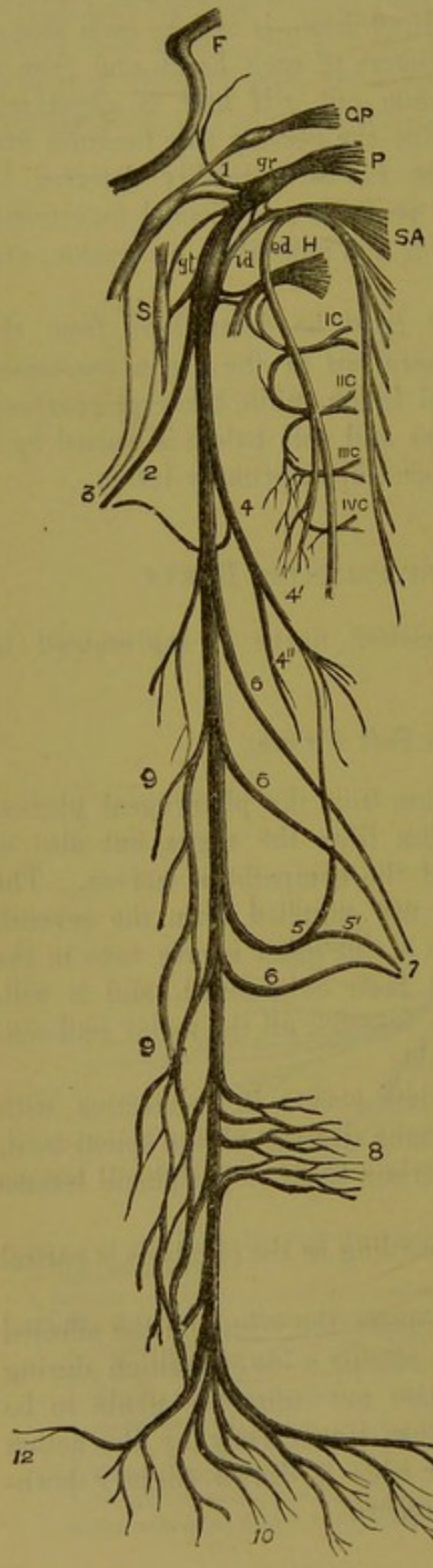


DIAGRAM OF THE PNEUMOGASTRIC AND SPINAL ACCESSORY NERVES, THEIR CONNECTIONS AND BRANCHES. (From HERMANN'S "Physiology.")

P, Pneumogastric nerve.

SA, Spinal accessory nerve.

H, Hypoglossal nerve.

GP, Glosso-pharyngeal nerve.

F, Facial nerve.

S, Superior cervical ganglion of the sympathetic.

gr, *Ganglion* of the *root* of the vagus.

gt, *Ganglion* of the *trunk* of the vagus.

1, Auricular branch of the vagus (Arnold's nerve).

2, Pharyngeal branch.

3, Convergence of nerves to form *pharyngeal plexus*.

4, Superior laryngeal nerve.

4', Internal branch of superior laryngeal nerve

4'', External branch of superior laryngeal nerve.

5, Inferior laryngeal nerve (recurrent).

5', Cardiac branch of inferior laryngeal nerve.

6 6 6, Cardiac branches of the vagus.

7, Convergence of branches of vagus to form cardiac plexuses.

8, Pulmonary branches.

9, Œsophageal branches.

10, Gastric branches.

11, Splenic branches.

12, Hepatic branches.

SA, Spinal accessory nerve.

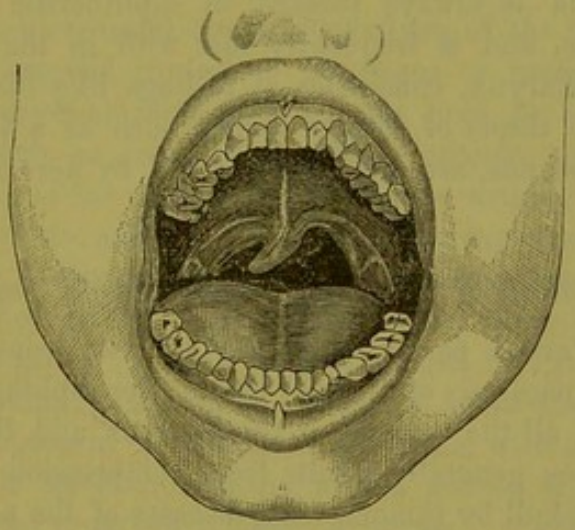
id, Internal division of spinal accessory.

ed, External division of spinal accessory.

IC, IIC, IIIC, IVC, Cervical nerves.

(2) *Combined paralysis of the levator palati and azygos muscle* occurs in lesions of the seventh nerve situated in or above the geniculate ganglion. In addition to the symptoms of paralysis of the levator the uvula is distorted, being arched obliquely forwards and usually towards the healthy side. In many cases, however, the tip of the uvula is directed not to the healthy but to the paralyzed side, the convexity of the arch being then turned to the healthy side (Fig. 106).

FIG. 106.



DISTORTION OF THE UVULA IN A CASE OF PERIPHERAL PARALYSIS OF THE RIGHT SIDE OF THE FACE. (After SANDERS.)

Sanders thought that the last form of distortion was caused by the action of the palato-pharyngeus exerting a greater effect on the uvula on the affected side than it can exert on the sound side, but this explanation is not very satisfactory.

(3) *Combined paralysis of the levator and tensor palati* causes the velum to hang down deeper than when the levator is alone paralyzed, and the velum is likewise displaced laterally owing to the unopposed action of the tensor of the opposite side, while there is no contraction on the affected side on reflex irritation. Speech has a more nasal quality and fluids regurgitate more freely through the nose than when the levator is alone affected.

(4) *Paralysis of the palato-pharyngeus* is recognized by the altered appearance of the isthmus of the fauces, the posterior pillars of these being widely separated from one another, and immovable.

(5) *Bilateral paralysis of all the muscles* of the soft palate causes the palate to hang loose and flapping from the roof of the mouth, and its activity is not called forth during deep inspiration, or during the

movements of deglutition and phonation. The speech has a strongly nasal quality; while fluids are ejected through the nose. Deglutition is rendered still more difficult when paralysis of the soft palate is associated, as frequently happens, with paralysis of constrictors of the pharynx.

The *sensory* disorders which accompany paralysis of the soft palate consist of hyperæsthesia or anæsthesia, and various paræsthesiæ of the mucous membrane of the soft palate and pharynx. Increased sensitiveness of these parts is often met with in nervous and hysterical subjects, while anæsthesia is always present in diphtheritic and progressive bulbar paralysis, and is frequently met with in the insane. Paræsthesiæ of the pharynx, consisting of feelings like that caused by the presence in the throat of foreign bodies, such as a fish-bone, hair, or some hard substance, are often experienced by nervous and hysterical subjects.

b. Paralysis of the Pharynx (Dysphagia Paralytica).

Etiology.—Partial paralysis of the superior constrictors may be associated with paralysis of the soft palate after diphtheria and syphilis, but paralysis of all the muscles of the pharynx and the muscular coat of the œsophagus generally results from compression of the nerves at the base of the skull by tumors, local diseases of the pons and medulla, or as a terminal phenomenon in apoplexy.

Symptoms.—When the muscles of the pharynx are alone paralyzed the only symptom present is difficulty in deglutition: the morsel passes to the back of the mouth, but remains on the root of the tongue in the glosso-epiglottidean fossa, or even over the epiglottis, and must, on account of the dyspnœa to which it gives rise, be removed by means of the finger. Fluids run along the dorsum of the tongue, and, passing readily into the larynx, give rise to attacks of suffocative cough; whilst the patient instinctively makes strenuous efforts to pass the fluid over the epiglottis by throwing the head backwards, and by an endeavor to bring the root of the tongue as near as possible to the upper end of the œsophagus. Paralysis of the superior constrictor allows fluids to regurgitate through the nose during deglutition, inasmuch as contraction of this muscle is necessary to complete the division which the soft palate forms between the buccal and nasal portions of the pharynx during swallowing.

c. Paralysis of the Œsophagus.

In isolated paralysis of the œsophagus the morsel of food passes from the pharynx into the œsophagus, but it remains fast in the cervical por-

tion of the tube or regurgitates into the mouth. When it remains fast in the œsophagus it may produce compression of the larynx and cause dyspnœa and the other symptoms which indicate the presence of a foreign body. In paralysis of the œsophagus a sound can be passed readily into the stomach.

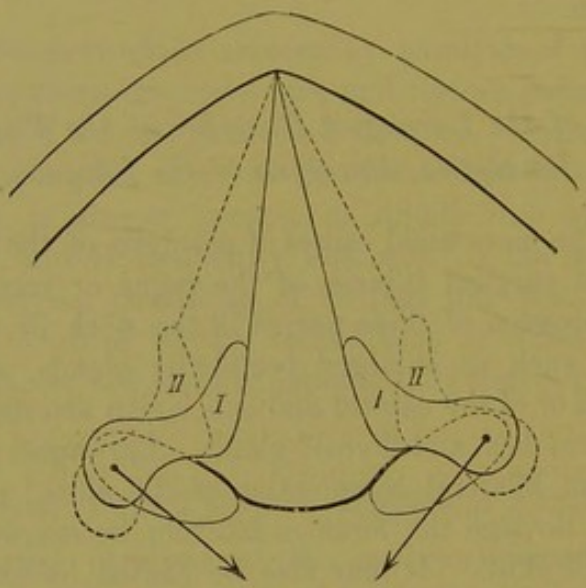
d. Paralysis of the Laryngeal Branches of the Vagus (Aphonia Paralytica, Paralysis of the Larynx).

Etiology.—The more usual causes of paralysis of the larynx are gunshot wounds and surgical injuries of the vagus or recurrent laryngeal nerves, or compression of these nerves in the neck or mediastinum by various tumors, such as enlarged lymphatic glands, aneurism of the arch of the aorta or of the carotid and subclavian arteries, and cancer of the œsophagus, trachea, and thyroid gland. Laryngeal paralysis results occasionally from bilateral compression of the spinal accessory nerves in their passage through the foramen lacerum by cancerous infiltration at the base of the skull. It may also be caused by exposure to cold, and excessive speaking, or it may be a sequel of acute diseases like diphtheria. As a symptom of central disease it is met with most frequently in hysteria, but it sometimes occurs in the course of tabes dorsalis, insular sclerosis, and progressive bulbar paralysis. Unilateral paralysis of the vocal cords may result from an apoplectic seizure when the lesion is situated in the medulla oblongata and more rarely from injury of the cerebral conducting path above the medulla.

Symptoms.—If paralysis of the laryngeal muscles gives rise to vocal and respiratory disorders it is named (1) *mixed laryngeal* paralysis, but if disorders of respiration are alone present it is named (2) *respiratory* paralysis, while if disorders of vocalization are alone present it is named (3) *phonetic* paralysis. Paralysis of the laryngeal muscles may also be divided into unilateral and bilateral, complete and incomplete paralysis, according to its extent, and each of these may be divided into total and partial paralysis, according to its degree. Before proceeding to describe the symptoms it may be as well to mention the various positions assumed by the glottis under different circumstances. Passing from the middle line outwards, these positions are: (1) *Complete closure* of the glottis (Fig. 108, *II, II*), produced by the combined action of the arytænoidei and crico-arytænoidei muscles; (2) the *cadaveric* position, in which the glottis is slightly open, as it is found in the dead body; (3) the *position of quiet breathing*, in which the glottis

is more open than in the last position, but still only moderately dilated (Fig. 107, *I*, *I*); (4) the *position of deep inspiration*, in which the

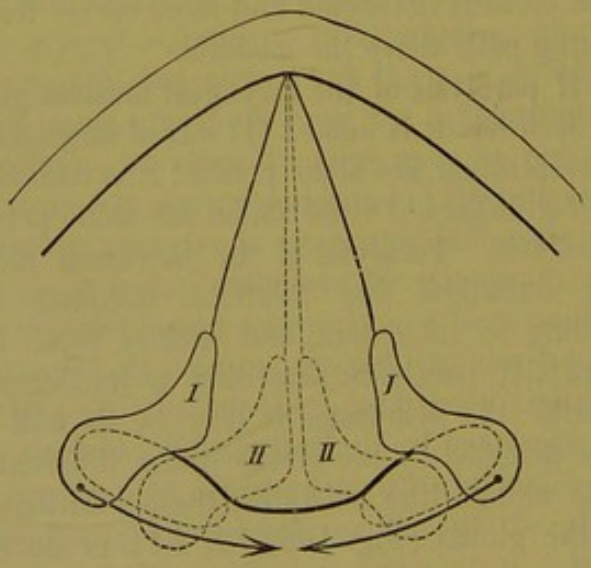
Fig. 107.



Schema of a Horizontal Section through the Larynx.

I I, The position of the arytænoid cartilages and vocal cords during quiet breathing. The arrows indicate the direction in which the crico-arytænoidei postici muscles act. *II II*, Position of the arytænoid cartilages in consequence of contraction of these muscles.

Fig. 108.

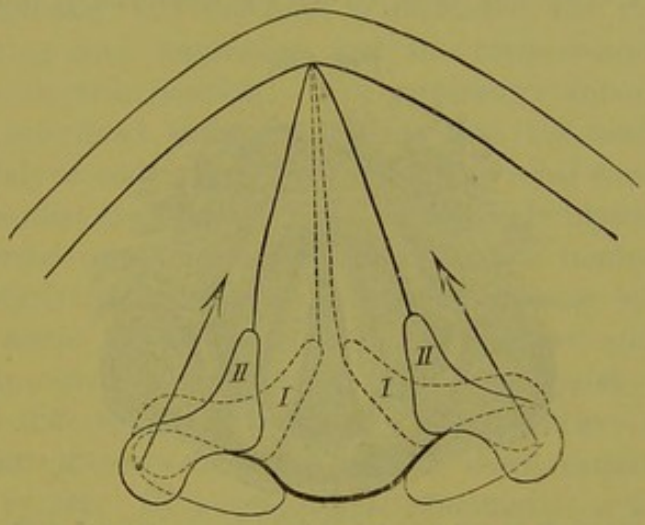


Schema of a Horizontal Section through the Larynx, showing the action of the Arytænoid Muscles. (From Landois's "Physiologie.")

I I, Position of the arytænoid cartilages during quiet breathing. The arrows show the direction of the action of the muscles. *II II*, Position of the cartilages when the muscles are contracted.

glottis is widely dilated (Fig. 107, *II, II*); (5) the position in which the glottis vocalis is closed and the glottis respiratoria open (Fig. 109, *I, I*).

Fig. 109.



Schema of Horizontal Section through the Larynx, illustrating the action of Crico-arytænoidei Laterales Muscles. (From Landois's "Physiologie.")

II II, Position of the arytænoid cartilages during quiet breathing. The arrows show the direction in which the muscles act. *I I*, Position of the cartilages when the muscles are contracted.

(1) Mixed Laryngeal Paralysis.

Mixed laryngeal paralyses are caused by disease of the recurrent laryngeal nerves, and the muscles which dilate the glottis as well as those which render tense and approximate the vocal cords, are paralyzed, giving rise to disorders of vocalization and respiration. The muscles become atrophied as in other forms of peripheral paralysis, and lose their faradic contractility.

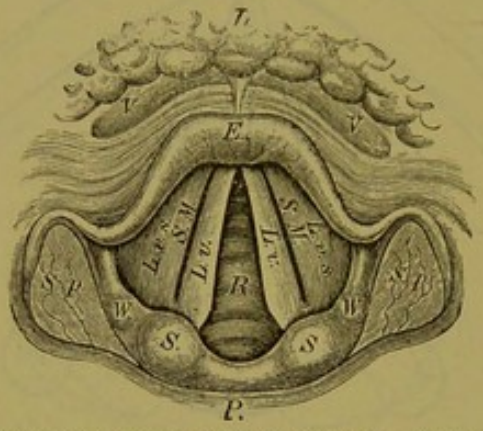
(*a*) In *complete bilateral paralysis* of the laryngeal muscles the glottis assumes the cadaveric position, and is immovable during attempts at phonation. The relaxed cords are drawn somewhat downwards during inspiration, and pushed slightly upwards, and probably slightly removed from one another during expiration, but these slight movements are very different from the active movements caused by contracting muscles. The voice is weak and may be reduced to an almost inaudible whisper. Respiration is not much interfered with when the patient is at rest, but dyspnœa and stridulous inspiration are readily induced on slight exertion (Figs. 110–113).

(*b*) In *complete unilateral paralysis* of the laryngeal muscles the vocal cord of the corresponding side is motionless, its free edge is slightly removed from the middle line, and the vocal cord of the sound

side alone vibrates during attempts at phonation. The voice is completely lost, but more frequently it is harsh and discordant, and is liable to break into falsetto tones on the slightest strain.

(*c*) In *incomplete paralysis*, whether unilateral or bilateral, the power of excursion of the vocal cord is merely diminished and not entirely lost.

Fig. 110.



Laryngoscopic appearances of the interior of the larynx. (From Landois's "Physiologie.")

L., The root of the tongue; *V. V.*, Glosso-epiglottidean ligament; *E.*, The epiglottis; *R.*, Glottis; *L. v.*, The true vocal cord; *S. M.*, Opening into the sinus of Morgagni; *L. v. s.*, The false vocal cords; *S. S.*, The projection of the cartilages of Santorini; *P.*, Pharynx wall; *W. W.*, The cartilages of Wrisberg in the ary-epiglottidean ligament; *S. p.*, The sinus pyriformis.

Fig. 111. Fig. 112. Fig. 113.

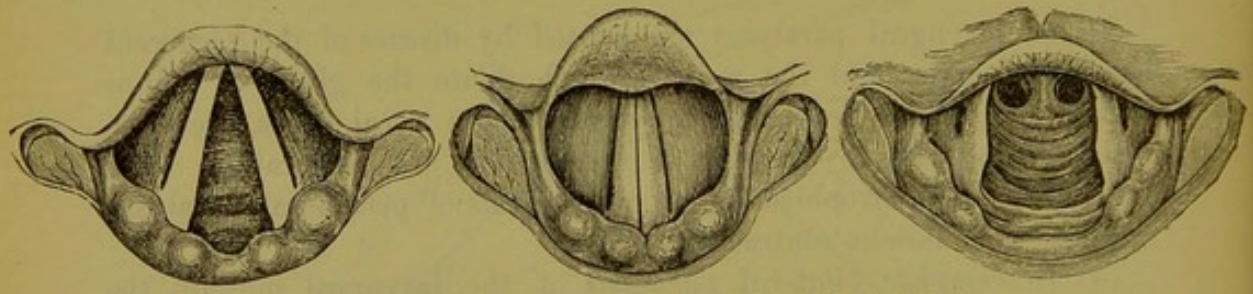


Fig. 111.—Laryngoscopic appearance of the larynx during quiet breathing.

Fig 112.—The laryngoscopic appearance of the larynx during vocalization.

Fig. 113 —The laryngoscopic appearance of the larynx during deep inspiration, showing the bifurcation of the trachea.

(2) Respiratory Laryngeal Paralysis.

Respiratory laryngeal paralysis occurs when the muscles which widen the aperture of the glottis—the crico-arytænoidei postici—are paralyzed. In such cases the voice is unaffected, but serious disorders of respiration may be present. Bilateral compression of the recurrent laryngeal nerves by aneurism or other tumor causes paralysis of the abductors of the vocal cords, while frequently leaving the adductors unaffected.

(*a*) In *complete paralysis* of the crico-arytænoidei postici the vocal cords assume the cadaveric position, just as in complete paralysis of all the laryngeal muscles. But in isolated paralysis of the abductors of the larynx the approximation and parallelism of the vocal cords can still be accomplished, and the voice is unaffected, but the glottis does not dilate during deep inspiration, and the cords cannot be separated beyond the cadaveric position. The respiratory function is at first only slightly interfered with, but after a time the healthy adductors undergo "paralytic contracture" and drag the vocal cords towards the middle line, so that the glottis is almost completely closed. The glottis is then converted into a narrow slit, and is quite inadequate to carry on ordinary respiration. The breathing now becomes laborious and of the "forced costal type," inspiration is prolonged and noisy, as in croup, but expiration is comparatively easy and quick, and in consequence of the difference of atmospheric pressure above and below the constricted part the larynx moves up and down considerably during each respiratory act. On laryngoscopic examination it is seen that the vocal cords are approximated so that only a narrow linear chink is left between them, and during forced inspiration the cords approach each other so closely as almost to close the glottis completely instead of separating from each other as in health. The cords are slightly separated during each expiratory act. During phonation the vocal cords and arytænoid cartilages approach each other in a perfectly normal manner.

(*b*) *Incomplete respiratory laryngeal* paralysis may exist for a long time without giving rise to difficulty of breathing or any manifest symptoms, but the condition could doubtless be detected in laryngoscopic examination.

(3) Phonetic Laryngeal Paralysis.

Phonetic paralyses occur when the muscles which render tense and approximate the vocal cords are paralyzed, and the affection of voice may vary from slight hoarseness to complete aphonia, according to the degree and extent of paralysis.

(*a*) In *bilateral paralysis* of the adductors the glottis is partially open, and both the arytænoid cartilages and the vocal cords are immovable during attempts at phonation. The glottis cannot be closed in coughing, on making an effort, or during deglutition, while there is complete aphonia.

(*b*) In *unilateral paralysis* of the adductors the vocal cord of the affected side is removed from the middle line, and cannot be approxi-

mated to its fellow. The affected vocal cord can only vibrate with its edge, and consequently the voice is feeble, and readily assumes the falsetto character.

(*c*) In *incomplete phonetic laryngeal paralysis* the tensor and constrictor muscles of the vocal cords are separately paralyzed. Aphonia is a symptom of paralysis of all these muscles, but it is most marked in those cases in which the space between the cartilages remains open, because the blast of air escapes through the patent glottis respiratoria. The voice may not be much affected in those cases in which the cartilages close and the ligamentous portion remains open, because the blast of air must pass through the glottis vocalis, and a certain degree of vibration of the cords induced.

(j) In *paralysis of the crico-thyroid* muscles, which occurs when the superior laryngeal nerve is implicated, either directly or through the spinal accessory nerve, the vocal cords cannot be rendered tense, and consequently the voice becomes hoarse and deep, while the production of high notes is difficult or impossible. The absence of tension of the crico-thyroid muscles during vocalization may sometimes be perceived by placing the finger over the crico-thyroid space. Disease of the superior laryngeal nerve also paralyzes the thyro-arytæno-epiglottidei, and the epiglottis, being drawn towards the tongue, fails to be depressed over the aperture of the glottis during deglutition, and consequently food and drink obtain entrance into the larynx. Anæsthesia of the larynx is also present, and consequently the reflex act of coughing is not set up until the foreign substance has passed below the level of the vocal cords, and thus an attack of pneumonia is likely to be provoked. On laryngoscopic examination the glottis is seen to be represented by a wavy line, and in unilateral paralysis the vocal cord on the affected side remains on a higher level than its fellow. The glottis closes completely during strong expiratory efforts like coughing, and the arytænoid cartilages are quite movable during attempts at phonation.

(jj) In *paralysis of the thyro-arytænoidei* the ligamentous part of the glottis remains open, while juxtaposition of the arytænoid cartilages takes place. Rheumatic laryngeal paralysis often assumes this form, but occasionally all the muscles may be affected.

(jjj) In *paralysis of the crico-arytænodei laterales* the glottis remains open in the form of a tolerably broad ellipse.

(iv) In *paralysis of the arytænoidei* the ligamentous portion closes almost completely, while the space between the arytænoid cartilages assumes a triangular form. This is the kind of paralysis which generally causes hysterical aphonia.

The *sensory disorders* which occur in diseases of the laryngeal nerves consist, in addition to neuralgia, of hyperæsthesia, anæsthesia, and various paræsthesiæ. Hyperæsthesia of the laryngeal mucous membrane gives rise to excessive reflex actions on slight provocation. Anæsthesia of the larynx occurs in diphtheritic paralysis, and may be, in association with anæsthesia of the pharynx, one of the earliest symptoms of bulbar paralysis. The paræsthesiæ consist of abnormal feelings as if a hair, fish-bone, or morsel of food had lodged in the larynx, while a laryngoscopic examination shows the absence of any foreign body.

(*d*) *Paralysis of the trunk of the vagus and of the gastric and cardiac plexuses* has already been considered.

6. Paralysis of the Spinal Accessory Nerve.

Etiology.—Paralysis in the region of distribution of the spinal accessory nerve is generally of peripheral origin, and is caused by traumatic injuries of the nerve or by compression by tumors, abscesses, or diseased bones of the skull. Paralysis may also be caused by exposure to cold or by neuritis in whatever way produced, and it may likewise arise as a part of progressive muscular atrophy. Partial paralysis of the nerve may be caused by disease of the four or five upper cervical vertebræ.

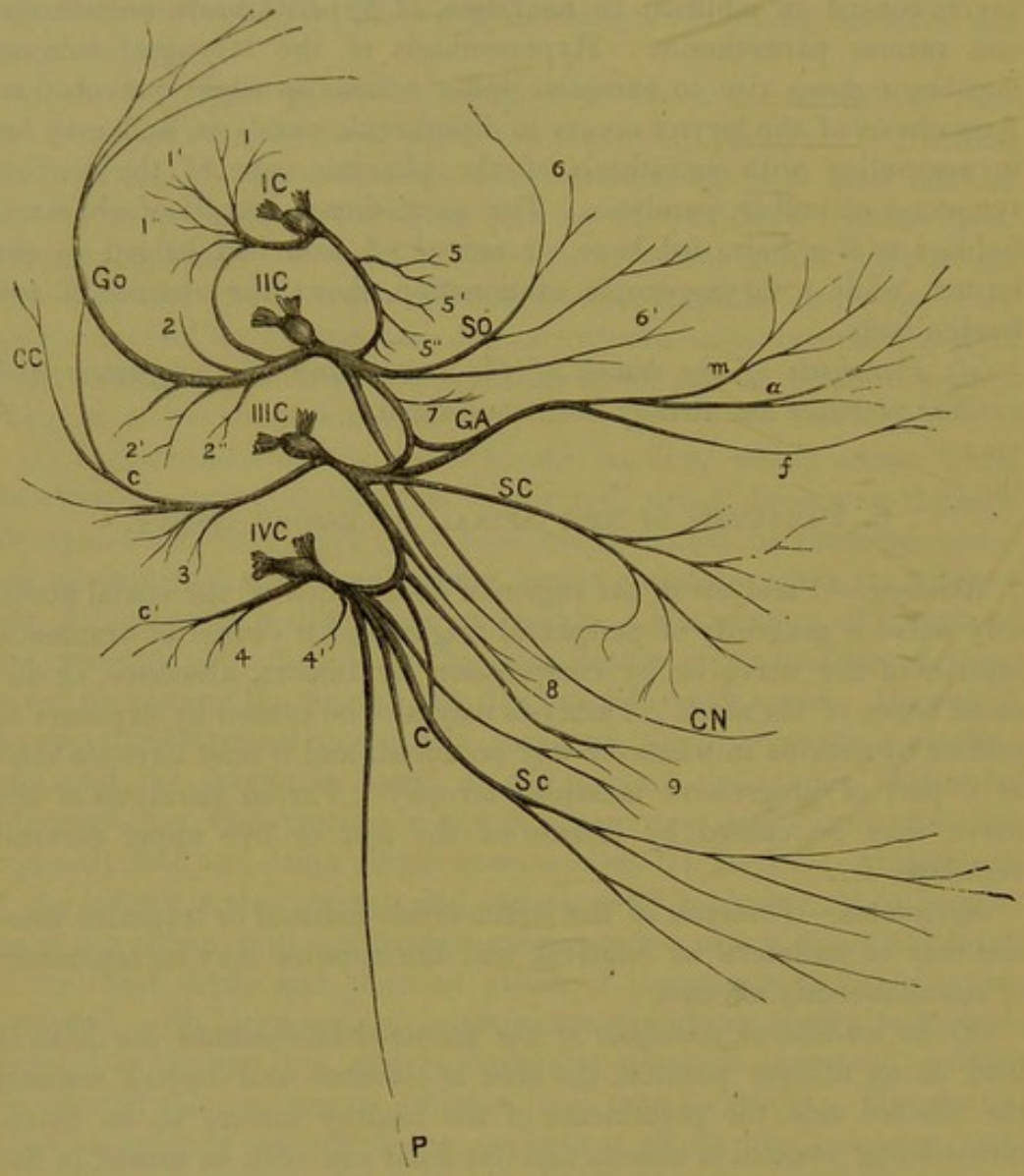
Symptoms.—Paralysis of the sterno-cleido-mastoid or trapezius muscles may be unilateral or bilateral, and the muscles may be separately or simultaneously affected.

(1) In *unilateral paralysis of the sterno-cleido-mastoid* the head is held in an oblique position, the chin is elevated and turned towards the affected side, the prominence of the healthy muscle on its movements being resisted is absent, and the head can only be moved in the direction of the affected muscle with difficulty and by the aid of the other muscles, but passive movements can be readily performed. In chronic cases the healthy muscle becomes contracted, and the head is made to assume a permanently oblique position.

(2) In *bilateral paralysis of the sterno-cleido-mastoid* muscles the head is held straight, but rotation of it can only be performed with difficulty, especially when the chin is elevated. The prominences of the muscles are absent, the neck looks wasted, and a slight depression is produced between the mastoid process and the sternum.

(3) In *unilateral paralysis of the trapezius* the scapula of the affected side is drawn somewhat downwards and forwards, and its inner border

Fig. 114.



Nerves of the Cervical Plexus.

IC, IIC, IIIC, IVC, First, second, third, and fourth cervical nerves.
1, Muscular branch to rectus capitus posticus major and minor.
1′, Muscular branch to obliquus superior.
1″, Muscular branch to complexus.
2, Muscular branch to obliquus inferior.
2′, Muscular branch to complexus and trachelo-mastoid.
2″, Muscular branch to splenius.
3, Muscular branch to muscles of the neck.
4, Muscular branch to muscles of the neck.
G o, Great occipital nerve, cutaneous to posterior part of scalp.
CC, Cutaneous branch to posterior part of scalp and back of neck.
5, Muscular branches to rectus capitis lateralis.
5′, Muscular branches to rectus capitis anticus major and minor.
5″, Communicating branches with hypoglossal and pneumogastric nerves.
SO, Small occipital nerve
6, Muscular branch to occipito-frontalis muscle.

is separated from the vertebral column and assumes an oblique position, the inferior angle being nearer than the superior to the vertebral column. The acromion process falls downwards and forwards, the projecting clavicle makes the supraclavicular fossa deeper than natural, and the posterior and superior angle of the scapula can be felt with unusual distinctness. Paralysis of the upper portion of the trapezius alone renders elevation of the arm above the horizontal line difficult.

(4) In *bilateral paralysis of the trapezius* the symptoms are present on both sides, both shoulder-blades fall outwards and forwards, the head readily sinks on the chest, and some difficulty is experienced in maintaining it in an upright position.

(5) In *combined paralysis of the sterno-cleido-mastoid and trapezius muscles* the symptoms of the separate paralyses are combined, and then the laryngeal muscles are not infrequently paralyzed.

7. Paralysis of the Muscles Supplied by the Cervical Plexus.

(1) *Paralysis of the Posterior Muscles of the Neck.*

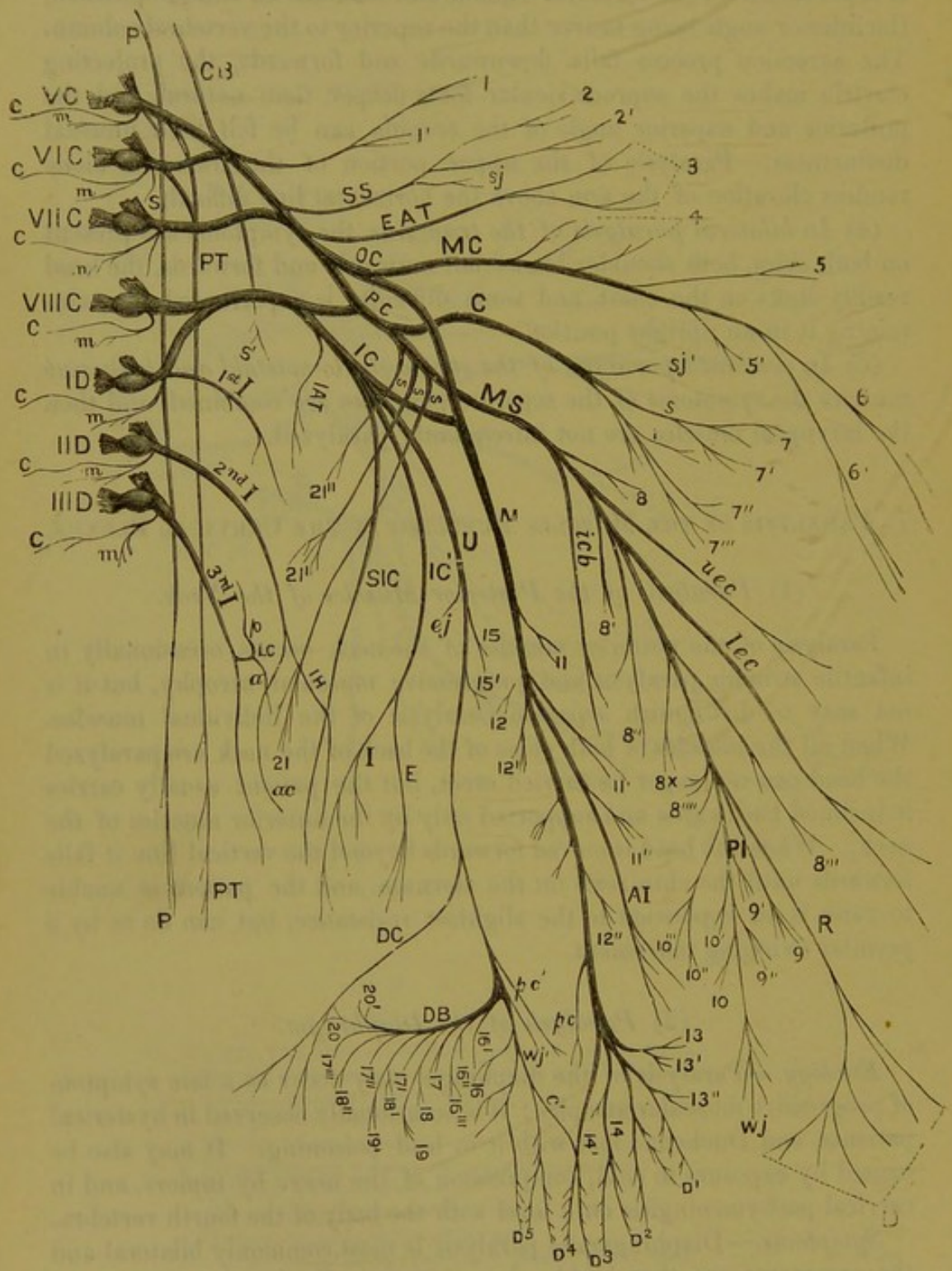
Paralysis of the posterior muscles of the neck occurs occasionally in infantile atrophic paralysis and progressive muscular atrophy, but it is not easy to distinguish separate paralysis of the individual muscles. When all the muscles of both sides of the back of the neck are paralyzed the head can no longer be carried erect, but the patient usually carries it inclined backwards and supported only by the anterior muscles of the neck. When the head is moved forwards beyond the vertical line it falls forwards until the chin rests on the sternum, and the patient is unable to raise it in opposition to the slightest resistance, but can do so by a peculiar swinging movement.

(2) *Paralysis of the Diaphragm.*

Etiology.—Paralysis of the diaphragm may occur as a late symptom of progressive muscular atrophy; it is occasionally observed in hysterical patients, and Duchenne met with it in lead poisoning. It may also be caused by exposure to cold, compression of the nerve by tumors, and in cervical pachymeningitis on a level with the body of the fourth vertebra.

Symptoms.—Diaphragmatic paralysis is most commonly bilateral and the symptoms are then highly characteristic. During inspiration the epigastrium and hypochondria are drawn inwards instead of being curved outwards, and if the hand be placed upon the epigastrium the protrusion caused in health by the descending diaphragm during inspira-

FIG. 115.



NERVES OF THE BRACHIAL PLEXUS. (After FLOWER.)

VC, VIC, VIIC, VIIIC, ID, IID, IIID, Fifth, sixth, seventh, and eighth cervical, and first, second, and third dorsal nerves.

CB, Communicating branch from the fourth cervical nerve.

P, Phrenic nerve.

C, C, Dorsal cutaneous branches from the fifth cervical to the third dorsal nerve.
M, M, Branches to the muscles of the neck and back.
B, Branch to the scalenus medius.
S′, Branch to the subclavius muscle.
I, I′, Branches to rhomboideus major and rhomboideus minor.
SS, Suprascapular nerves:
2, Branch to supraspinatus muscle.
2′, Branch to infraspinatus muscle.
sj, Branch to shoulder-joint.
PT, Posterior or long thoracic (external respiratory of Bell) supplies serratus magnus.
EAT, External anterior thoracic supplies pectoralis major.
IAT, Internal anterior thoracic to pectoralis major and pectoralis minor.
1I, First intercostal nerve.
2I, Second intercostal nerve.
IH, Intercosto-humeral joins nerve of Wrisberg.
AC, Anterior cutaneous nerves of the thorax.
3I, Third intercostal nerve.
LC, Lateral cutaneous:
a, Anterior branch.
p, Posterior branch.
OC, Outer cord of brachial plexus.
PC, Posterior cord of brachial plexus.
IC, Inner cord.
MC, Musculo-cutaneous nerve.
4, Branches to coraco-brachialis.
5, Branches to biceps.
5′, Branches to brachialis anticus.
6, Anterior cutaneous branch } to outer side of forearm.
6′, Posterior cutaneous branch }
IC′, Internal cutaneous nerve.
E, Anterior or external branch.
I, Posterior or internal branch to inner side of forearm.
SIC, Small cutaneous nerve (nerve of Wrisberg) to inner side of arm.
SSS, Subscapular nerves:
21, Long subscapular nerve to latissimus dorsi.
21′, Muscular branches to subscapularis and teres major
21″, Muscular branches to subscapularis.

C, *Circumflex Nerve.*
sj′, Branch to shoulder-joint.
s, Superior division.
7, Cutaneous.
7′, Muscular to deltoid.
i, Inferior division.
7″, Cutaneous.
7‴, Muscular to teres minor.

MS, *Musculo-spiral Nerve.*
8, Muscular to brachialis anticus.
8′, Muscular to triceps.
8″, Muscular to anconeus.
8‴, Muscular to supinator longus.
8⁗, Muscular to supinator brevis.
8×, Muscular to extensor carpi radialis longior.
icb, Internal cutaneous branch to inner side of arm.
uec, Upper external cutaneous branch to outer side of arm.
lec, Lower external cutaneous branch to outer side and back of forearm.
R, Radial nerve cutaneous to dorsal surface of thumb and two outer fingers.
PI, Posterior Interosseous.
9, Muscular branch to extensor carpi radialis brevior.
9′, Muscular branch to extensor ossis metacarpi pollicis.
9″, Muscular branch to extensor primi internodii pollicis.

10, Muscular branch to extensor secundii internodii pollicis.
10′, Muscular branch to extensor indicis.
10″, Muscular branch to extensor carpi ulnaris.
10‴, Muscular branch to extensor { digitorum communis and extensor minimi digiti.
icj, Branch to wrist-joint.

M, *Median Nerve*.
11, Muscular branches to pronator radii teres.
11′, Muscular branches to flexor profundus digitorum.
11″, Muscular branches to flexor longus pollicis.
AI, Anterior interosseous branch to pronator quadratus.
12, Muscular branch to flexor carpi radialis.
12′, Muscular branch to flexor sublimis digitorum.
12″, Muscular branch to palmaris longus.
13, Muscular branch to opponens pollicis.
13′, Muscular branch to abductor pollicis.
13″, Muscular branch to flexor brevis pollicis (outer half).
14, Muscular branch to first lumbricalis.
14′, Muscular branch to second lumbricalis.
pc, Palmar cutaneous branch.
D to D4, Digital cutaneous branches.

U, *Ulnar Nerve*.
ej, Branch to elbow-joint.
15, Muscular branch to flexor profundus digitorum (inner part).
15′, Muscular branch to flexor carpi ulnaris.
DC, Dorsal cutaneous branch. To dorsal surface of two inner fingers.
p′ e′, Palmar cutaneous branch.
c′, Communicating to median.
D3 to D4, Cutaneous to little finger and inner side of ring finger.
16, Muscular branches to palmaris brevis.
16′, Muscular branches to abductor minimi digiti.
16″, Muscular branches to opponens minimi digiti.
16‴, Muscular branches to flexor brevis minimi digiti.
17, Muscular branches to fourth dorsal interosseous.
17′, Muscular branches to third dorsal interosseous.
17″, Muscular branches to second dorsal interosseous.
17‴, Muscular branches to first dorsal interosseous.
18, Muscular branches to third palmar interosseous.
18′, Muscular branches to second palmar interosseous.
18″, Muscular branches to first palmar interosseous.
19, Muscular branches to fourth lumbricalis.
19′, Muscular branches to third lumbricalis.
20, Muscular branches to adductor pollicis.
20′, Muscular branches to flexor brevis pollicis (inner half).

tion cannot be perceived, but the epigastrium may be felt to protrude slightly during expiration. When the paralysis is unilateral these signs can only be recognized on one side. The breathing, even in complete paralysis, is not much accelerated during rest, but dyspnœa begins with the slightest exertion, and the respirations frequently rise from forty to fifty in the minute. The contents of the bladder and rectum are expelled with difficulty, because the patient is unable to strain, the action of the abdominal muscles being rendered ineffective by the absence of the counter-pressure afforded by the contracted diaphragm.

8. Paralysis of the Muscles Supplied by the Brachial Plexus.

The paralyses of the brachial plexus may be divided into those, *a*, of the muscles of the neck and trunk, and, *b*, those of the upper extremity.

a. Paralysis of the Muscles of the Neck and Trunk.

(1) *Paralysis of the Pectoralis Major and Pectoralis Minor.*—When these muscles are paralyzed the power of adducting the arm to the thorax or of resisting passive abduction of it is impaired; the patient is unable to seize the opposite shoulder with the hand; and if the muscles are likewise atrophied the anterior wall of the axilla is reduced to a flaccid fold of skin, the superior ribs and intercostal spaces are well marked, and the subclavicular fossa is deepened.

(2) *Paralysis of the rhomboid muscles of the levator anguli scapulæ* renders forced elevation of the scapula without rotation impossible.

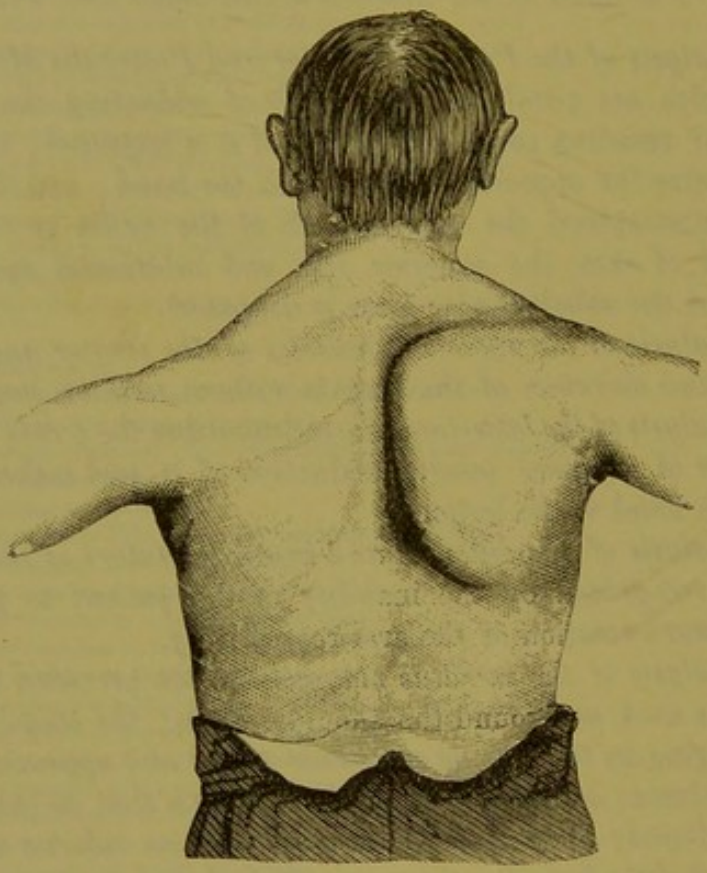
(3) *Paralysis of the latissimus dorsi* diminishes the power of adducting the arm, or of resisting passive abduction of it, and makes it difficult to move the hand to the buttock.

(4) *Paralysis of the outward and inward rotators of the upper arm* is readily recognized by the inability of the patient to produce outward or inward rotation of the arm respectively.

(5) *Paralysis of the serratus magnus* is often preceded by neuralgic pains in the neck and round the shoulder-blade; the scapula, when the arm is hanging by the side, is somewhat raised and approximated to the vertebral column, and it is so rotated on its axis that its inner border is directed obliquely upwards and outwards, and its inferior angle, which stands out slightly from the thoracic wall, is drawn close to the vertebral column (Fig. 116). The patient experiences difficulty in raising the extended arm beyond the horizontal level because the usual forward rotation of the inferior angle of the scapula is not affected, but by instinctively leaning the body to the opposite side he can raise the arm to a point midway between the horizontal and vertical positions, but not further. If the observer rotates the scapula forwards and fixes the bone in that position, the patient is enabled to raise the arm to a vertical position. When the arm is raised to a horizontal position the inner border of the scapula is drawn inwards to the vertebral column while pushing a mass of muscle before it, and if the paralysis be bilateral the inner borders of the scapula may actually touch one another. If the raised arm be brought forwards, the inner border of the scapula becomes more and more separated from the costal wall, so that a deep fossa is

formed in which the hand may easily be laid so as to feel the inner surface of the bone, and if the paralysis be bilateral the scapulæ enclose a deep hollow in which the muscular bellies of the rhomboid muscles may be seen to project. The patient experiences difficulty in crossing the arms in front of the chest, and in moving the apex of the shoulder

Fig. 116.



forwards as in delivering a blow in fencing, and he offers less resistance than in health to forcible retraction of the shoulder. The chest expands less and the digitations of the serratus magnus with the external oblique are not so marked on the paralyzed as on the healthy side during forced inspiration.

b. Paralysis of the Muscles of the Upper Extremity.

(1) *Paralysis of the Circumflex Nerve.*—The circumflex nerve supplies the deltoid and the teres minor muscles, but the symptoms are almost exclusively those of paralysis of the deltoid. The arm cannot be raised outwards or forwards, and during attempts to raise it the deltoid remains quite relaxed, and the arm hangs helpless along the side

of the thorax. The muscle frequently undergoes atrophy, and the shoulder-joint becomes so loose that a deep groove can be felt through the atrophied muscle between the head of the humerus and the glenoid cavity. There may be pain in the shoulder-joint and in the substance of the muscle, but other sensory disturbances in the region of distribution of the nerve are rare. Lesions of the circumflex nerve do not give rise to any very manifest disorders of sensibility, but there may be a slight degree of blunting of sensibility on the outer surface of the shoulder (Figs. 120, 121).

(2) *Paralysis of the Musculo-cutaneous Nerve.*—Paralysis in the region of distribution of this nerve greatly diminishes the power of flexing the forearm upon the arm, especially when the forearm is supinated so that the flexor action of the supinator longus can no longer come into play. Paralysis of the musculo-cutaneous nerve is accompanied by anæsthesia along the radial border of the forearm (Figs. 120, 121).

(3) *Paralysis of the Musculo-spiral Nerve.*—Paralysis of this nerve is frequently caused by injury of the nerve as it winds round the humerus. The most frequent cause of injury is compression of the nerve between the bone and some hard substance during the deep and prolonged sleep which follows intoxication. The nerve may also be subjected to pressure in the axilla by means of improperly constructed crutches. When the nerve is completely paralyzed the hand is maintained in a flexed position, and hangs helpless when the forearm is extended and pronated. The patient is unable to raise the hand or extend the fingers at the metacarpo-phalangeal joints; the thumb is flexed and adducted, and the fingers are flexed over the thumb. When the hand is placed on a table it cannot be raised or moved laterally, but the lateral movements of the fingers remain unimpaired. The patient is unable to supinate the forearm, especially when it is extended so as to exclude the action of the biceps. Paralysis of the supinator longus is readily recognized by requesting the patient to make a powerful effort to flex the arm against a resisting object when the forearm is maintained in a half-flexed position and midway between pronation and supination; it is then found that the muscle does not enter into contraction but remains flaccid and soft. In *lead paralysis* the supinator longus is always spared unless the paralysis implicates the deltoid and biceps; when the lesion is situated in the nerve as it winds round the humerus the triceps muscle is spared, but in crutch paralysis it is paralyzed and the patient cannot extend the forearm upon the arm. The action of the flexor muscles is apparently weakened, but this is due to the fact that the flexed position assumed by the hand approximates the points

of origin and insertion of these muscles. Disease of the musculo-spiral nerve when situated high up gives rise to anæsthesia in the areas of distribution of the superior and inferior external cutaneous branches (Figs. 120, 121, I'CB, ECB); but when the lesion is situated low

Fig. 117.

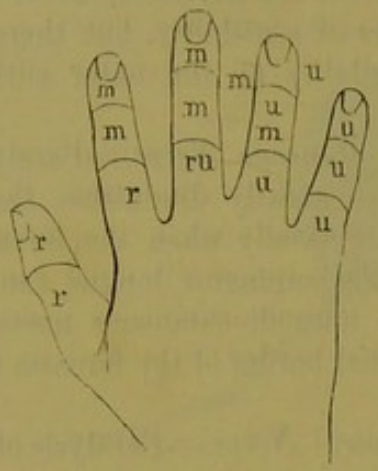


Fig. 118.

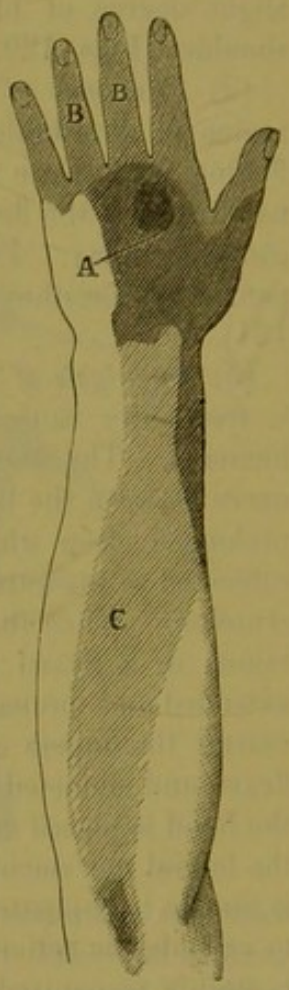


Fig. 119.

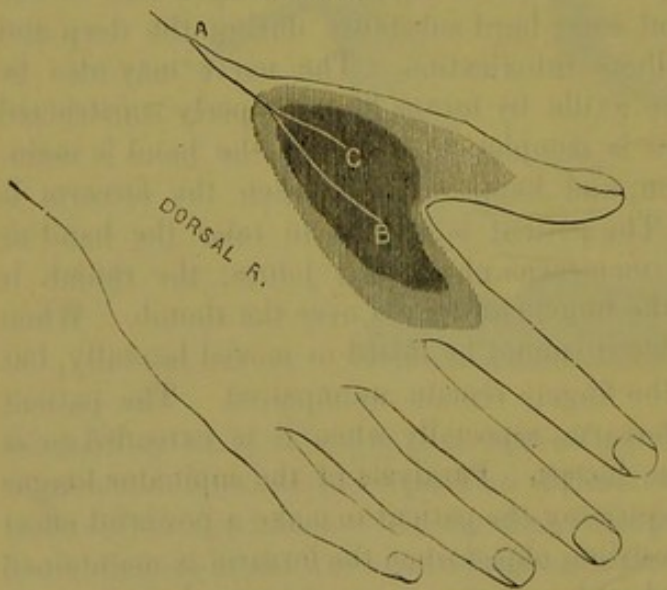


Fig. 117.—Distribution of the Sensory Nerves on the Back of the Hand. (After Krause) *r*, Radial; *m*, median; and *u*, ulnar nerve.

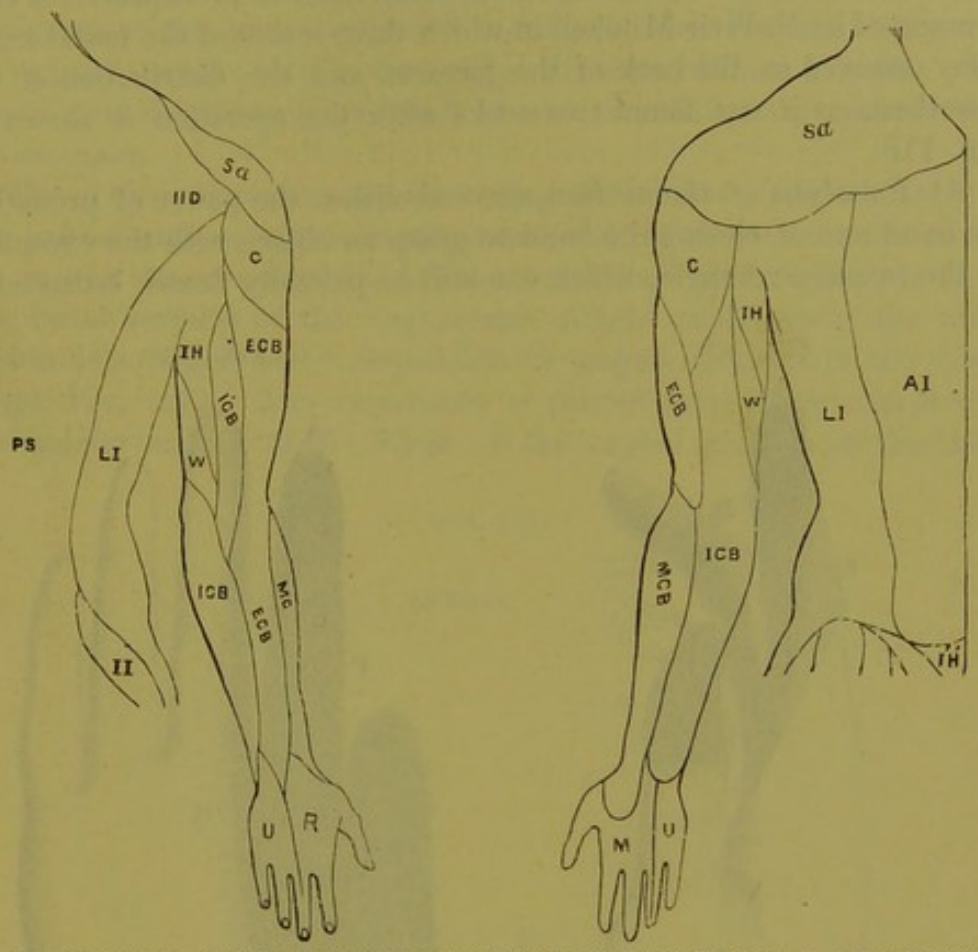
Fig. 118.—Showing the Distribution of the Anæsthesia in a Case of Division of the Musculo-spiral Nerve in the Arm. (After Létiévant.) A, Deeply shaded, to show the area of greatest anæsthesia; B, shaded in a medium degree, to show the area of partial anæsthesia; C, faintly shaded, to show the area of slight anæsthesia.

Fig. 119.—The Dorsal Aspect of the Hand Two Weeks after Resection of the Radial Nerve. (After Mitchell.) A B and A C, the length of the portion removed. The light shading represents the area in which tactile sensibility was diminished; the deep shading the area in which it was lost.

down, the dorsal surfaces of the thumb and of the index and half of the middle finger as far as to the second phalanx and the corresponding parts of the back of the hand are alone affected (Fig. 117, r). A slight

degree of anæsthesia may sometimes be found over the back of all the fingers, including the little finger, which is supposed to be entirely

Fig. 120. Fig. 121.



Figs. 120 and 121.—Cutaneous Nerves of the Trunk and Upper Extremity. (After Flower.)

Sa, Supraclavicular nerve.
IID, Second dorsal.
PS, Posterior branches of the spinal nerves.
LI, Lateral branches of the intercostal nerves.
AI, Anterior branches of the intercostal nerves.
II, Iliac branch of ilio-inguinal nerve.
I'H', Ilio-hypogastric nerve.
C, Circumflex nerve.
IH, Intercostal humeral.
W, Nerve of Wrisberg.
I'CB, Internal cutaneous branch of musculo-spiral nerve.
ECB, External cutaneous branch of musculo-spiral nerve.
ICB, Internal cutaneous nerve.
MB, Musculo-cutaneous nerve.
R, Radial nerve.
U, Ulnar nerve.
M, Median nerve.

innervated by the uluar nerve. This fact is well illustrated by a case reported by Létiévant, in which the nerve was divided in the inferior

and internal aspect of the left arm, and the distribution of the anæsthesia two and a half years after the injury is shown in Fig. 118. In most cases, however, the area of anæsthesia is much more limited than the anatomical distribution of the nerve would lead us to expect. A case is reported by S. Weir Mitchell in which three inches of the radial nerve were removed in the back of the forearm, and the distribution of the anæsthesia as it was found two weeks after the operation is shown in Fig. 119.

(4) *Paralysis of the median nerve* abolishes the power of pronating the hand and of closing the hand to grasp an object, with the exception of the two ulnar fingers, which can still be partially flexed, because the

FIG. 122.

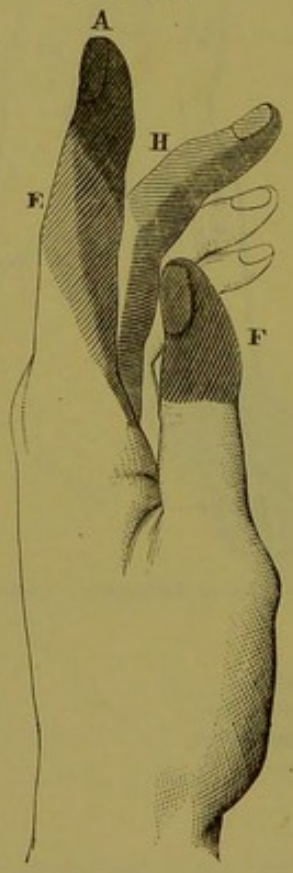
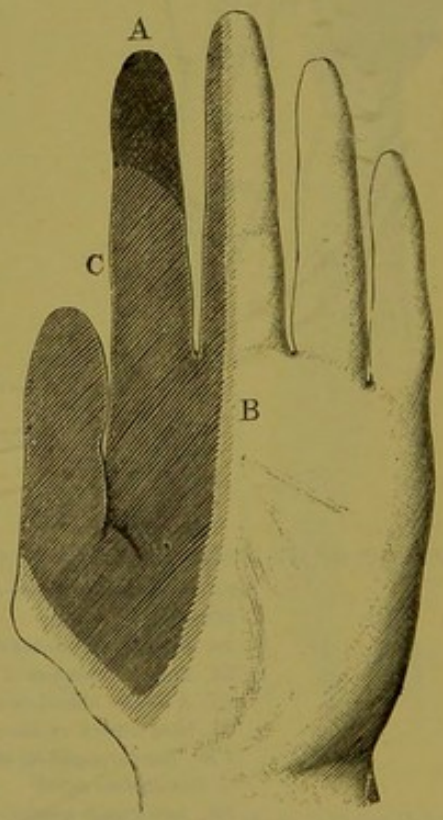


FIG. 123.



FIGS. 122 and 123.—RADIAL BORDER AND DORSAL ASPECT OF THE HAND, SHOWING THE DISTRIBUTION OF ANÆSTHESIA IN A CASE OF DIVISION OF THE MEDIAN NERVE. (After LÉTIÉVANT.)

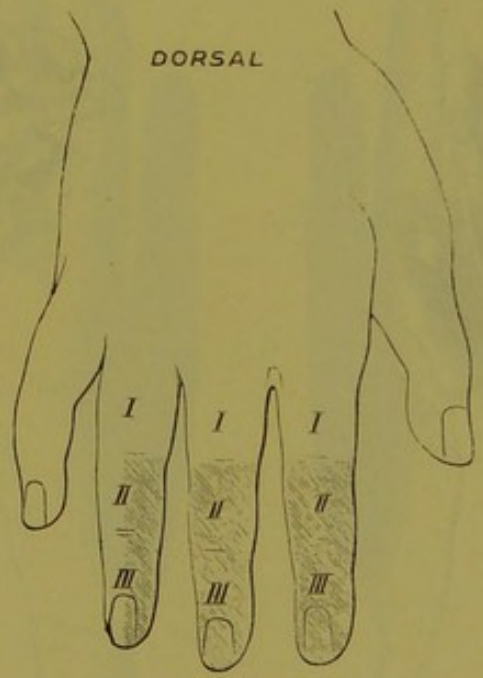
A, Deep shading representing more or less complete anæsthesia; B, E, and H, light shading, and C, medium shading, representing a slight and medium degree of anæsthesia respectively.

flexor profundus digitorum is in part supplied by the ulnar nerve. The patient is unable to abduct, oppose, or perform any of the more delicate movements of the thumb, which is permanently extended and adducted, while its metacarpal bone is drawn backwards so that it lies on a plane

with the metacarpal bones of the fingers, as in the hand of the monkey. Flexion at the wrist is accompanied by adduction, owing to the unopposed action of the flexor carpi ulnaris. Flexion of the first with extension of the second and third phalanges of the fingers can be effected by means of the interossei; and, indeed, the last two phalanges may be hyperextended owing to the unopposed action of these muscles. The muscles of the forearm and ball of the thumb become atrophied in severe cases.

The sensory disorders are, according to Létiévant, distributed over the palmar surfaces of the thumb, index, and radial border of the middle finger, the thenar eminence, and the radial half of the palm, and over the dorsal surfaces of the ungual and middle phalanges of the index and middle fingers and occasionally the ungual phalanx of the thumb (Figs. 122, 123). The anæsthesia is partial everywhere except over the palmar surface of the whole of the ungual phalanx of the index

FIG. 124.



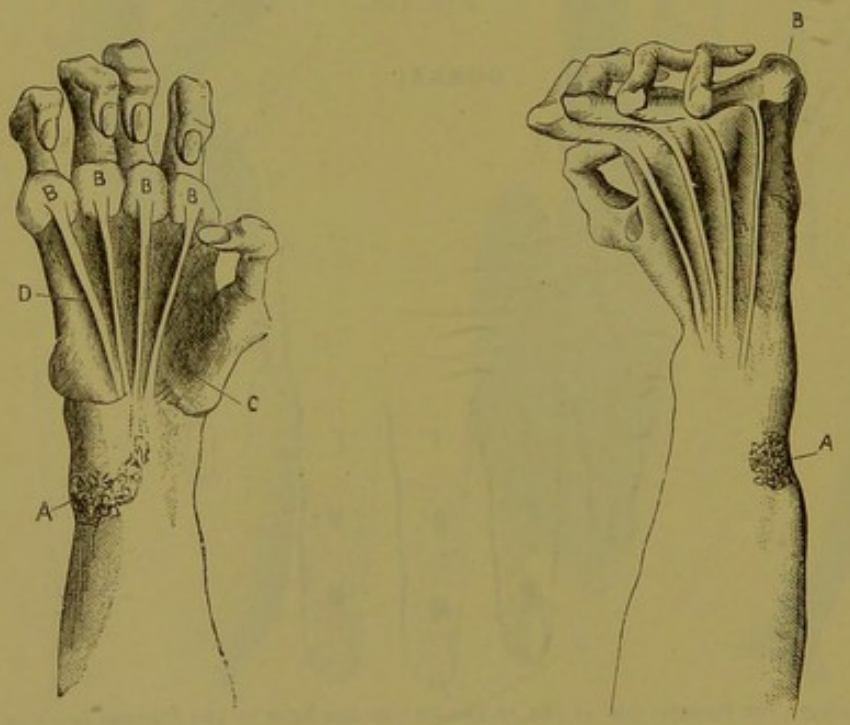
SHOWING THE DISTRIBUTION OF THE ANÆSTHESIA ON THE BACK OF THE FINGERS IN A CASE OF INJURY OF THE MEDIAN NERVE. (After BERNHARDT.)

finger, and part of the dorsal surface of the same phalanx. The different degrees of shading in Figs. 122 and 123 represent the varying degrees of anæsthesia, the very deep shading at A representing complete anæsthesia. A case is reported by Bernhardt in which the median nerve was paralyzed as the result of an injury. Four months after the injury the sensibility of the skin of the ball of the thumb and the radial half of

the palm was somewhat diminished, whilst that of the palmar aspect of the thumb, index, middle, and radial border of the ring finger was greatly lessened, the loss of sensibility becoming progressively greater from the basal to the ungual phalanges. On the dorsal aspect the sensibility of the skin of the thumb and little finger was intact over all the phalanges, but was much diminished over both borders of the middle and ungual phalanges of the index and middle fingers, and over the radial border of the corresponding phalanges of the ring finger (Fig. 124). In many cases of paralysis of the median nerve, however, the distribution of the anæsthesia is by no means so extensive as it was in the cases of Létiévant and Bernhardt.

(5) *Paralysis of the ulnar nerve* limits the power of ulnar flexion and adduction of the hand, and abolishes the power of completely flexing the last two fingers, of moving the little finger, of separating the fingers from and compressing them against the middle finger, and

FIG. 125.



1. MAIN EN GRIFFE. 2.

(1) HAND, PALMAR SURFACE. (2) DORSAL SURFACE. (After DUCHENNE.)

A, Wound of the ulnar nerve; B, Ends of the metacarpal bones; D, Tendons of the flexor sublimis; C, Muscles of the ball of the thumb.

of flexing the first and extending the second and third phalanges of all the fingers. The patient is also unable to adduct the thumb and apply it firmly to the metacarpal bone of the index finger. If the interossei and lumbricales are alone paralyzed, the combined traction of the extensors

and flexors of the fingers produces hyperextension of the first and flexion of the last two phalanges, and the hand assumes the claw-like appearance which is so characteristic of paralysis of the ulnar nerve above the wrist, and of certain cases of progressive muscular atrophy. In severe and protracted cases the first phalanges are dislocated backwards upon the metacarpal bones by the unantagonized action of the extensors of the fingers; the second phalanges become dislocated forwards on the first, and the third on the second from the unantagonized action of the flexor digitorum sublimis and profundus, respectively, and then the most characteristic form of the claw-hand (*main en griffe*) is produced (Fig. 125). The accompanying atrophy of the muscles gives to the hand some of its chief characteristics. The hypothenar eminence is flattened from atrophy of the muscles of the little finger, deep furrows

FIG. 126.

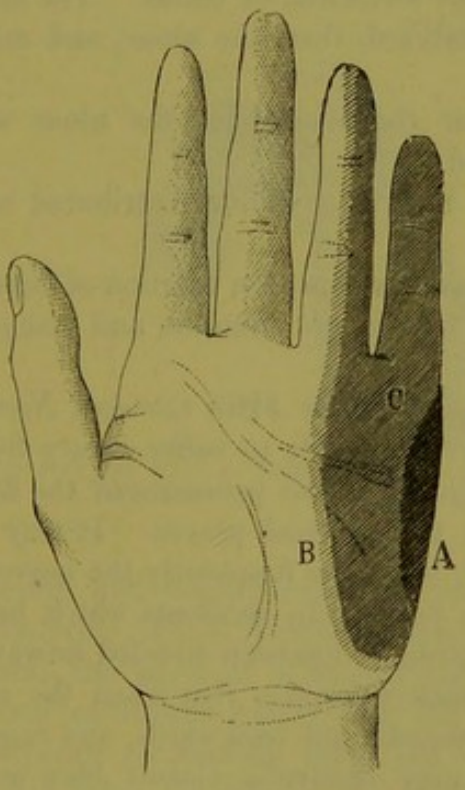
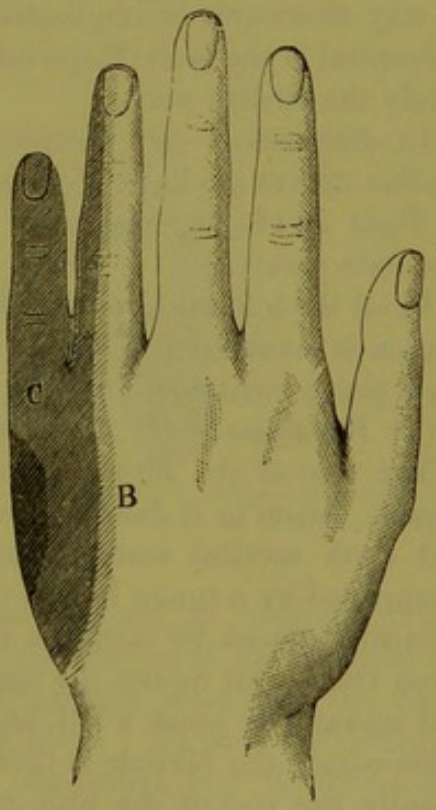


FIG. 127.



A, Deep shading, representing complete anæsthesia; B, light shading, represents slight, and C, medium shading, represents a medium degree of anæsthesia. (After LÉTIÉVANT.)

appear between the metacarpal bones from atrophy of the interossei; the metacarpal bone of the index finger may be felt immediately underlying the skin from disappearance of the abductor indicis; and when the mass of tissue which lies between the metacarpal bones of the thumb

and index finger is grasped by the observer it is felt to consist of little more than a fold of skin, owing to atrophy of the adductor pollicis and inner head of the adductor brevis pollicis.

The *sensory* disorders appear on the palmar and dorsal aspects of the little and half of the ring finger, and the corresponding part of the palm and back of the hand, but the anæsthesia is only complete over a very limited portion of the ulnar border of the hand (Figs. 126, 127). All severe lesions of the ulnar nerve are apt to give rise to glossy fingers, and various other trophic phenomena.

(6) *Combined Paralysis of the Nerves of the Brachial Plexus.*—Various kinds of paralysis occur after dislocation of the shoulder-joint.

In *subcoracoid luxations* the subjacent nerve trunks are especially liable to injury, and the whole of the nerves may be compressed or lacerated.

In *fracture* of the humerus one or more nerve trunks may be injured, or may afterwards be implicated in the formation of callus. The musculo-spiral is the most frequently paralyzed, then the ulnar, and more rarely the median nerve.

In *dislocations* and *fractures* near the elbow-joint the ulnar and median nerves are liable to be affected.

Tight bandaging may also give rise to a widely distributed and obstinate paralysis.

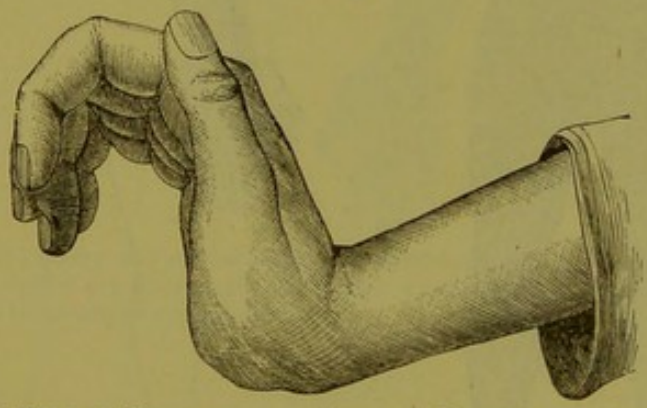
In all these forms of paralysis recovery is slow, the reaction of degeneration is manifested in the affected nerves and muscles, and many of them prove incurable.

(7) *Paralysis of the Muscles Supplied by the Fifth Cervical Nerve.*—The root of the fifth cervical nerve is liable to suffer injury from various causes as it descends over the transverse processes of the fifth and sixth cervical vertebræ to join the brachial plexus. It may be compressed by a tumor in this course, but more frequently the nerve is suddenly injured by accident at this point. In accidents which have given rise to this injury, the upper extremity has been directed forwards and upwards to break a fall, and in such a position that when the collision comes the clavicle is thrust upwards and backwards, and consequently the root of the fifth nerve may receive a violent blow as it passes over the transverse processes of one of the vertebræ. The first suggestion of this explanation came from Mr. Williamson, one of the house physicians at the Royal Infirmary, and the probabilities of it were subsequently verified on the dead subject by Mr. Collier, Demonstrator of Anatomy at Victoria University. The muscles which are paralyzed after this injury are the deltoid, biceps, brachialis anticus, and supinators of the forearm, and to a less degree the outward rotators

of the humerus. The *sensory* disorders consist of formication and numbness in the outer surfaces of the arm, forearm, thumb, and index finger. The same muscles are paralyzed in the form of paralysis described by Duchenne under the name of *obstetrical infantile paralysis* of the superior extremities. It is caused by turning or some other operative procedure during delivery.

(8) *Paralysis of the Inferior Roots of the Brachial Plexus.*—Paralysis of the roots of the eighth cervical and first dorsal nerves is observed in cases of pachymeningitis cervicalis hypertrophica when the lesion is situated on a level with the junction of the cervical and dorsal regions of the cord. The muscles supplied by the ulnar and median nerves are then paralyzed, while those supplied by the musculo-spiral nerve remain comparatively free, and consequently the hand, especially when the patient tries to grasp, assumes the claw form shown in Fig. 128. In most cases the second dorsal nerve is likewise implicated in

FIG. 128.



ATTITUDE OF THE HAND IN PACHYMENINGITIS CERVICALIS HYPERTROPHICA, WHEN THE DISEASE IS SITUATED ON A LEVEL WITH THE LOWER HALF OF THE CERVICAL ENLARGEMENT OF THE SPINAL CORD. (After CHARCOT.)

the lesion and consequently the anæsthesia caused by implication of the posterior roots is found in the area of distribution of the intercosto-humeral, the nerve of Wrisberg, the large internal cutaneous, and the terminal cutaneous branches of the ulnar nerve. In pachymeningitis the sensory conducting paths are often implicated, and the whole of the body below the level of the lesion is then anæsthetic, as is shown in Fig. 129. In a case of tubercular tumor of the spinal membranes which involved the roots of the seventh and eighth cervical and first dorsal nerves—the posterior root of the seventh cervical was not much implicated—the anæsthesia was distributed as is shown in Fig. 130.

(9) *Rupture of the Brachial Plexus.*—Rupture of the brachial plexus has generally been caused by the patient falling from a height and grasp-

ing some object during his fall, by severe pulling of the arm, or by the fall of a heavy weight on the shoulder. Immediately after the accident all the muscles of the upper extremity are paralyzed. The latissimus dorsi and the lower two-thirds of the pectoralis major are also paralyzed; but the upper third of the pectoralis major, the pectoralis minor, and the internal and external rotators of the humerus are unaffected, but

Fig. 129.

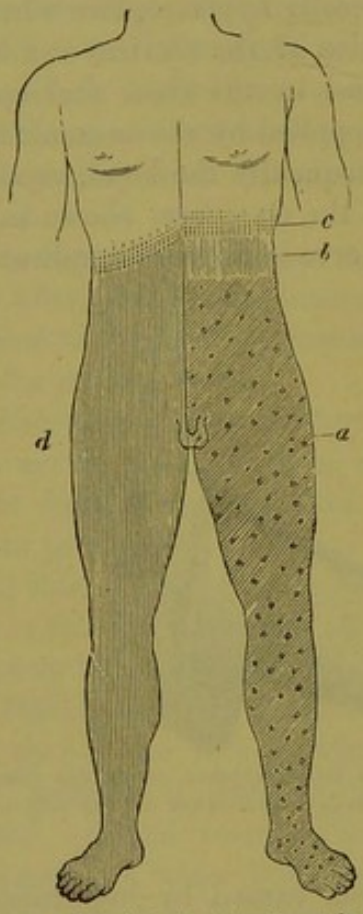


Fig. 130.

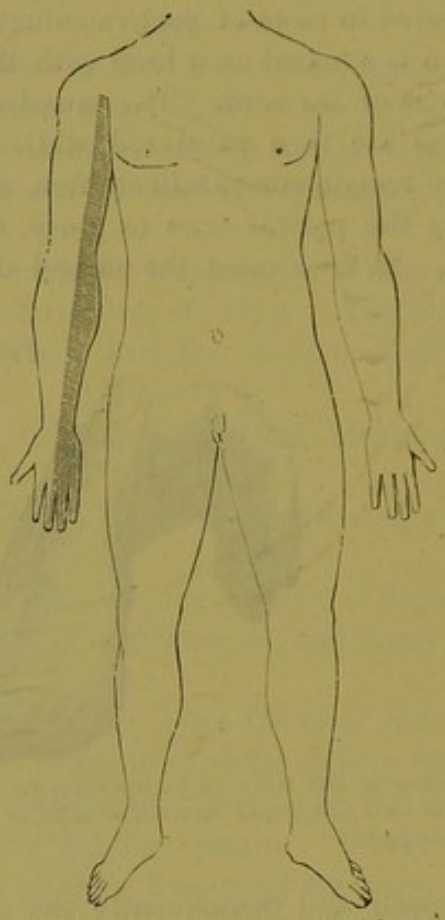
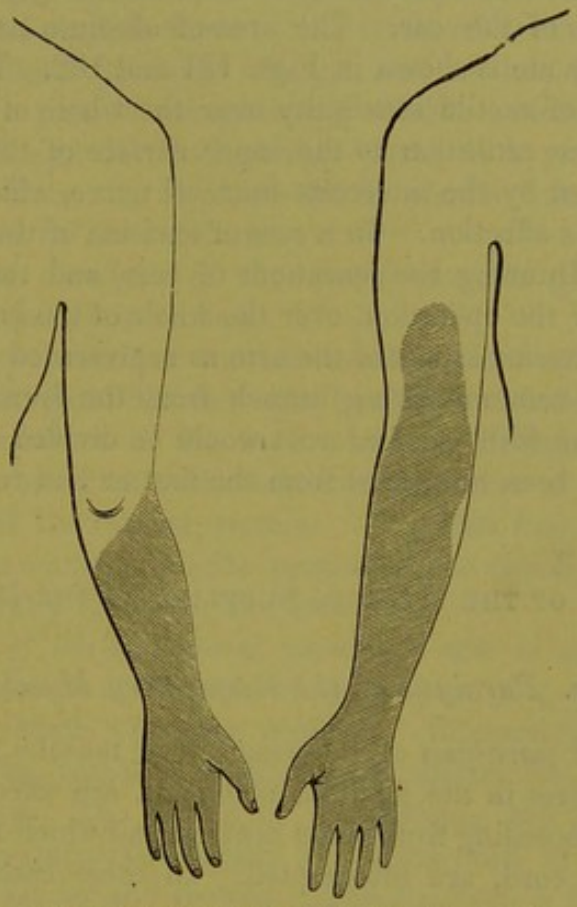


Fig. 129.—The shaded portion shows the distribution of anæsthesia in a case of cervical pachymeningitis, in which the lesion was situated on a level with the eighth cervical and first and second dorsal nerves.

Fig. 130.—The shaded portion shows the distribution of anæsthesia in a case in which a tubercular tumor was situated on a level with the roots of the seventh and eighth cervical and first dorsal nerves.

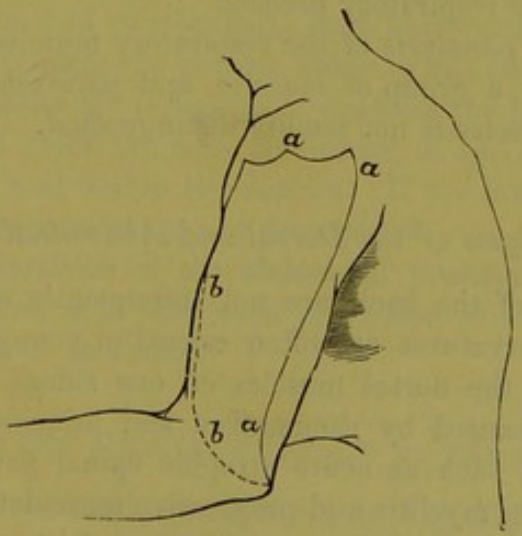
the serratus magnus may be relatively feeble, although not completely paralyzed. In a case under my care, however, the latissimus dorsi, the deltoid, biceps, brachialis anticus, and the supinators of the forearm were found to have completely recovered when the patient was examined eighteen months after the accident, although these muscles were known to have remained completely paralyzed for eight months. Oculo-pupillary phenomena are always observed in rupture of the brachial plexus. On the affected side there are paralytic myosis, diminution of

FIG. 131. FIG. 132.



FIGS. 131 and 132.—Posterior and anterior aspect respectively of the arm in a case of rupture of the brachial plexus, the shaded area showing the distribution of the anæsthesia. By an oversight the right arm is represented as being affected instead of the left.

FIG. 133.



The line *a a a*, on the outer surface of the arm, marks the boundary above and outside of which sensation is preserved. The *dotted* line *b b* marks the same for the *inner* surface of the arm. (After MAURY and DUHRING.)

the palpebral fissure, and often relative elevation of temperature in the external meatus of the ear. The area of absolute anæsthesia in the case observed by me is shown in Figs. 131 and 132. There was a relative diminution of tactile sensibility over the whole of the skin of the upper arm. The sensation to the inner surface of the arm would be probably supplied by the intercosto-humeral nerve, which would remain uninjured in this affection. In a case of excision of the brachial plexus by Maury and Duhring the sensations of pain and touch were absent, six months after the operation, over the whole of the hand and forearm, and over the anterior aspect of the arm as represented in Fig. 133, *a a*. In this case the communicating branch from the fourth and the fibres derived from the fifth cervical root would be divided, while in rupture these fibres had been uninjured from the first or had recovered.

9. Paralysis of the Muscles Supplied by the Dorsal Nerves.

a. Paralysis of the Respiratory Muscles.

(1) *Complete paralysis* of the respiratory muscles occurs when the respiratory centres in the medulla oblongata are affected, or when the motor tracts proceeding from these centres, and which run in the lateral columns of the cord, are interrupted. In these cases rapid death is inevitable.

(2) *Unilateral paralysis* of the respiratory muscles does not immediately endanger life. This form of paralysis may be caused by unilateral lesions of the spinal cord, but cerebral disease does not interfere much with the respiratory process.

(3) *Peripheral paralysis* of the respiratory muscles only affects individual muscles or a group of muscles, and paralysis of one or two of the intercostal muscles is not readily distinguished.

b. Paralysis of the Dorsal and Abdominal Muscles.

The extensors of the back are not infrequently more or less paralyzed. Spinal curvatures are often caused in young people by a relative feebleness of the dorsal muscles of one side. Paralysis of these muscles may be caused by rheumatism and injuries of the back, and by spinal disease, such as acute atrophic spinal paralysis and all the forms of ascending myelitis and progressive muscular atrophy, while it gives to pseudo-hypertrophic paralysis one of its most characteristic features.

(1) *Bilateral paralysis* of the extensors of the dorsal region gives rise to a large and equable curve of the vertebral column; the patients are bent and doubled up as in old age, and are unable to hold themselves erect, but passive straightening of the vertebral column can easily be effected, and this distinguishes paralytic kyphosis, as the condition is called, from the kyphosis which results from muscular contracture or disease of the vertebræ.

(2) *Unilateral paralysis* of the dorsal muscles gives rise to the various forms of paralytic scoliosis.

(3) *Bilateral paralysis* of the extensors of the lumbar region presents very characteristic features—the lumbar vertebræ are arched forwards, forming a hollow in the back which is increased by the upper part of the body being thrown backwards in order to compensate for the incurvation of the lumbar portion. A plumb line allowed to drop from the most prominent spinous process of the dorsal vertebra clears the sacrum generally by one or two inches.

(4) *Paralysis of the abdominal muscles* is rare as an isolated affection, but is common as a symptom of spinal paralysis, and occurs occasionally in progressive muscular atrophy. Bilateral paralysis of the abdominal muscles renders all expiratory efforts like coughing and singing ineffective. The power of compressing the abdomen is impaired, and consequently difficulty is experienced in evacuating the contents of the bladder and rectum. The abdomen is large and protuberant, its walls are relaxed, and the patient is unable to raise the upper part of the body from the recumbent position, or to sit up in bed without being propped up with the hands. In walking or standing the upper part of the body is bent slightly forwards and balanced exclusively by the lumbar muscles, and consequently each backward movement of the centre of gravity renders the patient liable to fall on his back, because the abdominal muscles are incapable of drawing the body forwards. A plumb line let fall from the most prominent of the spinous process of the vertebræ falls well within the sacrum. If the lower extremities are paralyzed, this symptom cannot be determined.

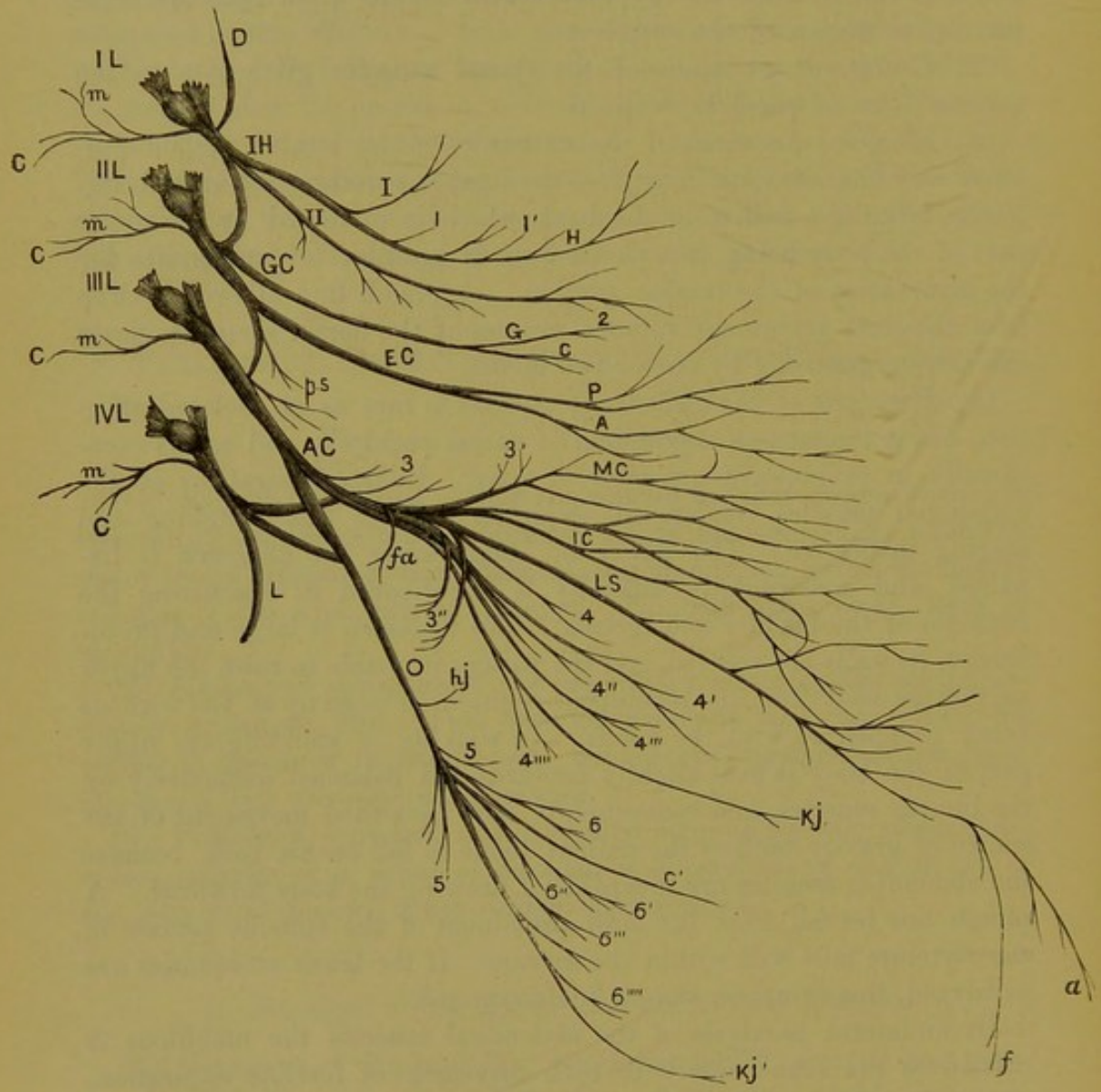
In unilateral paralysis of the abdominal muscles the umbilicus is carried to the sound side with each movement of forcible expiration, and during the act of straining.

10. Paralysis of the Muscles Supplied by the Lumbar Plexus.

a. Paralysis of the anterior crural nerve (Fig. 134, AC) arises from injuries of the vertebral column and pelvis, tumors, and extravasation of blood in the cauda equina, inflammation of the knee-joint, psoas

abscess, fracture of the thigh, dislocation of the hip-joint, wounds and injuries of the lower abdominal and crural regions, pelvic and crural tumors, and neuritis, while it is a frequent symptom of all forms of spinal paralysis.

FIG. 134.



LUMBAR PLEXUS.

IL, IIL, IIIL, and IVL.—The first, second, third, and fourth lumbar nerves, respectively.
D, Communicating branch from the last dorsal nerve.
CCCC, Posterior cutaneous branches.
mmmm, Branches to the muscles of the back.
IH, Ilio-hypogastric nerve.
I, Iliac branch, cutaneous, to surface of gluteal region.
H, Hypogastric branch, cutaneous, to surface of hypogastric region.
1, Muscular branch to obliquus internus.
1′, Rectus abdominis.

II, Ilio-inguinal nerve, cutaneous, to inguinal region and scrotum.
GC, Genito-crural nerve.
G, Genital branch to spermatic cord or round ligament.
2, Muscular branch to cremaster.
C, Crural branch, cutaneous, to surface of upper part of front of thigh.
EC, External cutaneous.
P, Posterior branch, cutaneous, to upper and outer part of thigh.
A, Anterior branch, cutaneous, to front of thigh.
ps, Muscular branches to psoas muscle.
AC, Anterior crural nerve.
3, Muscular branches to iliacus.
3′ Muscular branches to sartorius.
3″, Muscular branches to pectineus.
fa, Branch to femoral artery.
MC, Middle cutaneous to front of thigh.
IC, Internal cutaneous to inner part of thigh and leg.
LS, Internal or long saphenous.
a, Cutaneous over inner ankle.
f, Cutaneous to inner side of foot.
4, Muscular branch to rectus femoris.
4′, Muscular branch to vastus externus.
4″, Muscular branch to crureus.
4‴, Muscular branch to subcrureus.
4⁗, Muscular branch to vastus internus.
Kj, Branch to knee-joint.
O, Obturator nerve.
hj, Branch to hip-joint.
c′, Communicating with branches of internal cutaneous and internal saphenous.
5, Muscular branch to pectineus.
5′, Muscular branch to obturator externus.
6, Muscular branch to adductor longus.
6′, Muscular branch to gracilis.
6″, Muscular branch to adductor brevis.
6‴ 6⁗, Muscular branch to adductor magnus.
Kj′, Branch to knee-joint.
L, Communicating branch to fifth lumbar nerve.

Symptoms.—The patient is unable to flex the leg at the hip-joint or raise the body from the recumbent position. He cannot extend the leg, and when sitting he is unable to move the leg forwards. Standing and sitting upright are rendered insecure, while walking, jumping, and running are difficult or impossible, and the difficulty is much increased when both nerves are affected.

The *sensory disorders* extend over the lower two-thirds of the thigh, the region of the knee, and the inner side of the leg and foot.

b. Paralysis of the obturator nerve (Fig. 134, O) may be caused by strangulated obturator hernia, and the pressure of the head of the child, or of obstetric instruments, in difficult deliveries.

Symptoms.—The patient is incapable of adducting the thigh, of pressing the knees together, or of crossing one leg over the other. Outward rotation of the thigh is rendered difficult, and the affected leg soon tires in walking. A slight degree of anæsthesia extends down the inner side of the thigh as far as the knee.

c. Paralysis of the gluteal nerves (Fig. 135, SG) is caused by tumors and lesions of the cauda equina, fractures of the sacrum and pelvis, and various diseases of the spinal cord.

FIG. 135.

SACRAL AND COCCYGEAL NERVES.

VL, IS, IIS, IIIS, IVS, VS, VIS.—Fifth lumbar, and first, second, third, fourth, fifth, and sixth sacral nerves.

LS, Lumbo-sacral cord.

c, *c*, Posterior cutaneous nerves.

m, Branches to muscles of back.

1, Branches to pyriformis muscle.

3, Muscular branches to obturator internus.

3′, Muscular branches to gemellus superior.
3″, Muscular branches to gemellus inferior.
3‴, Muscular branches to quadratus femoris.
SG, Superior gluteal nerve.
2, Muscular branches to gluteus medius.
2′, Muscular branches to gluteus minimus.
2″, Muscular branches to tensor vaginæ femoris.
12, Muscular branches to gluteus maximus.
I G, Inferior gluteal nerve (cutaneous).
I P, Inferior pudendal nerve, cutaneous, to perineum and scrotum, or external labium.
C″, Cutaneous branch to back of thigh and upper part of leg.
P, Pudic.
dp, Dorsalis penis seu clitoridis.
13, Muscular branch to transversus perinæi.
13′, Muscular branch to erector penis.
13″, Muscular branch to compressor urethræ.
13‴, Muscular branch to accelerator urinæ.
13⁗, Branch to the bulb.
asp, Anterior superficial perineal to perineum and scrotum, or external labium.
psp, Posterior superficial perineal to perineum and scrotum, or external labium.
I H, Inferior hemorrhoidal.
IVS, Fourth sacral nerve.
14, Muscular branch to levator ani.
14′, Muscular branch to sphincter ani.
14″, Muscular branch to coccygeus.
GS, Great sciatic nerve.
hj, Branch to hip-joint.
4, Muscular branches to semi-tendinosus.
4′, Muscular branches to semi-membranosus
5, Muscular branches to adductor magnus.
5′, Muscular branches to biceps.
IP, Internal popliteal.
6, Muscular branch to gastrocnemius (inner head).
6′, Muscular branch to gastrocnemius (outer head).
6″, Muscular branch to popliteus.
6‴, Muscular branch to soleus.
PT, Posterior tibial nerve.
7, Muscular branch to tibialis posticus.
7′, Muscular branch to flexor longus digitorum.
7″, Muscular branch to flexor longus pollicis.
pc, Plantar cutaneous.
Int. P., Internal plantar nerve.
8, Muscular branch to abductor pollicis.
8′, Muscular branch to flexor brevis pollicis.
8″, Muscular branch to first lumbricalis.
8‴, Muscular branch to second lumbricalis.
8×, Muscular branch to flexor brevis digitorum.
*t*1 to *t*3, Digital branches.
Ext. P. External plantar.
*t*4 and *t*5, Digital branch to fifth and outer half of fourth toe.
10′, Muscular branch to flexor accessorius muscle.
10″, Muscular branch to abductor minimi digiti.
9, Muscular branch to flexor minimi digiti.
9′, Muscular branch to fourth lumbricalis.
DB, Deep branch of external plantar nerve.
9″, Deep branch of external transversalis pedis.
9‴, Deep branch of external third lumbricalis.
*p*3, *p*2, *p*1, Deep branch of external third, second, and first plantar interossei muscles.
*d*4, *d*3, *d*2, *d*1, Deep branch of external fourth, third, second, and first dorsal interossei muscles.
10, Deep branch of external adductor pollicis.
ES, External or short saphenous to outer side of foot.

EP, External popliteal.
CP, Communicans peronei.
C', Cutaneous to outer side of leg.
MC, Musculo-cutaneous nerve.
11, Muscular branch to peroneus longus.
11', Muscular branch to peroneus brevis.
AT, Anterior tibial nerve.
11'', Muscular branch to tibialis anticus.
11''', Muscular branch to extensor longus digitorum.
11'''', Muscular branch to extensor longus pollicis.
11×, Muscular branch to extensor brevis digitorum.
Kj, Muscular branch to knee-joint.
at, Articular branch to tarsus.

Symptoms.—The power of rotating the leg both inwards and outwards is interfered with, and that of abduction is impaired. Some uncertainty is felt in standing; the patient experiences great difficulty in ascending stairs, and when the body is inclined forwards it is difficult to raise it to the erect posture. The characteristic rotation of the pelvis, produced by the contraction of the gluteus medius during locomotion is not effected, and the muscle is not felt rigid on the side of the active leg, as in health, when the hand is laid over the pelvis above the trochanter. Disorders of sensibility are only present when other nerves are coincidently affected.

11. Paralysis of the Muscles Supplied by the Sacral Plexus.

Etiology.—Paralysis of the sciatic nerve or of one of its branches may be caused by injuries of various kinds, and compression of the nerve by tumors; by injury of the plexus during the extraction of the child by means of the forceps, tumors and abscesses on the pelvis, or compression of the gravid uterus, and by fractures of the vertebral column and tumors of the cauda equina. Paralysis of the nerve may also be caused by a rheumatic inflammation of its sheath, and by neuritis from extension of inflammation to it from neighboring diseased textures, while it sometimes occurs after acute diseases and as a symptom of hysteria. Paralysis in the region of distribution of this nerve is an almost constant symptom of disease of the spinal cord, and of many cases of cerebral paralysis.

a. In *paralysis of the musculo-cutaneous and anterior tibial nerves* (Fig. 135, MC and AT) the foot cannot be flexed or abducted, and can only be incompletely adducted. It hangs down in a flaccid condition, and in walking the toes, being depressed, are apt to trip the patient at every inequality of the ground. The necessary elevation is given to the foot in advancing during locomotion by flexion of the limb at the hip- and knee-joints, and the point of the foot is the last part to leave

the ground and on planting it down the outer border of the foot and the toes touch the ground first. This mode of progression is frequently observed in cases of infantile paralysis, and is very characteristic.

(1) In *paralysis of the tibialis anticus* the power of producing dorsal flexion and adduction of the foot is limited, and the inner border and the point of the foot can no longer be raised, though these movements may in part be vicariously executed by the extensor digitorum communis and the extensor longus pollicis.

(2) In *paralysis of the extensor digitorum communis* the power of producing dorsal flexion of the foot, and abduction of it in the flexed position is diminished, and extension of the basal phalanges of all the toes cannot be effected.

(3) In *paralysis of the extensor longus pollicis* the power of producing dorsal flexion is diminished and that of extending the great toe abolished.

(4) In *paralysis of the peroneus longus* abduction of the foot in the extended position is impossible, the arch of the foot becomes flattened, and the inner border no longer touches the ground because the head of the first metatarsal bone is no longer drawn downwards, and a peculiar kind of flat foot is thus produced.

(5) In *paralysis of the peroneus brevis* pure abduction of the foot is rendered impossible, although it may be effected along with dorsal flexion by means of the extensor digitorum communis and along with plantar flexion by means of the peroneus longus.

(6) In *paralysis of the extensor digitorum communis brevis* the power of extending the basal phalanges of the last four toes is impaired. These forms of paralysis may be combined in various ways and then the affected muscles can only be recognized by the most careful and prolonged investigation.

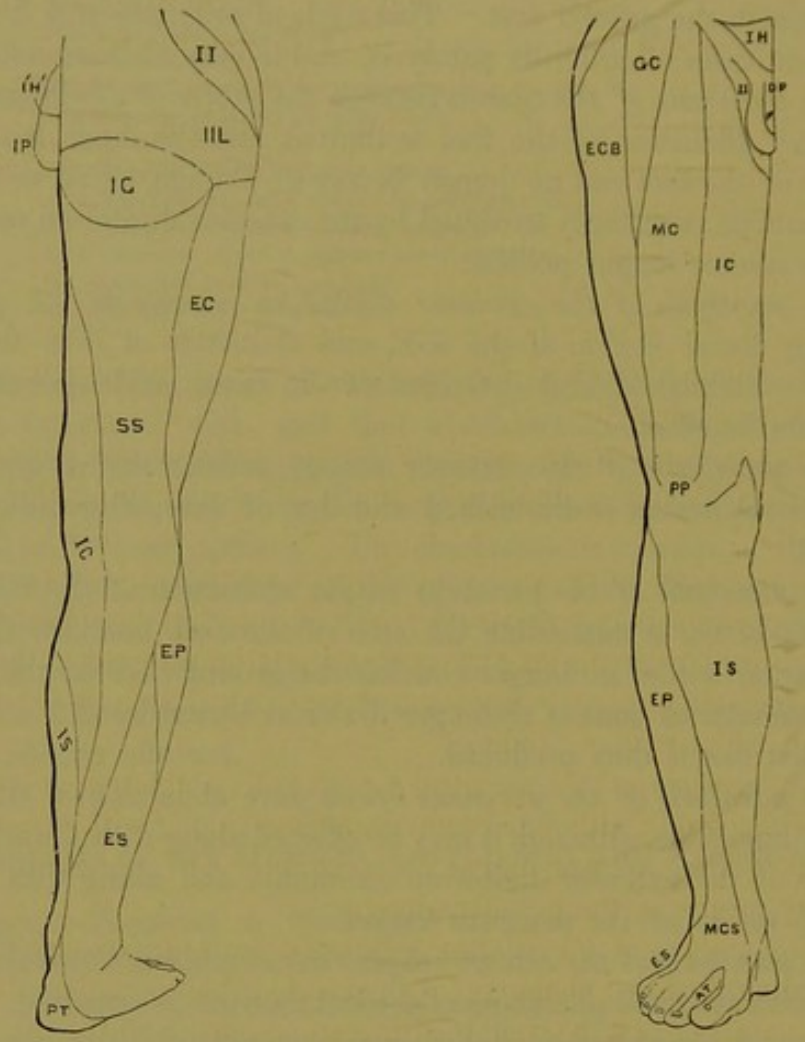
The *sensory* disturbances are limited to the anterior and external part of the leg, the dorsum of the foot, and the greater part of the toes (Fig. 136, MCS).

b. In *paralysis of the tibial nerve* the patient is unable to extend the foot, or produce flexion or lateral movements of the toes.

(1) In *paralysis of the gastrocnemius and soleus* the foot cannot be extended and the patient cannot stand upon the toes, and when secondary contraction of the anterior muscles of the leg ensues the foot assumes a hook-like position in which the heel alone touches the ground, forming talipes calcaneus.

(2) In *paralysis of the tibialis posticus* the power of abducting the foot and of raising its inner border is diminished.

FIG. 136.



CUTANEOUS NERVES OF THE LOWER EXTREMITY. (After FLOWER.)

Lumbar Plexus

IH, Ilio-hypogastric nerve.
II, Ilio-inguinal.
IIL, Second lumbar nerve.
GC, Genito-crural.
EC, External cutaneous.
MC, Middle cutaneous.
IC, Internal cutaneous.
IS, Internal saphenous.
PP, Plexus patellæ.

Sacral Plexus.

DP, Dorsalis penis of pudic.
IP, Inferior hemorrhoidal of pudic.
P, Superficial perineal of pudic and inferior pudendal of small sciatic.
IG, Inferior gluteal of small sciatic.
SS, Small sciatic.
EP, Branches from external popliteal.
ES, External saphenous.
MCS, Musculo-cutaneous.
AT, Branches of anterior tibial.
PT, Branch of posterior tibial.

(3) In *paralysis of the flexor communis digitorum* flexion of the two distal phalanges of the toes is impossible.

(4) In *paralysis of the flexor longus pollicis* flexion of the great toe is incomplete.

(5) In *paralysis of the abductor and adductor pollicis* the power of moving the great toe laterally is lost.

(6) In *paralysis of interossei* the patient is unable to flex the first or to extend the two distal phalanges of the toes, or to effect separation of them. The first phalanges become hyperextended on the metatarsal bones and the distal phalanges are strongly flexed, so that the toes no longer touch the ground with their bulbous extremities, and a claw-like position is thus produced, as occurs in the hand from paralysis of the corresponding muscles.

The *sensory disorders* are limited to the posterior surface of the leg, the sole of the foot, and the plantar surface of the toes (Fig. 136, PT).

c. In *paralysis of the trunk of the sciatic nerve* all the muscles of the leg and foot are paralyzed, and, in addition, the patient is unable to flex the leg upon the thigh, to approximate the heel to the gluteal region, or to offer any resistance when an attempt is made to extend the leg.

The region of the knee, the back part of the thigh, and ultimately the buttock and perineum are successively affected by anæsthesia, according as the lesion is situated at higher levels in the trunk of the nerve. If the lesion is situated in the hollow of the sacrum or in the cauda equina the anæsthesia affects the whole sacral region, the scrotum or labia, and penis, urethra, bladder, and rectum.

The *vaso-motor* disturbances consist of cyanosis, mottling and coldness of the skin of the paralyzed leg. Increase of temperature has occasionally been observed as a transitory symptom.

The *trophic* disturbances are muscular atrophy, ulceration of the skin, eruptions of herpes and pemphigus, and bedsores on the sacrum, ankles, and heels.

CHAPTER VI.

ATROPHIC PARALYSES (*continued*).

II. MULTIPLE NEURAL PARALYSES.

1. Idiopathic Progressive Multiple Neuritis.

This affection is usually ushered in by more or less fever; the patient soon complains either simultaneously or successively of tingling pains and feelings of coldness and numbness in the feet and hands, the territory of the ulnar nerve being peculiarly liable to be affected at an early period of the disease. Pressure over circumscribed spots of the nerves causes great pain, which radiates towards the periphery, and both the skin and the muscles in the area of distribution of the affected nerves are so extremely sensitive to pressure that the patient avoids any movement of the limbs, and complains bitterly of the slightest touch or of passive movements. Alongside this hyperæsthesia to pressure there is well-marked anæsthesia of certain forms of sensibility. Tactile sensibility is indistinct, and the patient is unable to localize accurately, with closed eyes, a point touching the skin, or to distinguish two points as separate until they are far removed from one another. In the early stage of the disease it is somewhat difficult to distinguish whether the motionless condition of the limbs arises from the reluctance of the patient to move them for fear of provoking pain, or from decided motor paralysis. Soon, however, distinct paralysis sets in, and the affected muscles undergo rapid atrophy and manifest the "reaction of degeneration." The paralysis begins in the small muscles of the hands and feet, gradually spreads upwards, and successively invades the larger muscles of the limbs and those of the trunk, while in some cases the muscles of the bladder and rectum are involved. The small muscles of the hands and the extensors of the forearm are usually affected to a greater degree than the other muscles of the upper extremities, and, consequently, the fingers and hands assume distorted positions similar to the claw-hand of progressive muscular atrophy. Some of the cutaneous reflexes may be normal, while others are variously altered. The plantar reflex is absent in the advanced stage of the disease; even at first it is absent to slight stimulation, but a strong stimulus is said to provoke an

exaggerated reaction. The tendon-reactions, and more especially the patellar tendon-reaction, appear to be abolished at an early period of the disease. The affected nerves are, as already mentioned, painful on pressure of particular points, and at times the nerve may be felt to be distinctly swollen at these points; while in some cases it may be the seat of an œdematous swelling of considerable size. The trophic disorders consist, besides the atrophy of the muscles already described, of alterations of the nails, which sometimes become brittle, cracked, strongly curved, and of a yellowish or brownish color; and of an increased development of hair, or in some cases disappearance of the hair from some spots of the skin, the skin of the fingers being especially liable to become hairless and glossy. Patches of the skin are liable to become of a red or bluish color from vaso-motor changes, and the subcutaneous tissue may also become decidedly œdematous.

In its onset progressive multiple neuritis is essentially an acute affection, considerable sensory and motor disturbances being established in the course of a few days. In its further progress the affection becomes chronic, and any change which takes place either by way of amendment or extension of the disease may occupy many weeks. After from nine to eighteen months, complete recovery may take place or the recovery may be partial, the muscles supplied by the ulnar nerve remaining sometimes permanently disabled. The disease sometimes terminates fatally from implication of the intercostal and bulbar nerves, but the fatal result is often caused by a complication like chronic nephritis, or an intercurrent affection like pneumonia or tuberculosis.

2. Lead Paralysis.

Almost all the muscles of the body may be affected by an atrophic paralysis in chronic lead-poisoning, although certain groups are attacked by preference. In partial paralysis the extensor muscles of the forearm are more frequently affected than any other group, and consequently, when the arms are held out horizontally, with the hands in a state of pronation the hands hang powerless, this condition being graphically called *wrist-drop*. The common extensors of the fingers are the first to be attacked, then the extensors of the index and little fingers, the extensor secundii internodii pollicis, the extensors of the wrist, the extensor primi internodii pollicis, and the extensor ossis metacarpi pollicis are successively invaded. The supinator longus is spared until a comparatively late period of the disease and it is never invaded unless the paralysis extend to the muscles of the upper arm. Occasionally the paralysis may begin in the muscles of the upper arm and then the

deltoid, biceps, brachialis anticus, and supinator longus are almost simultaneously attacked. When the muscles of the inferior extremities are paralyzed, the anterior muscles of the leg are generally the first to be attacked, and the extensor muscles of the thigh are the next most likely to be invaded. In some cases all the muscles of both upper and lower extremities are paralyzed, whilst on rare occasions the muscles of the thorax and back, those of phonation and speech, the intercostal muscles, and even the diaphragm have been implicated. Paralysis of the muscles of the glottis has been observed by Trousseau in horses employed in red lead factories. The sphincters remain always unaffected in the paraplegic form. The paralyzed muscles undergo rapid atrophy, and lose their faradic contractility before voluntary power is completely abolished, while the galvanic degeneration reaction appears in them at an early period. The patellar tendon-reaction is often lost in cases in which there is no perceptible atrophy of the muscles of the lower extremities, but I have found this reaction present in several cases in which the wrist-drop was very pronounced.

Trèmor is sometimes observed in lead-poisoning; it is often limited to the upper extremities, but may become general in aggravated cases. Ataxic symptoms have occasionally been observed along with anæsthesia.

Sensory disorders are often absent in cases of wrist-drop, but when the paralysis extends to the muscles of the lower extremities a good deal of anæsthesia may be present, especially in the area of distribution of the cutaneous branches of the external popliteal nerve. In some cases, however, the skin over the back of the hands and forearms is more or less anæsthetic, and a certain degree of loss of feeling may be observed over the surface of the chest and abdomen, whilst in a few cases the veil of the palate and uvula, and even the conjunctiva, may be completely anæsthetic. Symptoms of sensory irritation are also often present in chronic lead-poisoning. Cutaneous hyperæsthesia may accompany attacks of pain, while *arthralgia*, occurring in paroxysms and situated either in the extremities or in the jaws, is often a prominent symptom.

Lead colic is one of the most frequent and important symptoms of the disease; the pains, which are chiefly referred to the umbilical region, are more or less continuous, but they are liable to paroxysmal exacerbations of great severity. The special senses are sometimes affected. Diminution of taste on one-half the tongue and of smell in one nostril has been observed, but deafness is said to result only from local disease of the ear. But visual disorders are more frequent and more important than those of the other special senses. These consist of transitory amblyopia without ophthalmoscopic changes; persistent amblyopia

passing on to amaurosis of both eyes and rarely of one only, attended by atrophy of the optic nerve; amblyopia with double optic neuritis, and amblyopia with albuminuric retinitis in association with granular kidneys.

Psychical disorders are often present in aggravated cases. Before the outbreak of pronounced cerebral symptoms the patient generally suffers from headache, vertigo, and drowsiness during the day with sleeplessness at night; or there may be a state of agitation, or complete apathy. The psychical disorders consist of hallucinations of sight and hearing, with a quiet delirium, or the patient may have an attack of furious delirium. Convulsions are frequently present, and they may be partial, being then limited to the muscles of the face or to one or more of the limbs, or they may assume the form of an attack of eclampsia. These convulsions are generally followed by a stage of profound unconsciousness with stertorous breathing. Apoplectiform attacks are liable to occur in the last stage of lead paralysis, and inasmuch as the patient often recovers motor power quickly these attacks cannot always be due to hemorrhage.

3. Alcoholic Paralysis.

Persons who have indulged too freely in the use of alcohol are liable to suffer from a progressive paralysis which is accompanied by more or less rapid atrophy of the affected muscles. The affection generally begins, so far as the paralysis is concerned, by a gradually increasing weakness of the lower extremities. The patient, after suffering from numbness, tearing pains, and often also from distressing cramps in the lower extremities, experiences a progressively increasing difficulty in maintaining the erect posture, and on attempting to walk the legs are spread asunder in order to widen the base, while the gait is uncertain, or walking may be impossible without the support of two sticks or of an attendant. The movements of the hands are also sometimes irregular and uncertain and when an arm is stretched out to perform an intended movement the object may only be reached after numerous slight lateral and vertical jerking interruptions. At this stage of the disease the alcoholic subject, provided the person be sober during examination, appears to be in a moderately advanced stage of locomotor ataxia, and the similarity between the two diseases is rendered still more striking by the fact that the patellar tendon-reaction is absent in the alcoholic disease as well as in ataxia. A closer examination, however, will show that the alcoholic affection is, from the first, a paralytic one. Provided the patient is able to walk, it will be seen that the toe of the advancing

foot always drops, just as occurs in cases of infantile paralysis when the extensors of the foot and toes are paralyzed, and when the legs of the patient are uncovered in bed it will be seen that the feet assume the position of paralytic talipes equinus, or rather talipes equina varus, because the tibialis anticus is not paralyzed to the same degree as the extensors and peronei muscles. It will also be found that the extensors of the front of the thigh are very feeble. A methodical examination of the upper extremities proves equally instructive. The patient on holding his arms out horizontally, the forearms being in a state of pronation, may be able to hold the hands and fingers extended, but the slightest pressure on the backs of the fingers causes flexion at the metacarpo-phalangeal joints, on the back of the hand causes flexion at the wrist, and in many cases the little finger is seen to drop, the patient being unable to maintain it fully extended at the metacarpo-phalangeal joint. On electrical exploration the faradic contractility will be ascertained to be much diminished or completely lost in the extensors of the leg and those of the front of the thigh, and considerably diminished in the extensors of the forearm, while the galvanic excitability of these muscles manifests the qualitative changes of the "reaction of degeneration."

After another deep carouse the whole clinical picture of the disease changes. The patient now lies helpless in bed, being unable to stand, and possibly not even to sit up in bed. But the most remarkable feature of this stage of the disease is presented by the upper extremities. The extensors of the forearms are completely paralyzed and a double wrist-drop results just as in lead paralysis, and to render the similarity between the two diseases more striking the supinator longus is comparatively spared in the former as in the latter disease. The flexors of the fingers and wrist and the intrinsic muscles of the hand are also very feeble, but I have not seen them completely paralyzed. In the upper arm the triceps is much more feeble than the biceps, and consequently the forearm is maintained during repose at a right angle to the arm, but when the patient moves the extremity the forearm becomes flexed at an acute angle to the arm, and the curled-up attitude now assumed by the extremity may appear to a superficial observer to be imposed upon it by an active spasm and not by a paralysis, so that the disease has a certain resemblance to tetany. The movements at the shoulder are not much interfered with. On electrical exploration it will now be found that, speaking generally, the extensors of both lower and upper extremities give the complete, and the remaining muscles of the limbs Erb's partial, "reaction of degeneration."

If the disease advances further, the diaphragm becomes paralyzed and the abdomen sinks as the chest expands and protrudes slightly as

the chest contracts. At a still later period the intercostal muscles become paralyzed, and the patient after a short interval, in which the breathing is maintained by the upper thoracic muscles, dies from asphyxia. The order in which the various muscular groups are attacked is important, because in all the ascending forms of spinal paralysis the intercostal muscles are affected before the diaphragm, and the intrinsic muscles of the hand before the extensors of the forearm. The cutaneous *reflexes* and the mechanical irritability of the affected muscles are diminished or absent, while the tendon reactions are nearly always lost at an early period of the disease.

Sensory disturbances are always present. The patients suffer from tearing pains in the lower extremities which are apt to become peculiarly distressing at night, and they complain of a distressing burning of the feet, as well as of numbness and various other paræsthesiæ. Cutaneous hyperæsthesia is occasionally present, but it is by no means in so marked a degree as muscular hyperæsthesia, which forms one of the most characteristic features of the disease. The slightest pressure on the muscle calls forth loud cries from the patient, and suffices to induce in females a prolonged attack of hysterical sobbing. The hyperæsthesia of the lower extremities, most probably that of the muscles, is indeed sometimes so great that the weight of the bedclothes is intolerable to the patient, and the feet have to be protected by cradles. The sense of temperature is sometimes perverted in such a way that all objects feel cold on contact. In the more advanced stages of the disease, the hyperæsthesia gives place to more or less anæsthesia, sensory conduction is retarded, the tactile sense is diminished, and Remak's double sensation may be observed. At times, the spine is tender to pressure, but pain and stiffness of the vertebral column are not prominent symptoms.

Vaso-motor disorders are generally present in the form of redness of the feet, and of œdema of the ankles and backs of the feet and hands. The œdema of the lower extremities is sometimes present to so great a degree as to suggest the presence of a cardiac complication, but a physical examination negatives this idea.

The *trophic* disorders consist of local asphyxia, and even gangrene of the extremities has been occasionally observed as a complication. *Psychical* disturbances are by no means rare; they consist of restlessness, sleeplessness, marked loss of memory with diminished intelligence and a stupid and stolid expression, delusions or hallucinations, and delirium, these being present either separately or in various combinations. The girdle sensations, disorders of the movements of the pupils and eyeballs, disturbances of the functions of the bladder and rectum,

and arthropathies, which are so frequently observed and so characteristic of tabes dorsalis, are absent in alcoholic paralysis.

Alcoholic paralysis is said to occur with greater frequency in women than in men, but without questioning the truth of this statement, I am convinced that it is more frequent in men than is generally supposed. Out of eight well-marked cases of the disease which I have seen within the last six months, four were men and four women, and of these, two men and two women had complete double wrist-drop. One man died from respiratory paralysis and a woman from cerebral complication; of the remaining six, one has almost completely recovered and five are still under treatment.

The symptoms of the pronounced form of alcoholic paralysis in which double wrist-drop occurs begin more or less acutely, generally after an unusually deep carouse, but as a rule the affection assumes a chronic course, although a few cases are rapidly fatal from respiratory paralysis. In chronic cases death may take place from a cerebral complication or from an intercurrent disease such as cirrhosis of the liver. Most cases, however, recover completely, provided the use of alcohol is wholly prohibited, but a relapse is liable to take place on recurrence to the old habit, and when through long indulgence the affection becomes very chronic, the paralysis may prove incurable.

4. Diphtheritic Paralysis.

Diphtheritic paralysis begins occasionally towards the close of the febrile attack, but in most cases it does not declare itself until from eight days to three weeks or more after the disappearance of the false membrane from the throat. The first symptom to attract attention is a nasal quality of voice and the ejection of fluids through the nose, caused by paralysis of the veil of the palate, and when the back of the throat is examined it is seen that the soft palate hangs loose and flapping from the roof of the mouth, and that it is not retracted on reflex irritation or during phonation, deep inspiration, and attempts at deglutition. The patient can swallow solids better than fluids, inasmuch as the latter are more readily ejected through the nose than the former, but when the pharyngeal muscles become paralyzed, as they frequently do, the difficulty of deglutition becomes greater, and the patient is in danger of being starved from inability to swallow sufficient food, or of being suffocated by the bolus becoming impacted in the pharynx, or finding its way into the air-passages, the latter accident being much facilitated by the fact that anæsthesia of the mucous membrane of the superior part of the larynx is often superadded to the palatal and pharyngeal paralysis.

Difficulty of respiration may be caused by paralysis of the abductors of the vocal cords, or the voice may be altered from a slight degree of hoarseness up to complete aphonia, owing to paralysis of the muscles which render tense and approximate the vocal cords. The tongue, the lips, and the masticatory muscles may likewise be more or less paralyzed, and the symptoms then are very like those of progressive bulbar paralysis. But the ocular muscles, and especially the internal muscles of the eye are those which are most frequently attacked after the muscles of the throat. The patient is unable to read small print and cannot accommodate the eye for near objects, this defect being due to paralysis of the ciliary muscle, and mydriasis has occasionally been observed from paralysis of the sphincter of the iris. Paralysis of one or more of the external muscles of the eye is declared by the presence of diplopia or strabismus, while ptosis on one or both sides reveals paralysis of the elevator muscle of the eyelid. Complete paralysis of the facial muscles has not been observed in diphtheritic paralysis, but a certain degree of immobility of the face is often present, which, when conjoined to the defect of vision, gives to the patient an idiotic expression, although psychical disturbances are absent during the whole course of the disease, except perhaps some degree of apathy and a disinclination to make any intellectual effort. The paralysis now extends to the muscles of the limbs, and the inferior extremities are usually the first to be attacked, but in most cases the muscles of the superior extremities are subsequently implicated, and ultimately those of the trunk, including the muscles of respiration, may be invaded.

In most cases the motor disorder simply amounts to a certain degree of feebleness of the limbs, which manifests itself in an uncertain and staggering gait that may be mistaken for ataxia, the similarity between the two affections being rendered still closer by the presence in diphtheritic paralysis of certain sensory disorders, which will be immediately described, and absence of the patellar tendon-reflex. The paralysis after a time increases in intensity and extent, and the feebleness of the lower extremities may become so great that the patient is unable to maintain the erect posture and becomes confined to his bed, while the upper extremities are so much affected that the patient is unable to button his clothes, or to carry a spoon to his mouth, and the difficulty of seizing small objects is greatly increased by the presence of sensory disorders. The muscles of the neck may be paralyzed even before those of the extremities. After a time the erector muscles of the spine are invaded, and the patient may then be unable even to sit up in bed.

In some cases the paralysis assumes the hemiplegic form, but a careful examination shows that the side which is presumed to be healthy is

enfeebled, and that the hemiplegic distribution of the paralysis arises from the fact that there is a disproportion between the degree to which the motor weakness has attained on each side of the body. The diaphragm may ultimately be paralyzed, and should the intercostal muscles be simultaneously attacked the danger of death from asphyxia is great.

Sensory disorders always accompany the loss of motor power in diphtheritic paralysis. The mucous membrane of the soft palate and pharynx is anæsthetic, and the tactile sensibility of the mucous membrane of the lips, cheeks, and tongue is often diminished. The motor disorders of the extremities are accompanied, or possibly preceded, by sensory phenomena, the patient complains of numbness and tingling pains in the feet and hands, and of a feeling of cold and of weight in the limbs, the tactile sensibility is blunted, and in some cases there is complete anæsthesia. The inferior extremities are usually the first to be affected, and patients cannot then feel the ground on which they tread, or feel it only imperfectly; they often state that it seems as if they were walking on cotton, wool, or some other soft substance, and they are unable to walk with closed eyes without danger of falling, just as occurs in locomotor ataxia. When the hands are attacked the patients feel as if their fingers were covered with gloves, and tactile sensibility may be so blunted that the person is unable to seize and hold small objects without the aid of sight. The disorders of cutaneous sensibility are very variable in their extent and distribution, as well as in their degree of intensity. The anæsthesia begins in the periphery of the limbs and extends towards the trunk; it often remains limited to the distal segments of the extremities, but sometimes becomes general, and Trousseau mentions a case in which the patient had almost no consciousness of the passage of the urine and feces, although micturition and defecation were voluntarily performed. In a few cases the anæsthesia has been limited to the superior extremities, and in exceptional cases cutaneous hyperæsthesia, accompanied by articular pains, has been observed. The special senses are also sometimes affected in diphtheritic paralysis. We have already seen that vision is rendered indistinct by paralysis of the ciliary muscles, but in other cases amblyopia, proceeding to complete amaurosis, may result from insensibility of the retinæ. Taste has occasionally been found diminished, probably never abolished, but loss of smell and deafness have been more frequently observed.

The *reflex* action of the palate is diminished or lost at an early period of the disease, but there are not very accurate statements with regard to the reflex actions of the extremities. The patellar tendon-reactions are lost even before paralysis of the lower extremities becomes

very pronounced and consequently the liability to mistake diphtheritic paralysis, at a certain stage of its development, for locomotor ataxia is very great. The paralyzed muscles manifest the reaction of degeneration on electrical exploration.

Visceral disorders are numerous and important in this form of paralysis. We have already seen that the respiratory muscles may be attacked, and that the disease may terminate fatally by arrest of respiration. In other cases the heart becomes affected with paralysis. The cardiac disorder is manifested by an excessively slow, irregular, and feeble pulse, and by paroxysms of palpitation, accompanied by præcordial anxiety and dyspnœa. The nervous disorders are sometimes ushered in by a remarkable slowing of the pulse, attacks of syncope, and vomiting, all of these being symptoms which are to be referred to interference with the functions of the pneumogastric nerve, and at a late period of the disease the patient may suffer from præcordial agony, and the pulse becomes slow, irregular, and so small as to be almost undistinguishable, while death by syncope is not uncommon. I have known glycosuria to occur after an attack of diphtheria, and the patient has since made a complete recovery. Obstinate constipation is a frequent symptom of diphtheritic paralysis; it is usually a late symptom, and generally appears after the extremities are invaded, but in the last stage of the disease the rectum may be paralyzed, and then constipation is succeeded by an involuntary discharge of feces. After a time the urinary organs may become affected; the bladder becomes greatly distended, and unless the catheter be used, dribbling of urine occurs from overdistention; but this is succeeded by paralysis of the sphincter, with incontinence. The patient likewise loses his virile power, and in some cases there is complete impotence.

The duration of diphtheritic paralysis is very variable. When the veil of the palate alone is affected the paralysis disappears, as a rule, in from ten days to three weeks, but when the paralysis is widely distributed over the body it lasts from two to four months, but rarely persists beyond six months. Diphtheritic paralysis usually terminates in complete recovery or death, and only very exceptional cases are recorded in which the recovery was incomplete. About twelve per cent. of all cases terminate fatally, death taking place by asphyxia from respiratory paralysis or from food getting into the glottis, inanition from inability to swallow, nervous exhaustion, or syncope.

III. REFLEX ATROPHIC PARALYSES.

Etiology.—The most characteristic examples of this form of atrophic paralysis are to be found in chronic diseases of the joints, while the extensor muscles are often found to be feeble and emaciated. It is well known to surgeons that the deltoid is often feeble and wasted in chronic disease of the shoulder-joint, while Hunter and more recently Adams directed attention to the flattening of the gluteal region which is observed in chronic disease of the hip-joint. The extensors of the thigh are liable to become feeble and emaciated in diseases of the knee-joint, and the extensors of the fingers and thumb, and to a less extent the flexors also, are similarly affected in cases of rheumatoid arthritis. In some cases a slight and transitory synovitis gives rise to a degree of paralysis and wasting of the extensor muscles which is altogether disproportionate to the severity and duration of the joint affection.

The affection described under the name of *reflex* or *urinary* paraplegia does not differ in its *causation* from the paralysis of joint diseases, being set up by irritation of one of the viscera, such as the urinary organs, uterus, or intestinal canal. The cause of the paralysis is sometimes an injury or neuritis of a peripheral nerve. In severe cases of neuritis from injury the inflammatory process may pursue an ascending course and set up an acute or subacute myelitis, but in the cases under consideration at present it is probable that the symptoms are caused by irritation of the gray matter in the absence of serious structural changes.

Symptoms.—In paralysis of the extensors secondary to diseases of the joints, the affected muscles become soft and flabby, and emaciated, while their motor power is greatly diminished. The mechanical irritability of the affected muscles is increased, the reaction of their tendons is normal or slightly exaggerated, and lively contractions are said by Charcot to be obtained by passing sparks of static electricity through them, but the faradic and galvanic excitability is diminished without qualitative changes. In cases of rheumatoid arthritis the muscles of the body often undergo a diffused atrophy which predominates in the extensors. The deltoid, the triceps, the extensors of the wrist, fingers, and thumb, the gluteal muscles, the extensors of the thigh, and the extensors of the toes are feeble and emaciated, and although the flexors are likewise implicated, yet the paralysis in them being to a less degree they undergo adapted contractions and the limbs become then fixed in flexed positions. The mechanical irritability of the partially paralyzed muscles is in excess, the tendon-reactions are exaggerated, and even ankle clonus may be elicited in both ankles.

In *urinary* paraplegia the lower extremities are paretic, but never completely paralyzed; the reflexes are said now to be increased; and there is complete absence of pains in the loins, girdle pains, dysæsthesiæ, anæsthesia, muscular tension and contractures, paralysis of the bladder, and bedsores and other trophic disturbances. The gait corresponds to the well-known shuffling gait of old age and is never of the spastic variety. The paralysis is very variable in its degree and extent and rapid improvement may take place if there be an amelioration of the peripheral lesion. This affection is generally met with in old people with prostatic disease, and it may be associated with symptoms caused by anæmia of the cord from disease of the bloodvessels—vascular sclerosis—so that it is not always easy to determine the share each lesion takes in the production of the symptoms. The paralytic symptoms are sometimes caused by remote irritation. The most striking examples of paralysis from remote irritation are afforded by those cases in which a paraplegia, or even a paralysis of the upper extremities suddenly disappears on the expulsion of a tape, or of lumbricoid worms. The paralyses which result from injury of remote parts are well illustrated by several cases which occurred during the American war. The case of a soldier is reported by S. Weir Mitchell, who had received a bullet wound on the right side of the neck, which wounded the pharynx and fractured the hyoid bone, but did not implicate any important nerve trunk. The injury was immediately followed by paralysis of both upper extremities; the left extremity recovered rapidly, and the right improved considerably but remained feeble.

In another case reported by the same author, a wound in the soft parts of the thigh which did not injure any large nerve trunk, was followed by right-sided hemianalgesia and paralysis of all the four extremities. The left arm recovered rapidly, but the improvement in the other limbs was only slow, and the hemianalgesia persisted.

Neuritis sometimes gives rise to paralysis not only of the muscles supplied by the affected nerve itself, or by nerves derived from the same plexus, but also to paralysis of muscles supplied by nerves derived from the plexus of the opposite side or from a different plexus altogether. This form of paralysis is caused by a *secondary myelitis* set up by the diseased nerve. This condition is well illustrated by a remarkable case reported by Charcot and Féré, in which the sciatic nerves were injured by a severe blow over the point of their emergence from the pelvis. At first the patient suffered from the usual symptoms of inflammation of the sciatic nerves. At the end of three months after the injury, both lower extremities were so feeble that the patient was unable to maintain the erect posture, and then there was also an involuntary passage of the

urine and feces and loss of virile power. A careful examination of the patient ten months after the injury showed that all the muscles supplied by the left sciatic nerves were paralyzed and atrophied, and manifested, to a marked degree, the "reaction of degeneration," these muscles being probably affected by injury of the different fibres of the nerves. In addition to paralysis of these muscles on the left side, the corresponding muscles of the right limb were also feeble and emaciated, but they only gave the slightest form of the "reaction of degeneration," while the muscles supplied by the left superior gluteal nerve, which received no direct injury, were also paralyzed and atrophied and manifested the "reaction of degeneration." The muscles supplied by the anterior crural and obturator nerves were not paralyzed on either side. The presence in this case of paralysis of the sphincters, of the muscles of the right lower extremity, and of the gluteus medius and minimus of the left side shows conclusively that a consecutive myelitis of the gray substance was set up by the local injury to the nerves. The excess of the patellar tendon-reaction on the right side is explained by Charcot by supposing that the ganglion cells connected with the anterior crural nerve were maintained in a state of irritation.

CHAPTER VII.

ATROPHIC PARALYSES (*continued*).

IV. SPINAL ATROPHIC PARALYSES (POLIOMYELOPATHIES).

1. Paralysis Ascendens Acuta (Acute Ascending Paralysis, Landry's Paralysis).

Etiology.—Acute ascending paralysis often begins in the absence of any recognizable predisposing cause, but it is thought likely that syphilitic infection and rheumatism may develop a liability to it. Men are more frequently attacked than women, and the majority of cases have occurred between twenty and forty years of age. The exciting causes are exposure to cold, suppression of menses, and convalescence from acute disease, such as typhoid fever, variola, and pleurisy.

Symptoms.—Premonitory symptoms consisting of slight fever, shooting pains in the back and limbs, formication and numbness in the feet and finger-tips, and a feeling of great weariness, debility, and general discomfort may be present several days before the occurrence of paralysis. The characteristic symptoms of the disease now declare themselves by a paralysis which begins in the muscles of the feet and spreads upwards to those of the thigh. The paralysis progresses with so much rapidity that in a few days the lower extremities may be completely paralyzed, and the limbs lie flaccid and powerless in any position in which they may be placed, and without offering the slightest opposition to passive movements.

The paralysis continues to advance steadily upwards, the muscles of the trunk are invaded, and the patient is unable to sit up in bed; the small muscles of the hand are probably the next to be attacked, and delicate manipulations, like those of writing, become impossible; the muscles of the upper arm and shoulder are successively invaded; the acts of urination, defecation, and respiration are now rendered increasingly difficult by the progressive paralysis of the abdominal and intercostal muscles; and when the muscles of the tongue, lips, pharynx, and œsophagus become implicated a fatal termination is near.

A considerable degree of emaciation may occur in this disease as in other acute affections, but the paralyzed muscles do not undergo a

rapidly progressive atrophy, and with rare and doubtful exceptions the electric excitability of the paralyzed nerves and muscles remains normal. Reflex action, which is preserved or occasionally increased during the first few days of the disease, becomes diminished and finally extinguished. The patellar tendon-reactions have been found absent at an early period of the disease in those cases in which the state of these reactions has been tested.

The *sensory disorders* consist merely of numbness and tingling in the fingers and toes, but, as a rule, the cutaneous sensibility is found to be normal on objective examination, although occasionally a slight diminution of tactile sensibility has been observed at the periphery of the extremities. The pupils are sometimes unequal, and towards the end the pulse is very frequent. The functions of the bladder and rectum are usually unaffected, there are no bedsores, the general health is often good, and the intellect is clear throughout the whole course of the disease. The disease, as we have seen, generally begins in the lower extremities, but in some cases it begins in the upper extremities, or, rarely, in the region of distribution of the bulbar nerves, and then it pursues a descending course.

The duration of the affection is somewhat variable. In some cases it terminates fatally in the course of two or three days, while occasionally it lasts from two to four weeks, the average of fatal cases being from eight to twelve days. Death is caused by arrest of respiration subsequent to implication of the bulbar nerves. The disease may end in recovery. It may cease to progress at any stage of its development, and even after the cranial nerves are implicated the case must not be regarded as altogether hopeless. In favorable cases improvement begins at an early period of the disease, the parts last attacked being the first to recover.

2. Poliomyelitis Anterior Acuta (Kussmaul) (Acute Inflammation of the Anterior Gray Horns, Acute Atrophic Spinal Paralysis).

Etiology.—This affection is most frequently met with in childhood, the disease appearing between the ages of six months and three years in thirty-two out of forty-four cases observed by West. It may, however, occur at so early an age as ten days, and a disease essentially the same occurs in the adult. Heine asserts that the disease occurs in the healthiest children, and that neither sex nor hereditary predisposition appears to exercise any influence in its causation. It appears,

according to Drs. Barlow and Wharton Sinkler, to be more common during the summer than the winter months.

Injuries of various kinds are often said to be the cause of the disease, and nurses are frequently blamed unjustly by parents who, unable to believe that such a striking phenomenon as paralysis can occur suddenly without appreciable cause, imagine that the child has been lamed by a fall through the carelessness of its attendant. The most usual exciting causes are painful dentition, and exposure to cold, more especially when the body is overheated, and the affection often occurs in children, and occasionally in the adult, during or soon after an attack of measles, scarlatina, smallpox, typhus, and other acute affections.

Symptoms.—Although this disease is essentially the same in children as in adults, yet the symptoms differ so much in the two as to demand separate description. The disease will be first described as it occurs in children.

a. Infantile Spinal Atrophic Paralysis.

It will conduce to clearness if, like Laborde, we divide the clinical history of this affection into the periods of (1) invasion; (2) remission; (3) regression of paralytic phenomena; (4) atrophy with deformities. It must, however, be remembered that these periods overlap, and that this subdivision is merely adopted for the sake of convenience.

(1) *The Period of Invasion.*—The disease is commonly ushered in by a more or less intense fever, which is often preceded by general malaise, pain in the head or in the back, mental irritability, fretfulness, and startings of the limbs. As a rule, the fever is of short duration, lasting only from one to two days. In some cases it passes off in a few hours, while in others it continues from fourteen to sixteen days, or even longer. As the fever increases the cerebral symptoms become more pronounced, confusion of ideas and slight somnolency are observed, and the child may become unconscious, or delirium of varying degrees of intensity may supervene. The disease was ushered in by convulsions in thirty out of seventy cases collected by Duchenne, these convulsions being, most probably, the ordinary eclamptic attacks which so frequently precede every acute febrile disease in childhood. All general symptoms are sometimes absent; the child is put to bed apparently in good health, and is found paralyzed in the morning.

(2) *The Period of Remission.*—After the initial symptoms have subsided it is observed that the patient is unable to move one or more of his limbs, or the paralysis may not attract attention until the child is

taken out of bed for some purpose and then the relaxed and helpless condition of the affected extremities can hardly fail to be noticed. The paralysis is probably never fully developed at once; it increases gradually and reaches its maximum extent and degree in the course of a few hours, in a day or two, and occasionally not until a longer period has elapsed. Relapses sometimes occur. After a first attack one limb is paralyzed and a few days afterwards, during which rapid improvement has been taking place, the child is seized with a second febrile attack and other limbs become paralyzed. A case is reported by Laborde in which the paralysis did not become permanently established until the third attack.

The *distribution* of the paralysis is very variable. It often involves not only the four extremities, but also the muscles of the vertebral column, and even the intercostal muscles and those of the neck are not always spared. Sometimes the lower extremities alone are paralyzed, but the upper extremities are seldom exclusively affected. The paralysis assumes in some cases the form of a hemiplegia, and in these the side of the neck, of the face, and of the tongue may be implicated at first and may on rare occasions remain permanently paralyzed.

Sensory disorders are almost entirely absent during the whole course of the disease. At the outset patients may complain of pains and various paræsthesiæ, but these symptoms are of short duration. A certain degree of cutaneous hyperæsthesia or rather hyperalgesia has been described as being present during the febrile stage, but this tenderness to touch probably depends upon affections of deeper structures, such as rheumatic inflammation of joints. The cutaneous sensibility is sometimes blunted in the paralyzed extremities in old standing cases, but this probably depends upon underlying nutritive and vascular changes.

Reflex action is completely lost in all the muscles which are severely paralyzed and it is much lowered or temporarily extinguished in the muscles which are slightly affected.

The *tendon-reactions* are also absent in the paralyzed muscles.

The *functions of the bladder and rectum* are rarely affected. During the first days there may be retention, but more frequently there is incontinence, and the stools may be passed involuntarily. These disorders disappear in from three to eight days from the commencement, except in young children, in whom a slight incontinence of urine may remain for some time.

(3) *The Period of Regression.*—After a certain time, which varies from a few days to a few weeks, some of the paralyzed muscles begin to improve, and in a few cases complete recovery takes place. The cases which recover have been described by Kennedy under the name

temporary spinal paralysis. But, as a rule, only some of the muscles are completely restored, while the rest remain permanently paralyzed. When the paralysis is general it often happens that the upper half of the body is the first to show signs of amendment, the paralysis disappearing rapidly from the neck, upper extremities, and trunk, and becoming restricted to the lower extremities. This improvement, which Laborde calls the period of *first regression*, is followed after a variable interval of time by a second period of amendment, which the same author calls the period of *second regression.* During the second regression some of the muscles of the lower extremities undergo a progressive improvement and one limb may be restored to full motor power, while one or more of the muscular groups of the other limb may remain permanently affected, particularly the anterior and external group of the leg. In some cases improvement takes place from below upwards instead of from above downwards, and then the paralysis becomes permanently localized in a superior extremity, or occasionally in the trunk or neck. The permanently paralyzed muscles are implicated in groups according as they are associated in their action, and not in accordance with the peripheral distribution of their motor nerves.

(4) *The Period of Atrophy and Deformities.*—All the muscles which are severely paralyzed become the subjects of a *rapidly progressive atrophy*, and even those which are but slightly affected waste to some extent, but soon recover. The atrophy becomes well marked in the course of a few weeks, in the muscles which are severely affected, and after a time they become so attenuated that the bones seem to underlie the skin. Sometimes the muscular atrophy is masked by the accumulation of fat in the connective tissue, and consequently the loss of volume is not always a trustworthy test of the degree to which the muscle has become atrophied.

The *electrical reactions* of the affected nerves and muscles may vary from a simple diminution of the normal reactions to the *partial* or the *complete* reaction of degeneration. It was first shown by Duchenne that the faradic irritability of both nerves and muscles sinks quickly in those which are severely attacked, and becomes lost in them on the seventh day or during the course of the second week. He laid it down as a rule that all the paralyzed muscles in which the faradic irritability is only more or less diminished during the course of the second week do not remain permanently paralyzed, and that the restoration is the more prompt and complete the less the faradic irritability is diminished.

Arrest of development of the osseous system often occurs, and it may be altogether out of proportion to the degree in which the muscles are paralyzed. The greater part of the muscles of a limb may, indeed, be

lost while the bones are almost entirely unaffected, and, conversely, a limb may be considerably shortened while only one or two muscles are atrophied. The paralyzed lower extremity may be found from two to six inches shorter than the corresponding healthy limb, and the upper extremity may be similarly affected, although not generally to the same degree. The long bones are thinner than normal; they are porous, friable, and yielding; their epiphyses and processes grow smaller and less distinct; the paralyzed hand or foot is shorter, narrower, and thinner than the sound one; and even the pelvis may be arrested in its development.

The *joints* become unusually movable, partly from disappearance of the articular extremities of the bones and partly from relaxation and stretching of their ligaments, and occasionally the changes undergone are so great that the patient is able to dislocate a joint without experiencing any discomfort.

The *skin* of the affected extremity is flabby and so inelastic that it retains for a long time slight pressure marks, such as that made by the stocking. The surface of the limbs is of a mottled or bluish color, it is remarkably cold to the touch, and in old cases its temperature may be from 5° to 12° F. lower than that of the corresponding healthy limb. The skin is liable to chilblains, and indolent ulcers form on slight provocation, these nutritive changes being accompanied, and probably caused by a diminution in the calibre of the arteries.

The *deformities* occurring in the affected limbs give to this disease some of its most characteristic features. Some pathologists believe that the deformities are produced by the predominant action of the healthy muscles, the normal *tonus* being destroyed in the affected muscles, but Volkmann asserts that the deformity is produced mainly by the weight of the limb itself, because the position generally assumed by the foot is only a higher degree of that which it assumes when unsupported and left free from the action of the muscles. The influence of gravity on the position of the limb ought certainly not to be ignored, but the position assumed by the foot in talipes calcaneus when the muscles of the calf are paralyzed shows that gravity is not the only, perhaps not the chief, force which is operative in the production of these deformities. Two other factors, at least, must be taken into account. The first is that the paralyzed muscles often permit the limb to assume a position in which the ends of their healthy antagonists are more or less permanently approximated, and the latter consequently become permanently shortened by undergoing "adapted atrophy." The second factor is that the paralyzed muscles themselves may become permanently shortened either from arrested development or from pro-

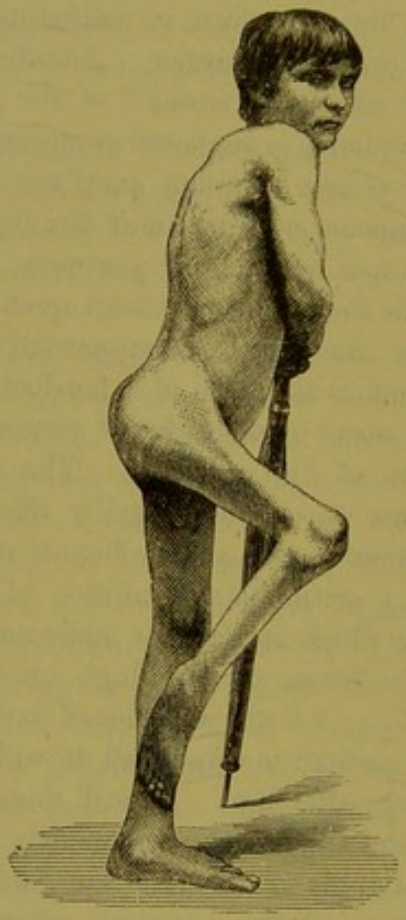
liferation and subsequent retraction of their connective tissues. Of all the deformities which occur in infantile paralysis, talipes equinus and equino-varus are the most frequent, because the muscles most frequently paralyzed are the long extensors of the toes, the tibialis anticus, the extensors of the great toes, and the peronei muscles. When the anterior group and the adductors of the foot are affected at the same time talipes equino-valgus, and when the muscles of the calf alone are affected talipes calcaneus is produced, but this form is exceedingly rare, and simple talipes varus is of still rarer occurrence. Another common deformity is the "pes cavus"—"talus pied creux" of the French, in which the sole is hollowed and the instep is rendered prominent. Duchenne thinks it is caused by a more or less complete paralysis of the muscles of the calf, along with simultaneous contraction of the flexors of the foot, either the long flexors of the toes or the long peroneus. The great laxity of the ligaments allows the foot to become bent upon itself from the transverse tarsal joint when the foot is unsupported, but when it is placed upon the ground it assumes the form of "flat-foot."

Various deformities occur in the inferior extremity, according to the extent and localization of the paralysis. The anterior and internal muscles of the thigh are those most usually affected above the knee, and then the predominant action of the flexors of the leg on the thigh maintains the leg in a permanent condition of partial flexion (genu recurvatum), while the thigh is likewise abducted. This deformity is always associated with flexion of the thigh on the body, and talipes equino-varus (Fig. 137). All the muscles of both legs are sometimes paralyzed, so that the patient is compelled to walk on his knees, dragging his small, thin legs after him. In still more aggravated cases the muscles of both legs and thighs are permanently paralyzed, so that the small, flexible limbs dangle about like the limbs of a doll (jambe de polichinelle). Curvatures of the vertebral column generally result in infantile paralysis from the attitudes imposed by other deformities, but occasionally the curvature is caused more or less directly by the paralysis. Of the direct curvatures *lordosis* is the most frequent and important; it is caused by partial paralysis of the sacro-spinal muscles, and in order to prevent the permanent bending forwards of the body, by the predominant action of the flexors, the patient voluntarily throws the trunk backwards, so that the weight of the trunk is borne by the flexors, while the tension is taken off the partially paralyzed extensors. In this form of lordosis the pelvis is pushed forwards, and the buttocks become less prominent than in health.

The deformities of the upper extremities are much less frequent and serious than those of the lower extremities. The muscles of the

shoulder, and particularly the deltoid, are the most usual subjects of paralysis and atrophy in the upper extremity. In the severer forms of paralysis of the muscles about the shoulder-joint the humerus becomes separated from the glenoid cavity, so that a dislocation is readily produced or may occur spontaneously, the arm hangs powerless by the

FIG. 137.



CASE OF INFANTILE PARALYSIS (GENU RECURVATUM). (After LARMUTH).

side, and, to use the apt comparison of Heine, dangles about like the loose end of a flail. The distortions of the forearm and hand are not so frequent or important as to require description.

The general health of the patient is not interfered with in this disease; the organic functions are well performed and the patient may live to extreme old age.

b. Acute Atrophic Spinal Paralysis of Adults.

Acute atrophic spinal paralysis of adults is essentially the same disease as acute atrophic spinal paralysis of infants. The differences

between the clinical features of the two affections result from the facts that the brain of the adult does not respond so readily to the acute symptoms of the initial *stage* as that of the infant; that the adult organism is not so liable to fever as that of the infant; and that in the adult the ligaments and joints being fully developed offer greater resistance to the resulting deformities than do those of infants. The disease begins by pain in the back and extremities, paræsthesia, and more or less fever. In addition there may be headache, vomiting, somnolency, or even slight delirium, but convulsions have never been observed.

The paralysis is developed more or less rapidly, generally in the course of a few hours; and, as in the case of children, it is more or less widely spread, complete, and associated with more or less flaccidity of the paralyzed muscles. Reflex action is either much lowered or abolished in the paralyzed muscles, but may be retained in those which are slightly affected. Temporary weakness of the bladder may be present at first.

The initial general symptoms pass off in a few days; soon afterwards the paralytic symptoms begin to improve, and complete restitution of motor power may take place in the course of some weeks or occasionally not until the lapse of nine months, these cases forming the *temporary spinal paralysis of adults*. But the restoration of motor power is, as a rule, partial, the muscles which remain permanently paralyzed suffering a rapidly progressive atrophy as in the case of children and affording the usual evidences of the reaction of degeneration. The skin becomes loose, flabby, and inelastic, and the affected extremities are cold and cyanotic.

Paralysis of one extremity is most commonly met with in infants, but, according to Müller, paralysis of the four extremities, or of both the lower extremities, occurs more frequently in adults. Out of forty-seven cases collected by him the paralysis implicated all the extremities in twenty-two, both lower extremities in eleven, both upper extremities in three, an upper and lower extremity of the same side in one, and an upper and lower extremity of opposite sides in one. The right upper extremity was paralyzed in one, the left in two, the right inferior in two, and the left inferior in one case.

The sensory disorders which may have existed at the beginning soon subside, and the sensibility becomes normal; the sexual functions are throughout unaffected; there are no bedsores, and the general health is good.

Paralytic contractions supervene with their resulting deformities, but never attain the same degree as in children, because the joints and ligaments in adults are resistant and the long bones have attained their full development.

3. Poliomyelitis Anterior Chronica (Chronic Atrophic Spinal Paralysis).

Etiology.—The causes of this disease are exceedingly obscure; it was thought that adults were alone attacked, but it is now known that it occurs in children. The most frequent exciting causes are injuries, such as a fall on the back or hip, exposure to severe cold, damp dwellings, and alcoholic and sexual excesses.

Symptoms.—The disease usually begins with a feeling of lassitude and fatigue in walking, pain and stiffness in the loins and lower extremities, slight fever, gastric disturbance, headache, and various paræsthesiæ, such as tingling and formication of the feet and hands. After a time distinct muscular weakness is felt in one or both lower extremities, which gradually increases until complete paralysis is established. The intrinsic muscles of the foot, and the extensors of the toes and foot are the first to be affected, and subsequently the muscles of the calf, the flexors of the thigh on the trunk, the flexors and extensors of the leg on the thigh, and the extensors of the thigh on the body become successively invaded. The disease pursues its ascending course and the muscles of the back and abdomen become paralyzed, so that the patient is no longer able to sit up in bed, and expiratory acts like coughing and sneezing, as well as urination and defecation, are rendered difficult and ineffective. As the intercostal muscles are gradually attacked, it is seen that the breathing becomes more and more diaphragmatic, and at the same time the small muscles of the hand show signs of atrophy while the grasp is feeble from paralysis of the long flexors of the fingers. The flexors of the forearm, the extensors of the forearm, the supinators, and the muscles of the arm and shoulder are next attacked in succession. It is important to observe that in this disease the intercostal muscles are attacked before the diaphragm, and the intrinsic muscles of the hand and the flexors of the fingers before the extensors of the forearm, this mode of invasion being the reverse of what occurs in alcoholic and lead paralysis and other forms of multiple neuritis. But, although the disease usually begins in the lower extremities and pursues an ascending march, it sometimes begins in the upper extremities and pursues a descending course, or rather pursues an ascending and a descending course, inasmuch as the bulbar nuclei are liable to be invaded as well as the motor cells of the dorsal and lumbar regions. Although the two lower and the two upper extremities are usually affected, the paralysis is frequently more pronounced on one side of the body than on the other. The paralyzed muscles are soft and flaccid, tension is not pro-

voked in them by passive movements of the affected extremities, and they undergo rapid atrophy. The calves of the legs become converted into loose and flabby sacs, the muscles of the thighs and gluteal regions grow thin and soft, and the bones of the lower extremities may ultimately be felt immediately underlying the skin, while scarcely a trace of the muscular masses is left. The patient now lies on his back with the various segments of the lower extremities extended upon one another; the upper extremities lie immovable by his side in any position in which they may be placed; the arms and forearms become greatly emaciated from disappearance of their muscular masses; the thenar and hypothenar eminences are flattened; and the hands assume characteristically distorted positions.

Fibrillary contractions may be present in the early stages of the atrophy, but these soon disappear. Reflex action is lost at an early period of the disease, and the tendon-reactions are likewise soon abolished. The electrical reactions of the paralyzed nerves and muscles are, on the whole, the same as those met with in acute atrophic spinal paralysis, being only modified to some extent in correspondence with the slow development of the former disease as compared with the latter. A case of this disease came under my observation in which the faradic contractility of the paralyzed nerves and muscles was at first much increased during the early stage, but it was completely lost after a few weeks, and then the galvanic reactions manifested the qualitative changes which characterize the "reaction of degeneration."

The *sensory disorders* consist of tingling and formication, but the cutaneous and muscular sensibilities are found to be normal on objective examination.

The skin of the paralyzed limbs may become of a blue color and the surface may be cold, while the lower extremities may be œdematous, but there are no bedsores nor cutaneous eruptions, the functions of the bladder, rectum, and sexual organs remain unaffected, and the general health is satisfactory. During the course of the disease the small joints of the hand are liable to become swollen, red, and painful, and the patient may be covered with an abundant sour-smelling perspiration like that of acute rheumatism. The acute symptoms subside, but the joints are apt to remain, to some extent, permanently deformed like those of rheumatic arthritis. The course of the disease is variable, the paralysis may supervene suddenly or be preceded for some time by premonitory symptoms, and when the lower extremities are the first to be attacked, the upper extremities may be implicated in a few weeks or not until the lapse of months or years. In fatal cases death is usually caused by implication of the bulbar nuclei and respiratory paralysis, but

occasionally it results from exhaustion. Very frequently the disease becomes arrested in its upward course, and, after a stationary period of variable duration, improvement takes place in the muscles which were the last to be invaded. During recovery the electrical reactions return slowly and gradually to the normal formula. Recovery from this disease is not often complete. Groups of muscles, especially the anterior and external muscles of the legs, remain paralyzed and atrophied, and the patient is partially disabled for life.

4. Periependymal Myelitis (Syringomyelia, Hydromyelia).

Etiology.—The causes of this disease are the same as for other forms of myelitis.

Symptoms.—This affection begins somewhat abruptly by paralysis of some muscular groups, the patient finding suddenly that he is unable to move the fingers, hands, or more rarely an entire limb. The muscles lose their faradic contractility, and become atrophied at an early period of the disease, and the affected extremities assume distorted positions. In the early stage of the disease the muscles may manifest fibrillary contractions, and occasionally involuntary movements of a limb or of the trunk have been observed. Patients often complain of vague pains along the vertebral column, but in most cases there are no other sensory disorders. In some cases, however, the patient suffers from numbness and tingling of the lower extremities, and there may be an extensively diffused anæsthesia, as in a case recorded by Schüppel, which was frequently exhibited during life in Niemeyer's clinic. An ataxic gait has never been observed, and the bladder and rectum always remain unaffected.

The course of the disease is slow, and it may be temporarily arrested, or may even regress, and then the atrophied muscles gradually regain their motor power and volume, but relapses are liable to occur. The muscles of the lower extremity most frequently affected are the flexors of the foot on the leg, and of the thigh upon the pelvis; and of the upper extremities, are the extensors of the fingers and of the hand; then the small muscles of the hand, and lastly the flexors of the forearm, and the muscles of the arm and shoulder. The disease pursues an ascending course when the lower extremities are the first to be paralyzed, and both an ascending and a descending course when the upper extremities are attacked first, and bulbar paralysis is ultimately liable to supervene, and to cause death. In some cases the paralytic symptoms pursue a rapidly ascending course, and the disease may then be mistaken for an acute ascending paralysis.

5. Progressive Muscular Atrophy.

Etiology.—Hereditary predisposition is generally supposed to play a very important part in the production of progressive muscular atrophy, but it is most probable that the cases in which the disease appears in numerous members of the same family really belong to the group which Erb has described under the name of the "juvenile form of progressive muscular atrophy," a group which is more allied to pseudo-hypertrophic paralysis than to genuine progressive muscular atrophy, and until the two groups of cases are separated from one another by a rigid analysis, we are not in a position to estimate the part which heredity plays in the production of progressive muscular atrophy. The disease attacks males more frequently than females, the proportion, according to Friedreich, being one hundred and forty-three males to thirty-three females. The disproportion between the sexes probably depends on men being more exposed, than women, to the exciting causes of the disease.

Progressive muscular atrophy is often developed during convalescence from acute diseases, such as typhus fever, measles, acute rheumatism, and cholera, and it occasionally begins in women soon after parturition.

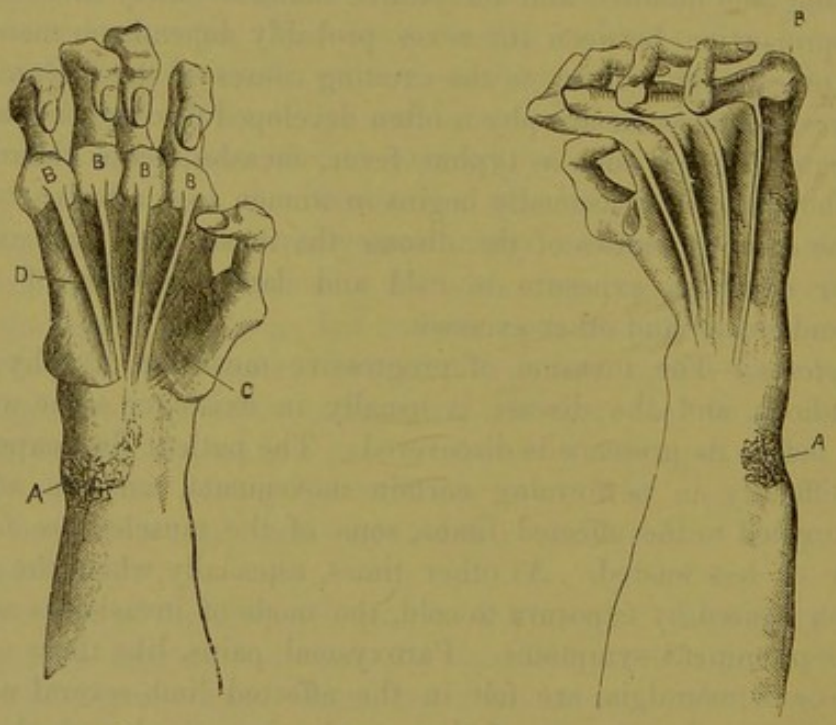
Of the exciting causes of the disease the most usual are excessive muscular exertion, exposure to cold and damp, injuries of various kinds, and sexual and other excesses.

Symptoms.—The invasion of progressive muscular atrophy is slow and insidious, and the disease is usually in existence some weeks or months before its presence is discovered. The patient first experiences some difficulty in performing certain movements, and on attention being directed to the affected limbs, some of the muscles are found to be more or less wasted. At other times, especially when the disease has been caused by exposure to cold, the mode of invasion is attended by more prominent symptoms. Paroxysmal pains, like those of rheumatism or of neuralgia, are felt in the affected limb several weeks or months before the atrophy of the muscles is noticed, and when once the atrophy begins in these cases it proceeds more rapidly, and becomes more generalized than in the painless variety.

The disease usually begins in one of the upper extremities, more commonly in the right, and the first muscles to be attacked are either those of the thenar or hypothenar eminences, the interossei, or the muscles about the shoulder. It will be shown hereafter that the cases in which the atrophy begins in the muscles of the lower extremities and the lumbar muscles are most probably not examples of true progressive paralysis. During the progress of the disease certain muscles

or groups of muscles are attacked while their neighbors are spared, and the healthy and less atrophied muscles overcome the resistance of the more diseased, and thus produce distortions, which are rendered more characteristic by the wasting of the muscles. The disappearance of the interossei is shown by the deep furrows which appear between the metacarpal bones; the thenar and hypothenar eminences are flattened, and the disappearance of the muscles of the palm brings into view the diverging flexor tendons which are stretched between the wrists and the bulging bases of the fingers. The deformity produced by paralysis of the interossei gives to the hand the appearance of the talons of a bird of prey, and consequently it has been called the claw-shaped hand, or *main en griffe* (Fig. 138). This deformity, however, is not peculiar to

FIG. 138.



1. MAIN EN GRIFFE. 2.

(1) HAND, PALMAR SURFACE. (2) DORSAL SURFACE. (After DUCHENNE.)

A, Wound of the ulnar nerve; B, Ends of the metacarpal bones; D, Tendons of the flexor sublimis; C, Muscles of the ball of the thumb.

progressive muscular atrophy, inasmuch as it may be caused by injury of the ulnar and median nerves.

Atrophy and paralysis of the opponens and abductor pollicis cause the thumb to be extended and abducted when the flexors of the hands and fingers are affected, the anterior and internal aspects of the forearm are flattened, and when the extensors of the hands and fingers are

attacked the posterior aspect of the forearm presents a thin and wasted appearance. Atrophy of the supinator longus gives rise to a flattening of the external border of the forearm. When the disease begins in the small muscles of the hand and ascends gradually towards the trunk, the supinator longus is often the only muscle of the forearm which retains its plumpness, but when the disease begins in the muscles of the shoulder and extends to the periphery, the external border of the forearm becomes flattened at an early period from implication of the supinators.

When the muscles of the shoulders are affected the arms may hang by the side and rather in front of the patient and seem as if they were merely attached to him by strings and did not belong to him; the natural rounded configuration of the shoulder is replaced by a hollow in which the palm of the hand may be lodged under the projecting acromial and coracoid processes of the scapula, which stand out in relief. The biceps and the other muscles of the arm may also waste, so that the limb loses its roundness and becomes flattened, and the humerus appears to be surrounded merely by a bag of skin. When the abdominal muscles are affected, the lumbar curve is greatly exaggerated by the unopposed action of the erectors of the spine, and the abdomen is loose and protruding, but the thorax is held well forwards, so that a plumb-line let drop from the most prominent of the spinous processes of the vertebra will pass well within the sacrum. When the atrophy is unequally distributed on both sides of the body, scoliotic or kyphotic bending of the vertebral column is produced. When the erectors of the spine and the extensors of the thigh are simultaneously affected the resulting deformities and the gait are similar to those observed in pseudo-hypertrophic paralysis and need not be described here.

Implication of the muscles of the lower extremities is rare except in the later stages of the disease, but when they are attacked the various forms of paralytic club-foot may appear.

The accessory respiratory muscles, such as the pectoralis major, serratus magnus, and trapezius, are frequently invaded; and although the wasting and loss of power of these muscles do not directly endanger life, yet they may do so indirectly, inasmuch as a slight intercurrent attack of bronchitis may lead to asphyxia from the inability of the patient to make a strong expiratory effort to clear the tubes of mucus. In the later stages of the affection the diaphragm and the intercostal muscles become attacked, expectoration fails, mucus collects in the tubes, and the patient dies asphyxiated. In other cases a progressive bulbar paralysis is superadded to the atrophy of the other muscles, and the

disease is then generally fatal from arrest of respiration caused by paralysis of the centre in the medulla. The loss of muscular paralysis keeps pace with the atrophy, and is, as a rule, directly in proportion to the degree of the latter, and so long as any muscular fibres are left, they can be made to contract by voluntary effort. For a long time, indeed, the various movements are capable of being performed, although with diminished power, and it is only in the last stage of the disease that complete immobility of the limb is produced. When once a muscle is attacked, it wastes, as a rule, in a perfectly uniform manner, but in some cases a portion only of the length of the muscle undergoes atrophy, while the remainder maintains its volume, and to a considerable degree its power also.

The *fibrillary contractions* of the affected muscles, consisting of vibratory tremors or quivering of the fibres, are frequently observed throughout the whole active stage of the disease. They occur spontaneously, but may be provoked by gently tapping the surface, by exposing to air parts which are usually covered, by electrical excitation, and by active or passive movements of the affected muscles. These fibrillary contractions are sometimes the earliest symptom of a fresh advance of the disease, and they disappear altogether when the atrophy has reached an extreme degree, or when its progress is arrested. Tonic or clonic contractions of entire muscles or of groups of muscles may occasionally occur, which are accompanied by an intense pain like that of cramp at the calf. During the stage of active fibrillary contractions the idio-muscular contractility may be so much increased that the slightest tap on the tendons, fasciæ, neighboring bones, or on the bellies of the muscles themselves occasions widely diffused contractions, such as are frequently observed in advanced cases of acute phthisis. When the muscles of the upper extremity, for instance, are undergoing active atrophy a slight tap on the lower end of the radius may cause almost the whole of the muscles of the forearm and arm, as well as the pectoral muscles, to enter into contraction, and the forearm may be jerked upwards just as occurs in cases of spasmodic paralysis; and when the atrophy invades the muscles of the lower extremities the knee-jerk may be so lively as to give rise to considerable difficulty in diagnosis.

The *cutaneous reflex actions* are often exaggerated during the active stage of atrophy, and this renders still greater the similarity of the affection to a spasmodic paralysis. As the atrophy advances, the cutaneous reflexes and tendon-reactions become diminished and finally disappear.

The *electrical reactions* of the paralyzed muscles may also be at first

increased. During the active stage of atrophy feeble electrical currents either passed through the nerve or the muscle induce very active contractions, although their energy is diminished according to the degree of atrophy. As the atrophy advances the strength of current required to produce a minimum contraction becomes greater and greater, and after a time the qualitative changes in the galvanic reactions which characterize the "reaction of degeneration" are observed.

The *sensory disorders* of progressive muscular atrophy are of subordinate importance. In some cases the muscular disease is preceded by paroxysms of pain in the affected parts, which sometimes radiate along the branches of certain nerves, and at other times appear to have their origin in the sensory nerves of the muscles, the pain being then aggravated by compression of the affected muscles or by passive movements of them. A moderate degree of anæsthesia may be present in the hands and tips of the fingers in the later stages of the disease, while complete analgesia of circumscribed areas of the skin is not uncommon, and in some cases the area of analgesia is much more extensive than that of the atrophied muscles. The farado-cutaneous sensibility may also be diminished, and the patient often complains of sensations of cold, numbness, formication, and other paræsthesiæ in the hands and feet.

The *vaso-motor* disturbances consist of a local spasm of the blood-vessels giving rise to coldness and pallor of the affected parts, which is apt to be followed by relaxation of the vessels and consequent warmth and redness. In the early stages of the disease the temperature of the affected extremities is increased, according to Frommann, by 0.2° or 0.3° C., while in the later stages it may sink 3° or 4° C. below the normal. In the later stages of the disease the patient is sometimes covered with a profuse sweat (hyperidrosis) which is probably due to vaso-motor paralysis.

Trophic disorders may sometimes occur in addition to the muscular atrophy. The skin is sometimes the subject of atrophy, but never to a very pronounced degree. In the early stages of the disease the patient may be attacked with painful swellings of the phalangeal joints (arthritis nodosa), which are most probably related to the arthropathies of tabes dorsalis.

Oculo-pupillary symptoms, consisting of paralytic myosis and diminution of the palpebral fissures, have occasionally been observed; but it is surprising how rarely these phenomena are met with when it is considered how frequently the lesion is localized in the spinal cord on a level with the cilio-spinal region. In the early stage of progressive muscular atrophy the patient may complain of chills, and there may be a continuous, though slight, increase of temperature, which lasts for days

or months. This febrile condition may probably be due at times to an accompanying arthritis nodosa. In the later stages of the disease transitory or permanent elevations of temperature may occur, which are, perhaps, due to such complications as diseases of the lungs or acute bedsores. No constant changes have been found in the urine. The course of progressive muscular atrophy is essentially chronic, but its duration is very variable and uncertain. In twenty-eight cases analyzed by Dr. Roberts the mean duration was thirty-eight months; of these four recovered and their mean duration was fourteen months; the disease was arrested in thirteen cases with a mean duration of twenty-seven months, and the remaining eleven cases died with a mean duration of upwards of five years. The disease may progress steadily until nearly all the voluntary muscles are implicated, and the unfortunate patient is reduced to such utter helplessness that he is quite unable to feed himself or turn in bed. The advance of the disease is, however, seldom continuous, even when it is progressive. Its course is often interrupted by remissions which may last for weeks, months, or even years, or its progress may be permanently arrested after some groups of muscles have been irretrievably destroyed. In a few cases the atrophied muscles may be restored by treatment to their former volume.

6. Primary Labio-glosso-laryngeal Paralysis (Chronic Progressive Bulbar Paralysis (Wachsmuth)).

Etiology.—It does not appear that heredity exercises much influence in the production of labio-glosso-laryngeal paralysis. The disease occurs most frequently between the ages of forty and seventy years, although it occasionally attacks younger people. The cases described as occurring in children are probably examples of insular sclerosis in which the first diseased patches are localized on a level with the bulbar nuclei and nerves. The disease attacks men twice as frequently as women, and all ranks of society appear to be equally liable to it.

The exciting causes of progressive bulbar paralysis are exposure to cold, blows on the back of the neck, emotional excitement, excessive mental activity, straining of the affected muscles in singing and speaking, and bad and insufficient food. Syphilis is a frequent cause of the disease.

Symptoms.—The disease is generally preceded by slight premonitory symptoms, such as pain in the head and back of the neck, slight dizziness, and great diminution or complete loss of the reflex irritability of the larynx, œsophagus, and pharynx.

The symptoms of chronic progressive bulbar paralysis begin stealthily

and creep on gradually. A slight affection of speech is usually the first symptom to attract attention, articulation becomes indistinct, and the pronunciation of certain letters presents special difficulty, while the tongue and lips are soon fatigued, so that prolonged reading aloud or speaking is impossible. The articulatory difficulties are followed by a gradual weakness of the lips, and of the palate. The expression of the face is altered, the voice becomes nasal, and the muscles of mastication and deglutition are so readily fatigued that the patient is soon compelled to eat only pulpy food, and is unable to swallow much at a meal.

The initial debility and fatigue of the muscles may extend over a period of years before the stage of distinct paralysis is reached, but when once decided paralysis is established the disease assumes a more progressive character, and advances steadily and surely to a fatal termination.

When the tongue becomes more or less paralyzed the patient experiences an ever-increasing difficulty in pronouncing the dental and guttural sounds, and inasmuch as the vowel *i* requires the greatest raising of the tongue for its production, its pronunciation is the first to suffer, and then the pronunciation of the consonants *r*, *s*, *l*, *k*, *g*, *t*, and lastly *d* and *n* become difficult, imperfect, and finally impossible. After a time the patient is unable to effect the coarser and less complicated lingual movements. He may at first be able to protrude the tongue, but not to raise the tip towards the hard palate or towards the nose after protrusion, while inability to move the tip laterally indicates a still greater degree of paralysis. As the paralysis increases the tongue cannot be lengthened into a point or rolled into the form of a tube, and ultimately the patient is unable to protrude the organ, which now lies behind the lower row of teeth completely helpless, and motionless, or maintained in constant vibration by fibrillary tremors. The tongue may maintain its normal aspect, or it becomes large and flabby, or more frequently it is sodden, grooved longitudinally, wrinkled, and shrunken, while simultaneous atrophy of the papillæ give a glazed appearance to the surface.

Deglutition is rendered difficult in the early stages of the disease by the weakness of the tongue alone. The patient experiences a difficulty in collecting the food so as to form it into a bolus, and in pressing it back against the soft palate and into the pharynx. In order to overcome this difficulty the patient, after chewing his food well, takes a drink and throws his head backwards, while at other times he assists the imperfect movements of his tongue with his fingers, using them to extract the food which has lodged between the teeth and cheeks, and to

push the bolus to the back of the tongue till it is caught by the reflex movements of the pharyngeal muscles.

The glottis is not completely closed during deglutition, even at this early stage, because the root of the tongue cannot be sufficiently elevated during the second stage of the act in order to allow the bolus to glide over the depressed epiglottis. The closure of the glottis is also rendered still more imperfect by the presence of paralysis of the muscles which pass from the inferior maxilla to the hyoid bone, a defect which prevents the larynx from being raised as in health during the second stage of deglutition, and consequently the glottis fails to be closely applied to the epiglottis. The imperfect closure of the glottis causes food and fluids to find their way into the trachea, and these give rise to distressing paroxysms of cough and dyspnœa. The saliva cannot be swallowed, and accumulates in the mouth, and, owing to the paralysis of the orbicularis oris, flows from it in a continuous stream.

The *facial muscles* are implicated soon after, or sometimes before the muscles of the tongue, the orbicularis oris being the first to suffer. With the increasing weakness of the orbicular muscle the patient becomes unable to whistle, blow, compress his lips, or kiss; he experiences difficulty in pronouncing the vowels *o* and *u*, and with the advance of the paralysis the labial consonants *p*, *f*, *b*, and *m* become increasingly difficult to articulate.

Paralysis of the palate renders the formation of the explosive labial consonants still more difficult, because the current of air necessary to force the lips suddenly asunder escapes through the nose, and the consonants *p* and *b* are consequently turned into *me* and *ne*. If the patient's nose be closed, these letters are better pronounced. Paralysis of the palate also gives a nasal resonance to the voice, and permits food and fluids to escape readily through the nose during efforts at deglutition. When the muscles of the tongue, lips, and palate are simultaneously paralyzed, speech becomes more and more indistinct, and the patient can only give utterance to inarticulate and grunting sounds, but the vowel *a*, being independent of the articulatory movements, can still be pronounced.

The other facial muscles most liable to be implicated are the quadratus and levator menti, the muscles of the palpebral and nasal regions are never affected, and even the elevators of the superior lips and the buccinators are only occasionally paralyzed. The paralyzed muscles are almost always distinctly atrophied, and consequently the lips look thin, sharp-edged, and furrowed, and fibrillary contractions are not infrequently observed in them. The patient now presents a very striking and characteristic appearance. The lower lip hangs loose and pendulous, the mouth is somewhat increased in breadth and cannot be

closed, and the naso-labial folds become deepened and give to the patient a lachrymose expression. During states of emotional excitement the lower part of the face remains comparatively motionless, and contrasts strongly with the vivacious movements of the upper half of the face, and with the brightness and activity of the eyes.

The saliva now flows from the mouth in a continuous stream and causes much annoyance to the patient, inasmuch as it soaks through the pillow at night, and requires to be constantly wiped from the lips with a handkerchief during the day. The saliva is sometimes unchanged in quality, while at other times it becomes so viscid that it may be drawn out of the mouth in long ropes. It appears to be generally secreted in normal quantity, but sometimes it is increased to six or eight times the quantity secreted in healthy persons.

Mastication is indirectly impaired from paralysis of the tongue and facial muscles, but after a time it becomes still further interfered with by direct implication of the masticatory muscles. The pterygoid muscles are the first of the masticatory muscles to be attacked, and with paralysis of these the power of effecting the lateral movements of the lower jaw is lost, and with the progressive feebleness of the remaining muscles of mastication the power of chewing the food becomes increasingly difficult, feeble, and finally impossible.

The *pharyngeal muscles* are now invaded and portions of food lodging in the pharynx increase the risk of foreign particles entering the larynx, while at other times the whole bolus sets impacted on a level with the glottis and causes imminent danger of suffocation.

The *constrictors of the glottis* are paralyzed soon after or simultaneously with the pharyngeal muscles, and the danger of swallowing either solids or fluids is now much intensified, and solid particles freely entering the trachea not only cause distressing paroxysms of cough and dyspnœa, but are also apt to pass into the bronchi and give rise to a fatal pneumonia.

The *œsophagus* becomes ultimately paralyzed if the patient survive so long; the power of deglutition is now completely lost, and the patient must starve unless he is fed with the stomach pump.

The *laryngeal muscles* are sometimes found paralyzed: the laryngoscope reveals paresis or paralysis of the vocal cords, and the voice becomes hoarse and feeble, until finally there is complete aphonia.

The *circulation* is variously affected according to the stage of the disease. There is no trustworthy record of retardation of the pulse which could with probability be referred to irritation of the vagus, but a pulse rising before death to from 130 to 150 per minute, or even higher, has been frequently recorded, and is probably caused by paralysis

of the vagus. In the terminal period of the disease patients often suffer from fainting fits, accompanied by great anxiety and a sensation of impending death, and, indeed, death may result from an attack of syncope. These phenomena are probably caused by disease of the cardiac centres of innervation.

When the *respiratory mechanism* is affected a fatal termination is near. The respiratory movements become feeble, and owing to the implication of the spinal accessory nerves the auxiliary muscles of respiration are paralyzed, and superior thoracic breathing is impossible. The inefficiency of the respiratory movements renders the breathing shallow, and all attempts at coughing or blowing the nose are weak and powerless. After a time the pneumogastric nuclei are invaded, and spontaneous paroxysms of dyspnœa with a tendency to syncope supervene. The attacks of dyspnœa become more and more frequent as the disease advances, while the breathing power becomes feebler and feebler until ultimately the patient dies from asphyxia. But the patient often dies before this advanced period of the disease is reached, the fatal issue being generally caused by an attack of bronchial catarrh or of pneumonia.

Atrophy of the paralyzed muscles is usually most marked in the tongue and lips, these parts being likewise kept in constant movement by fibrillary contractions. The apparent loss of volume and the degree of paralysis do not always run a parallel course, and the tongue sometimes retains its normal volume while completely paralyzed, but a subsequent microscopical examination will show that the muscular fibres have undergone extensive degeneration.

The *electrical excitability* is said by most authors not to undergo any noteworthy changes, but Erb states that he has met with the most marked "reaction of degeneration" on direct irritation of the muscles of the chin, lips, and tongue. The electrical irritability of the nerves was normal or but slightly diminished.

The *sensibility* generally remains unaffected throughout the whole course of the disease. Buzzing in the ears and deafness have occasionally been present, and in some cases various paræsthesiæ, or even a considerable degree of anæsthesia of one or both sides of the face, loss of common sensation on the tongue, and pain in the occipital and upper cervical regions have been observed.

The *intelligence* remains quite clear to the last, but the temper is somewhat excitable, and patients often manifest an inclination to laugh or weep on slight provocation.

Reflex irritability is often much diminished or lost in the tongue, soft palate, pharynx, and even in the larynx before the appearance of any

other symptom, but it is sometimes retained in these parts until a late period of the disease.

Vaso-motor disturbances have not been recorded, and there is no fever during the whole course of the disease.

General nutritive disorders occur sooner or later in the course of this disease, owing in great part to the insufficient quantity of food which the patient is able to take. The patient becomes after a time greatly emaciated, and, unable to get up, he sits in bed with the upper part of the body propped up, and with the head resting on pillows and inclined to one side in order to let the saliva flow out of the mouth. When this condition is reached death soon supervenes, either from a paroxysm of dyspnœa, or suddenly and quietly from arrest of the heart's action.

The *course* of this form of bulbar paralysis is always slow and chronic, but surely progressive. There is seldom a remission of long duration and recovery has never been observed when the diagnosis of the primary disease was beyond question. The disease is usually fatal in from one to five years.

Complications.—Progressive muscular atrophy is the most important and frequent complication of bulbar paralysis, the latter disease being sometimes the primary affection, and at other times a terminal phenomenon supervening in the course of the former. The two affections are, indeed, essentially the same disease, both as regards the clinical symptoms and the anatomical changes formed after death.

Amyotrophic lateral sclerosis is another important complication of progressive bulbar paralysis. In the cases which concern us at present the phenomena of bulbar paralysis are primary, but in the course of the disease the lower extremities become the subjects of a gradually increasing paralysis which is characterized by the muscular tension, and exaggerated tendon-reactions which are held to indicate disease of the lateral column of the cord.

7. Ophthalmoplegia Externa vel Progressiva.

Etiology.—The two known causes of the disease are syphilis and rheumatism.

Symptoms.—The first symptom to attract attention is usually a drooping of the eyelids, which gives to the patient a peculiar sleepy appearance. Soon afterwards all the muscles of the eyeballs manifest signs of weakness, the affection is usually bilateral, and every possible combination of paralysis may occur, but the muscles are always attacked in groups and not singly. The pupils are in a medium state of dilata-

tion, and their reflex contraction to light is either sluggish or lost. The condition of accommodation has not always been tested, but in some cases it was found normal. White atrophy of the optic disks, with blindness, was present in one-third of the cases reported by Hutchinson. In some cases the fifth nerves, and in others the facial nerves were involved in the disease, while in one case reported by Hutchinson the palate was affected and smell was lost. In some of the recorded cases the patellar tendon-reaction was absent, and other symptoms indicative of locomotor ataxia were present. It seems, indeed, likely that a considerable number of the reported cases were examples of locomotor ataxia in which the ocular troubles were unusually well marked.

V. MYOPATHIC ATROPHIC PARALYSES.

Pseudo-hypertrophic Paralysis and Erb's Juvenile Form of Progressive Muscular Atrophy.

Etiology.—Pseudo-hypertrophic paralysis was supposed to begin almost always in infancy, but if we include Erb's juvenile form of muscular atrophy, it will be seen that adults are frequently attacked, although in these cases the symptoms make their appearance between five and thirteen years of age. The disease is much more common in boys than in girls; out of two hundred and twenty cases collected by Gowers one hundred and ninety were boys and thirty girls. Hereditary predisposition to the disease can often be traced. Two children in the same family are often attacked, and Meryon met with a family in which eight children were the subjects of the disease, while other equally striking examples have been reported by others. It is very probable that all the cases of supposed progressive muscular atrophy in which numerous members of the same family were attacked, such as the cases reported by Hemptenmacher, Friedreich, Hammond, and Naunyn, really belong to Erb's juvenile paralysis, and will therefore have to be transferred to this group. A remarkable circumstance about the heredity of this disease is that it is mainly confined to the male sex, yet the descent, so far as is known, is always through the mother's side. The disease is not, as a rule, transmitted directly from parent to offspring, inasmuch as the majority of its victims are attacked at an early age, and they do not, therefore, become parents. For the same reason the disease cannot be regarded as an example of atonism. It must, therefore, be inferred that a certain predisposition is transmitted which, with the concurrence of other unfavorable circumstances, such as an eruptive fever, develops the disease.

The exciting causes of the disease are not well known. Exposure to cold and damp appears to be occasionally the determining cause, while at other times it has followed an eruptive fever, variola, or measles, and several cases have been ushered in by convulsions.

Symptoms.—When pseudo-hypertrophic paralysis begins in infancy the parents do not notice that anything is wrong until the child arrives at the age when he ought to begin to walk. At this period it is noticed that when the child is placed on his feet he does not instinctively move his legs to walk, but they double helplessly under him, or the child may have begun to walk, but it is observed that he is soon fatigued, and that he is unable to stand steadily or walk without stumbling. The parents are not readily alarmed at the inability of the child to walk, because the lower limbs appear to be unusually well developed.

The appearance of muscular strength is caused by an increase in volume of some of the muscles, but this increase is due, as we shall hereafter see, to morbid changes occurring chiefly in the connective tissue of the muscle, and which are always attended by a diminution of its power. The apparent hypertrophy of the muscles generally begins by enlargement of one calf, the other soon becoming affected. The gluteal muscles are soon invaded, and then the disease successively attacks the lumbo-spinal muscles, the muscles of the thigh, trunk, shoulders, and upper arms, while the muscles of the neck, face, tongue, forearms, or hands may occasionally be attacked, but only, as a rule, at a late period of the disease. The affected muscles may attain an enormous volume, and stand out so prominently under the skin that Duchenne uses the term "hernial protrusions" to describe their appearance. The muscles feel hard and resisting to the touch, so that the whole appearance of the patient often suggests the idea of Herculean strength instead of the great feebleness which really exists. But even in the midst of all this apparent development of muscular power there are not wanting visible indications of the real nature of the malady, inasmuch as some of the muscles are always found atrophied and their wasted condition contrasts strongly with the excessive size of the others. In the majority of cases the gastrocnemii and the gluteal muscles are increased in size, while those of the thigh often undergo some degree of atrophy, and consequently the slender thighs afford a marked contrast to the enlarged calves and buttocks. In the upper part of the body the atrophy frequently predominates. The deltoids are usually enlarged, but the remaining muscles of the arm and the pectoral muscles undergo atrophy, while those of the forearm and hand are often spared until a late period of the disease. Of the muscles which move the shoulder the rhomboids and the serrati are the most liable to be attacked, and

these are generally atrophied instead of being enlarged. In a large majority of cases, therefore, the upper part of the body is contrasted by its slenderness with the unusual development of the lower extremities, and perhaps the greatest contrast is afforded by the thin arm, which often consists of scarcely anything more than the humerus surrounded by skin, and the normal condition of the forearm on the one hand, with the prominence of the enlarged deltoid on the other.

The relative distribution of the hypertrophied and atrophied muscles just described is that usually met with in the cases named by Erb "the juvenile form of progressive muscular atrophy," but I can see no essential difference between these cases and those in which the muscular hypertrophy is more widely distributed. Pseudo-hypertrophy of muscles does not, indeed, appear to be a necessary part of the diseases which may be grouped together as myopathic atrophic paralyses. In the cases described by Leyden under the name of "hereditary muscular atrophy," the slight degree of increase of volume observed in some of the muscles is quite subordinate to the widely diffused atrophy of others. Cases have been described by Zimmerlin which appear to belong to the myopathic atrophic paralyses, and in which the muscles of the shoulders and arms were atrophied without any admixture of pseudo-hypertrophy, and cases have been described by Landouzy and Déjerine which must also apparently be included in this category, and in which the symptoms began in infancy by atrophy of the muscles of the face, the muscles of the shoulders and arms being invaded about the age of eleven years. In short, the group of myopathic atrophic paralyses presents several varieties, but we shall at present proceed with the more usual form of the disease as represented by the affection known under the name of pseudo-hypertrophic paralysis.

The relative degree of paralysis of groups of muscles gives rise as usual to certain deformities, and one of the most notable of these occurs in the foot. The gastrocnemius muscle undergoes progressive shortening, partly perhaps owing to the internal changes it is undergoing, and partly owing to the increasing paralysis of the anterior muscles of the foot. But whatever may be the cause, the result of this shortening is that the patient experiences difficulty in bringing the heel to the ground, and as the disease advances a permanent condition of talipes equinus or equino-varus is established. The foot also becomes more hollow and the plantar arch increased, while paralysis of the interossei causes the first phalanges to be maintained in a state of exaggerated extension on the metatarsal bones, and the distal phalanges to be flexed, so that the toes assume the peculiar claw-like appearance which Duchenne has called *griffe des orteils*. A remarkable curvature of the spine in the

lumbo-sacral region, named by Duchenne *lordosis* or *saddle-back*, is one of the most constant symptoms of the disease. This deformity is not seen until the patient stands or walks; the shoulders and the upper part of the vertebral column are then carried backwards, so that a plumb-line let fall from the most prominent spinous process of the vertebral column falls behind the sacrum. This deformity is produced by comparative feebleness of the extensors of the pelvis and the erector muscles of the spine. Weakness of the extensors allows the pelvis, and with it the lower vertebræ, to incline forwards when the patient assumes the erect posture, and a compensatory backward inclination of the dorsal spine is rendered necessary in order to keep the centre of gravity in the normal position. The posterior displacement of the upper part of the body also causes the line of gravity to fall behind the trochanters of the femur and takes off all strain from the enfeebled erector muscles of the spine and the gluteal muscles. The attitude of the patient on assuming the erect posture is quite characteristic of the disease. The feet are kept widely apart, and the patient cannot approximate them without risk of falling. In the early stage of the disease the heels are brought to the ground, but after a time the patient has to balance himself upon his toes, and at the same time the dorsal curve becomes very pronounced, and in consequence of these deformities the power of the patient to maintain the erect posture becomes very precarious and the slightest push suffices to throw him down.

The gait of the patient is no less characteristic than his attitude in the erect posture. We have seen that the feet are held apart, and in walking the body is inclined first to one side then to the other, so that the gait resembles the waddling of a duck. When the feet are kept widely apart the centre of gravity must be carried at each step well over to the side of the active leg, in order that the line of gravity may pass through the centre of the arch of the foot planted on the ground. Duchenne thought that the oscillation of the body in walking was an instinctive movement on the part of the patient in order to avoid putting a strain upon the paralyzed gluteus medius. But in a case of muscular atrophy observed by myself the gluteus medius was completely paralyzed on each side, yet instead of the waddling gait so characteristic of pseudo-hypertrophic paralysis, the head and body were moved forwards during locomotion almost in a straight line, and without the lateral inclinations being at all equal to those observed in healthy persons. From an examination of several cases of this disease it appears to me that the lateral inclinations, instead of being caused by paralysis of the gluteus medius, are mainly effected by alternate contractions of these muscles, and are rendered necessary partly by the legs being held

widely apart, and partly by the inability of the patient to clear the toes of the advancing foot from the ground by producing dorsal flexion of the foot. As the disease advances the patient, as we have seen, becomes unable to bring the heel to the ground, and at each step the body must be delicately balanced in order that the line of gravity may pass through or a little behind the ball of the active foot, and the slightest displacement of the centre of gravity will cause the patient to fall. It is, therefore, necessary that at each step the body should be inclined well over to the side of the active leg, and the patient aids himself in maintaining the centre of gravity vertically above the ball of the foot on the ground by moving his arms about like a rope dancer.

The manner in which the patient attains the erect posture is equally characteristic. When he is laid down, or falls, he drags himself up with the aid of his arms if a chair or other article of furniture is sufficiently near for him to lay hold of. But when he has to get up without extraneous aid, the body is first raised on the hands and feet. In this attitude the feet are planted on the ground, the different segments of the lower extremities are slightly flexed upon one another, the body is flexed on the lower extremities, and more or less horizontal, the head is directed downwards, and the tips of the fingers of both hands rest on the ground in front of the toes. The patient next raises his hand, say the left, and places it above the left knee. The body is now drawn over to the opposite side so that its weight rests mainly on the right leg, and by one vigorous push of the left arm the left knee is thrust backwards, and the leg and thigh are thus extended upon one another, while the body is at the same time thrust upwards. Simultaneously with this action the weight of the body is transferred from the right to the left leg, and the right hand is elevated and planted above the knee on the corresponding side, and a backward push on that knee causes the different segments of that limb also to become extended upon one another. The patient has now attained the second posture, and in this position his feet are planted on the ground, the lower extremities are directed upwards or backwards, and the body is inclined upwards while the arms prop up the trunk by passing from it to the knees where the hands are placed. The fact that the weight of the upper part of the trunk is directed through the arms to the knees redistributes the force in such a way that the knees are pushed backwards, and thus the thighs and legs tend to become extended upon one another, the heels are forced downwards, and so the patient is fixed firmly to the ground, and the trunk is maintained in its upward and forward attitude without any strain being placed upon the enfeebled extensors of the body. The next great effort of the patient is directed to attain the

erect posture, and in accomplishing this object it is said that he thrusts his body upwards by gradually climbing up his thighs. This description is very graphic, but it is only partially accurate. The movement by which the pelvis is elevated upon the thighs is, indeed, a very complicated one, and is effected not by simple extension, but by a kind of screw movement.

The patient experiences great difficulty in getting up steps, owing to the feebleness of the gluteus maximus. He lays hold of the railing with one hand, say the right, and drags his body upwards at each step. The right arm is, however, assisted by the left. The left hand is planted above the knee upon the same side, and each time the left leg is raised a step the body is thrust upwards by the various segments of the left arm being extended upon one another. The disease now becomes stationary for two or three years, and sometimes for a much longer period, and as the general health is good and the muscular development apparently very powerful, the parents cannot believe that the affection is incurable. This illusion is, however, after a time destined to be dispelled. The feebleness of the lower extremities gradually increases, so that the child cannot maintain the erect posture, while the muscles of the superior extremities also become both paralyzed and atrophied, and even the hypertrophied limbs begin to waste. The patient, now arrived at adolescence, may live on for several years in a condition of almost complete paralysis, until finally death takes place from exhaustion, implication of the respiratory muscles, or, more usually, from some intercurrent affection.

The patellar tendon-reaction is lost at a comparatively early period of the disease, and the electrical reaction of the muscles remains normal so long as any muscular fibre is left. The skin over the affected parts often presents a mottled appearance, and the superficial temperature of the inferior extemities has sometimes been found higher than that of the trunk.

The disease is associated with a certain amount of mental incapacity. Patients are sometimes slow in acquiring speech, others are obtuse in intelligence, and some are idiots. The disease is unaccompanied by suffering, there is no alteration of sensibility, and the functions of the bladder and rectum are unaffected, while the general health is not interfered with until the terminal period of the disease.

The *course* of the disease is always chronic. It begins insidiously, and consists of a first stage, in which there is progressive enfeeblement of the lower extremities, saddle-back, and waddling gait, and which may last from a few weeks to some years before the commencement of the next stage. This second period is characterized by apparent hyper-

trophy of a certain number of muscles usually beginning in those of the calf, and extending gradually to other muscles of the trunk and upper extremities. The stage of hypertrophy attains its maximum extent and intensity generally eighteen months from the beginning of the second stage. The symptoms now remain stationary for two or three or for many years. Some of the cases described by Erb under the name of "the juvenile form of progressive muscular atrophy," live to a moderately old age, and the disease may then remain more or less stationary for from ten to twenty or more years.

The third stage of the disease is now ushered in by a still further enfeeblement of the affected muscles, and by the extension of the paralysis to the superior extremities. Abduction and elevation of the arm are at first rendered difficult, and by and by the paralysis gradually implicates the other movements of the arm.

The last stage of the disease generally begins about puberty, although it is sometimes deferred until a moderately mature age. The slight power of movement of which the patient was capable becomes gradually lost, so that he can only sit in a chair or recline on a couch. The patient may continue to live for a long time in this condition, but eventually death supervenes from exhaustion or some intercurrent malady.

Morbid Anatomy and Physiology of the Atrophic Paralyses.

1. *Changes in the Nervous System.*—Paralysis of the muscles supplied by individual nerves is caused by a local neuritis, by injuries of the nerve from external wounds, or by compression of it from tumors growing in the nerve or in neighboring structures. When a nerve is seriously injured in any part of its course, the peripheral part undergoes the Wallerian degeneration, and the muscles supplied by it are not only paralyzed, but also undergo atrophy.

The morbid anatomy of progressive multiple neuritis has already been described. The most important changes observed in lead paralysis have been found in the intramuscular nerve fibres, and in the musculo-spiral nerve. The connective tissue and the sheath of the primitive fibres are thickened, while the axis-cylinders are sometimes distinctly visible, and at other times disappear altogether. Vitreous degeneration and atrophy of the ganglion cells have been found by some observers, while others failed to detect any change in the spinal cord.

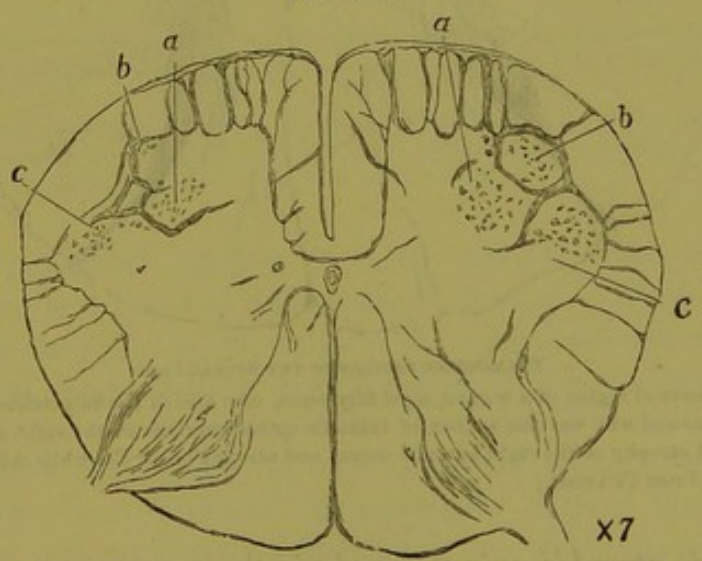
In diphtheritic paralysis the nerves of the palate have been found altered in the same manner as occurs in the peripheral end of a nerve

after section, and Déjerine found similar alterations in the anterior roots of the nerves on a level with that portion of the spinal cord from which the paralyzed muscles are innervated. Changes have also been observed in the anterior gray horns of the spinal cord, but they are slight, and the balance of evidence appears to favor the idea that the primary lesion is situated in the course of the nerves. In alcoholic paralysis also the most recent observations seem to prove that the paralysis is caused by a neuritis of the peripheral nerves. No anatomical changes have as yet been discovered in those cases of reflex paralysis which are attended with wasting of the extensor muscles of the inflamed joint.

In the spinal atrophic paralyses the chief lesions have been found in the anterior gray horns of the spinal cord.

In *acute ascending paralysis* post-mortem examinations have yielded, for the most part, completely negative results, no morbid changes whatever having been detected in the spinal cord, peripheral nerves, or

FIG. 139.

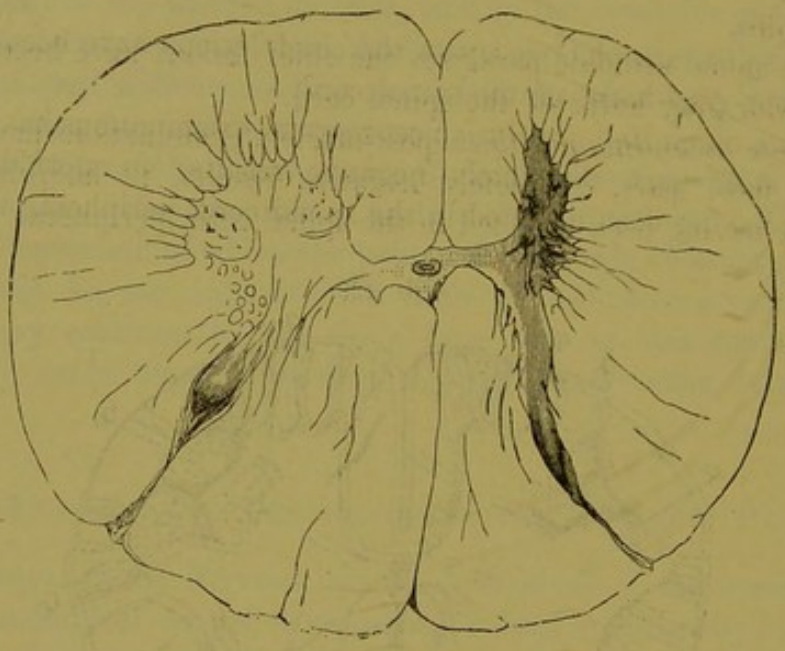


The letters *a*, *b*, *c* indicate respectively the central, antero-lateral, and postero-lateral groups of ganglion cells On the left side the group *b* has almost entirely disappeared, causing a marked falling in of the circumference of the gray matter. The groups *a* and *c* are fairly well represented on the left side, but the cells composing them are not so numerous as on the right. The internal group has disappeared from both sides. (After HUMPHREYS.)

muscles. In other recorded cases, which apparently were examples of the disease, the usual signs of an acute myelitis were discovered, while in a case reported by Déjerine and Goltz decided evidences of a degenerative neuritis were found in the anterior roots of the spinal nerves. It is very probable that some of the cases recorded as examples of Landry's paralysis really belong to the group of progressive multiple neuritis.

Acute spinal atrophic paralysis appears to be an acute inflammation which remains more or less limited to the anterior gray horns of the spinal cord (Fig. 139). The antero-lateral columns may also manifest a greater or less degree of sclerosis, while the anterior roots and the efferent fibres of the peripheral nerves give evidence of having undergone a degenerative atrophy. In old-standing cases the anterior horn of the affected side, when the disease is unilateral, undergoes marked atrophy as compared with the healthy one (Fig. 140).

FIG. 140.



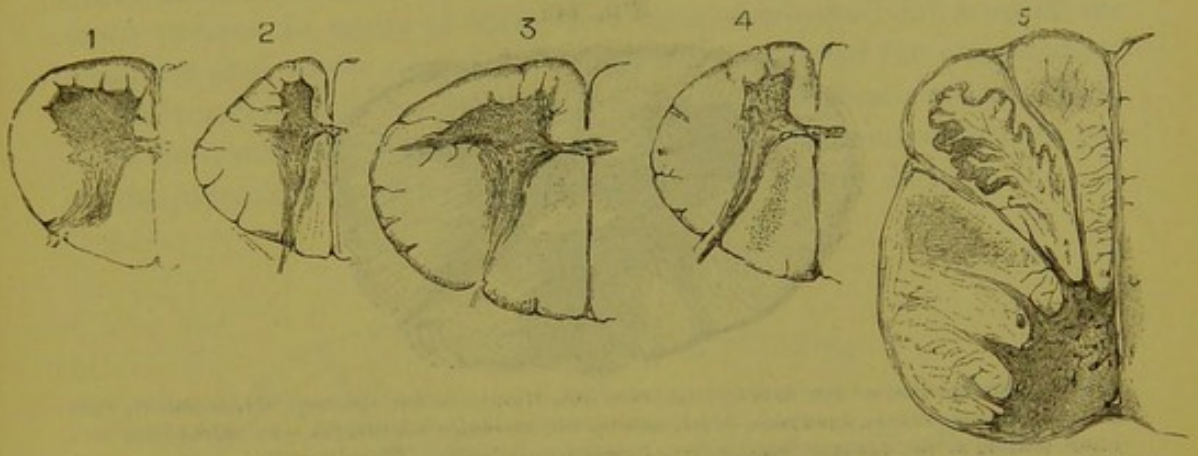
TRANSVERSE SECTION OF THE SPINAL CORD

taken from the cervical region of a woman, aged fifty years, who died in the Salpétrière, of general paralysis of the insane, and who was the subject of infantile spinal paralysis of the right superior extremity. There was fibroid atrophy of the right anterior cornu, and atrophy of all the white columns of the corresponding side. (From CHARCOT.)

In *chronic atrophic spinal paralysis* the post-mortem examinations hitherto obtained have been few, but cases have been recorded by Cornil, Webber, Aufrecht, Dreschfeld, and myself. The chief changes were found in the anterior gray horns, and consisted in disappearance and atrophy of the ganglion cells, destruction of their processes, thickening of the walls of the bloodvessels, and exudation of the white and of a few red blood-corpuscles into the perivascular spaces. The case recorded by myself was that of a young girl aged fifteen years, who had died from respiratory paralysis after having the typical symptoms of the disease for seven months. The ganglion cells had almost completely disappeared from the anterior horns throughout the entire

length of the spinal cord (Fig. 141, 1 to 4), while the upward continuation of the anterior horns in the medulla was similarly affected but to a less degree (Fig. 141, 5). Slight changes were observed in the columns of Goll, which showed that the lesion was not strictly limited to the anterior horns, although they were the portions which chiefly suffered.

FIG. 141.



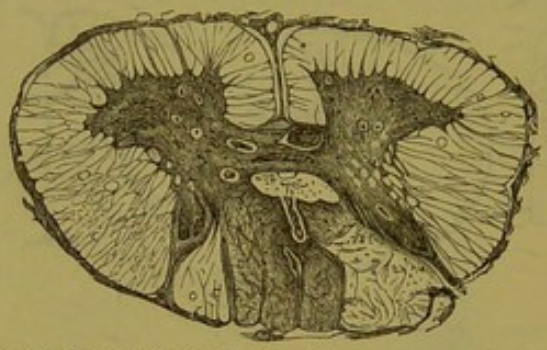
TRANSVERSE SECTIONS OF THE SPINAL CORD AND MEDULLA OBLONGATA AT DIFFERENT LEVELS, FROM A CASE OF CHRONIC ATROPHIC SPINAL PARALYSIS, SHOWING THE DISAPPEARANCE OF THE GANGLION CELLS. (YOUNG.)

1, Middle of the lumbar enlargement; 2, Middle of the dorsal region; 3, Middle of the cervical enlargement; 4, Section on a level with the origin of the second cervical nerve; 5, Section of the medulla oblongata on a level with the middle third of the olivary body.

In *periependymal myelitis* the gray column which surrounds the central canal is the seat of inflammatory changes. These changes consist in some cases of a proliferation of the epithelioid cells which line the central canal, while in other cases the morbid process appears to begin in the neuroglia which surrounds the canal, and the central gray column becomes converted into a solid mass of fibroid tissue which grows from within outwards, and invades the remaining parts of the gray matter. The central core may be solid throughout the whole extent of the cord, but in most cases a portion of the interior undergoes softening and a cavity is formed which is filled with serous fluid. The cavity varies greatly in its dimensions; it sometimes extends the whole length of the cord, while at other times it is only a few lines in length; and on transverse section it is sometimes large enough to admit the tip of the finger, while at other times it is only large enough to be visible to the naked eye. The cavity is usually situated, not in the position of the central canal, but in that part of the posterior columns which adjoins the posterior commissure, and the central canal can generally be found lying in front of it. The cavity is always surrounded by a ring of fibroid tissue, which grows from within outwards, and invades the gray sub-

stance of the cord, and which may even extend to the white substance, especially to the lateral columns. In some cases the cavity forms the predominating feature of the morbid change, while in others the cavity is insignificant and almost the whole of the central gray column is converted into a dense mass of sclerosed tissue. In the annexed woodcut (Fig. 142) borrowed from Leyden, a section of the cervical enlargement

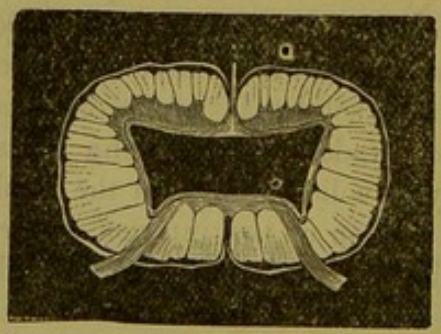
FIG. 142.



TRANSVERSE SECTION OF THE SPINAL CORD FROM THE MIDDLE OF THE CERVICAL ENLARGEMENT, FROM A CASE OF SYRINGOMYELIA, SHOWING A CAVITY BEHIND THE POSTERIOR COMMISSURE, AND DESTRUCTION OF A LARGE PORTION OF THE GANGLION CELLS OF THE ANTERIOR GRAY HORNS. (From LEYDEN.)

of the spinal cord, from a case of syringomyelia, is represented; the central gray column is changed into a substance of a gelatinous consistence, which became softened into cavities at certain points. In a case observed by Sir W. Gull a considerable dilatation of the spinal canal (Fig. 143) was found in the cervical region, and on this level the

FIG. 143.



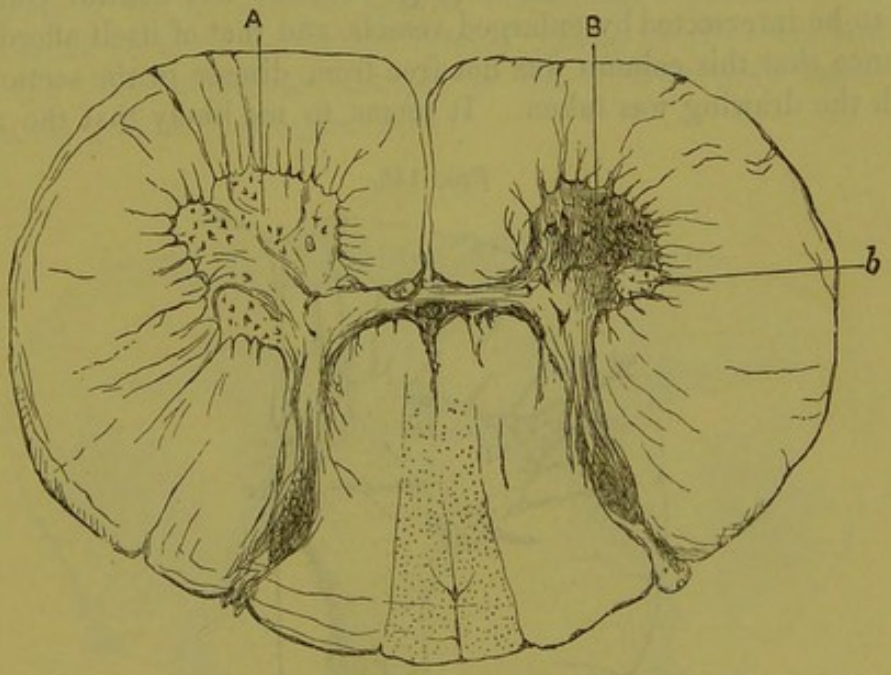
TRANSVERSE SECTION OF THE CERVICAL ENLARGEMENT OF THE SPINAL CORD, SHOWING A CENTRAL CAVITY, WHICH HAS DESTROYED CONSIDERABLE PORTIONS OF THE ANTERIOR GRAY HORNS. (After GULL.)

gray substance of the cord was so much compressed as to have almost disappeared. In a case recorded by Westphal the cavity was said to have resulted from the softening of a gliosarcomatous tumor, but in a later paper by the same author it is admitted that the tumor was most probably a mass of inflammatory tissue.

Treatment.—The treatment is the same as that for the subacute ascending general paralysis of Duchenne.

In *progressive muscular atrophy* the spinal cord has, according to Eulenburg, been examined in forty-nine cases, and out of these, morbid changes have been found in thirty-four, while the cord was healthy in fifteen cases. But too much importance must not be attached to the cases in which the results were negative, inasmuch as changes may have been present in many of them, and been overlooked for want of the requisite skill on the part of the person who conducted the examination, while it is certain that cases have been included in these statistics which must be transferred from the group of progressive muscular paralysis to the juvenile form of muscular atrophy described by Erb.

FIG. 144.



TRANSVERSE SECTION OF THE CERVICAL REGION OF THE SPINAL CORD, FROM A CASE OF PROGRESSIVE MUSCULAR ATROPHY. (CHARCOT.)

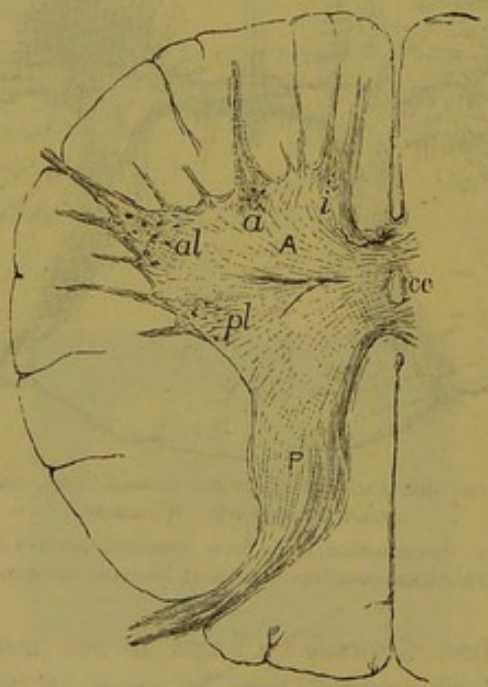
A, Left anterior gray horn; the ganglion cells have persisted, but are much altered in appearance. B, Right anterior gray horn, almost complete atrophy of the cells, one group only (*b*) having persisted.

Attention was first directed by Luys to the morbid changes of the gray substance of the spinal cord in cases of progressive muscular atrophy. In a case of advanced progressive muscular atrophy he found the capillary vessels of the gray substance of the anterior horns increased in number, the walls of the vessels thickened and surrounded by granular exudation, numerous corpora amylacea scattered through the gray substance, and disappearance of a considerable number of the ganglion cells and disintegration of others. The degeneration affected the left

anterior cornu principally, this localization being the counterpart of the fact that the atrophy was most pronounced on the left side. The spinal cord has been examined in six cases of the disease by Lockhart Clarke, and the observations of Luys have been confirmed by him in all essential respects, and similar observations have been made by Duménil, Hayem, Charcot and Joffroy, Pierret and Troisier, and others.

In a case reported by Charcot the ganglion cells of the left anterior gray horn (Fig. 144, A) could still be distinguished, but were observed to be in an advanced stage of atrophy. In the right anterior horn (Fig. 144, B), however, the cells could only be distinguished in one group—the postero-lateral (Fig. 144, *b*)—while the cells of the remaining group were completely destroyed. But it has appeared to me that too little attention has hitherto been paid to the condition of the central gray column. In this diagram (Fig. 144) the left central column is seen to be intersected by enlarged vessels, and that of itself affords some evidence that this column was not free from disease in the section from which the drawing was taken. It seems to me likely that the morbid

FIG. 145.



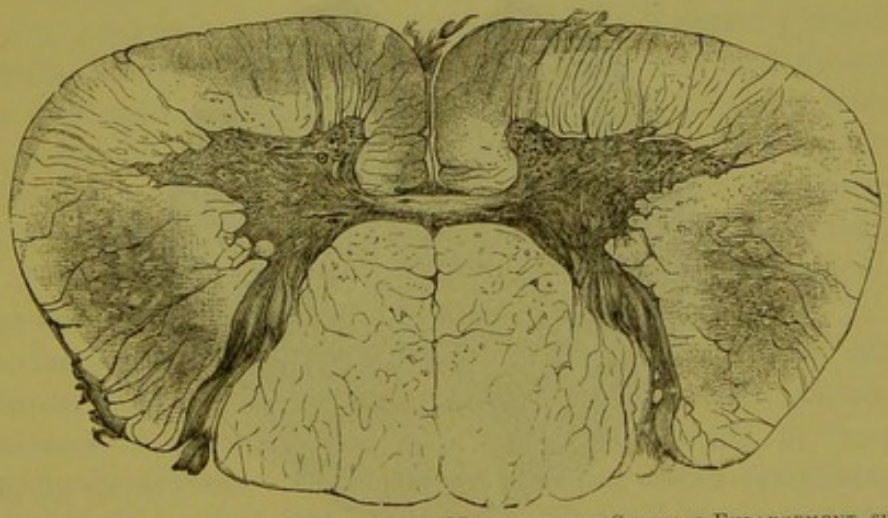
TRANSVERSE SECTION FROM THE MIDDLE OF THE CERVICAL ENLARGEMENT OF THE SPINAL CORD, FROM AN ADVANCED CASE OF PROGRESSIVE MUSCULAR ATROPHY. (YOUNG.)

cc, Central canal; *i*, Internal; *al*, Antero-lateral, and *pl*, Postero-lateral groups of ganglion cells.

process begins on each side of the central canal, probably in the tissues immediately adjoining the central artery, and that it extends outwards and forwards, as well as upwards and downwards from this point as a centre. In a transverse section of the middle of the cervical enlarge-

ment in my possession, which I owe to the kindness of Dr. Dreschfeld, it was unmistakable that the central gray column was more diseased than any other part of the section. This column was traversed by enlarged vessels, and almost all structure was obliterated, while the various groups of ganglion cells on the anterior horns were still distinctly recognizable. The cells of the median area and the marginal cells of the other groups were destroyed, but considerable traces of the large cells of the groups were still distinctly visible (Fig. 145). I have also observed in one of my sections a streak of degeneration to pass along the posterior branch of the central artery and into the substance of the posterior horn, and this may explain why patches of cutaneous analgesia are sometimes met with in progressive muscular atrophy. In the accompanying woodcut (Fig. 146) borrowed from Leyden's work on the diseases of the spinal cord, it is also distinctly represented that the most diseased portions occupy the central column of the cord, and that thence the disease extends laterally to the anterior gray horns and between the groups of ganglion cells.

FIG. 146.



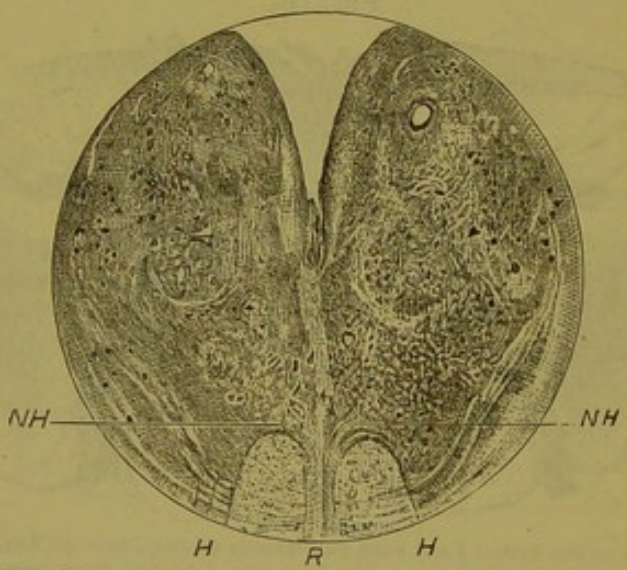
TRANSVERSE SECTION OF THE SPINAL CORD FROM THE MIDDLE OF THE CERVICAL ENLARGEMENT, SHOWING THAT THE CENTRAL COLUMN AND A LARGE PORTION OF THE ANTERIOR GRAY HORNS ARE DISEASED. (From LEYDEN.)

The *anterior roots of the nerves* were first observed to be diseased by Cruveilhier in the case of the showman Le Compte, who died of a progressive muscular atrophy of five year's duration, and similar observations have since been made by others. The peripheral nerves have likewise been found diseased by Virchow, Hayem, Charcot and Joffroy, Bamberger, and many others. The changes observed consisted of hyperplasia of the neurilemma, multiplication of nuclei, and a fibrillary thickening of the sheath of Schwann.

In addition to the affection of the anterior horns and peripheral fibres, the posterior columns, the posterior horns, the posterior roots, and the intervertebral ganglia have been found in a state of degeneration. The cervical sympathetic was found diseased in a case examined by Schneevogt, the cervical ganglia were almost entirely changed into fat cells, while the cords of the cervical and thoracic sympathetic abounded in fat cells which compressed the nerve fibres. More or less similar observations were made by Jaccoud, Swarzenski and Duménil.

In *progressive bulbar paralysis* the essential morbid changes appear to consist of a degenerative atrophy of the ganglion cells of the gray nuclei in the floor of the fourth ventricle. The cells shrink and become filled with yellow or brown pigment, their nuclei disappear, and finally the cells themselves are only represented by angular, glistening, and pigmented masses. The connective tissue is found to be increased and to contain Glüge's corpuscles; the nuclei and Dieter's cells are multiplied, and the vascular walls are hypertrophied or have undergone fatty degeneration.

FIG. 147.



PORTION OF THE GRAY SUBSTANCE ON THE FLOOR OF THE FOURTH VENTRICLE ON A LEVEL WITH THE MIDDLE OF THE HYPOGLOSSAL NUCLEUS, FROM A CASE OF PROGRESSIVE MUSCULAR ATROPHY WITH BULBAR PARALYSIS, SHOWING THE DESTRUCTION OF THE GANGLION CELLS OF THE NUCLEI OF THE HYPOGLOSSAL AND PNEUMOGASTRIC NERVES (NH). (From LEYDEN.)

R, Median raphe; *H*, *H*, Fibres of the hypoglossal nerves. The accessory nuclei have evidently disappeared.

The nuclei of the hypoglossal nerves appear to be the starting point of the disease, and the nuclei of the spinal accessory and pneumogastric nerves are afterwards invaded, but the disease does not always extend to the nuclei of the glosso-pharyngeal nerves. The nuclei of the facial nerves are attacked at a very early period of the disease, and

the nuclei which are connected with the inferior branches of the nerves, and which I have named the accessory nuclei, are especially liable to be invaded. The motor nuclei of the fifth nerves have been found affected, but the nuclei of the sixth and acoustic nerves and the trigeminal sensory nuclei seem never to suffer. The annexed diagram (Fig. 147), borrowed from Leyden, represents the morbid changes observed in the medulla oblongata in bulbar paralysis. Remnants of the fundamental cells of the hypoglossal nuclei may still be observed, while no traces of the accessory nuclei are left. This would seem to indicate that the morbid process begins in the embryonic tissue which immediately underlies the ependyma of the fourth ventricle, and that with advance of the disease the changes extend deeper and deeper into the gray matter and among the oldest formed cells. Other changes have been described, but they appear to be secondary to the alterations in the ganglion cells. Atrophy of the roots of the hypoglossal, facial, and spinal accessory nerves was discovered by the earlier observers with the naked eye, and degenerative atrophy of the nerve fibres was subsequently observed on microscopic examination. The muscles are found to undergo essentially the same degenerative changes which are observed in progressive muscular atrophy. The anterior pyramids of the medulla oblongata have often been found diseased, and the morbid changes were generally traced upwards into the pons and downwards into the antero-lateral columns of the spinal cord. It is probable that in all these cases the lower extremities were affected during life with a spasmodic paralysis. The bulbar affection is often associated with lesions of the anterior gray horns of the spinal cord, indicating a complication during life with progressive muscular atrophy.

In a fatal case of *ophthalmoplegia externa* reported by Mr. J. Hutchinson, degeneration of the roots of the oculo-motor nerves and disappearance of the cells from their nuclei were found, at the post-mortem, by Dr. Gowers. In a case of locomotor ataxia complicated by ophthalmoplegia externa, reported by Dr. Buzzard, extensive changes were discovered by Dr. Bevan Lewis in the nuclei of the sixth nerve and in the ascending root of the fifth nerve, and in a case of this kind which died recently under my care, I noted the disappearance of cells from the nuclei of the third pair of nerves, and degenerative changes in the ascending and descending roots of the fifth nerve and in the fasciculus rotundus. It appears likely that two kinds of cases are included in this group. In the one the ganglion cells of the nuclei of the ocular nerves undergo a primary degeneration just as occurs in bulbar nuclei in progressive bulbar paralysis, while in the other, the primary disease is found in the cranial homologues of the posterior

root-zones, the ascending and descending roots of the fifth nerve and the fasciculus rotundus, and the ganglion cells of the oculo-motor nerves are only implicated secondarily.

In *pseudo-hypertrophic paralysis* and the *juvenile form of progressive muscular atrophy* the spinal cord and peripheral nerves are normal or show only minor changes, which must be regarded as secondary to the changes in the muscular system.

2. *Changes in the Muscles.*—When a peripheral nerve is divided or injured the muscle supplied by it undergoes a progressive atrophy, the various degrees of which have already been described. Essentially similar changes occur in the muscles in the various forms of progressive multiple neuritis, but in reflex paralysis the muscular fibres only undergo a simple wasting without proliferation of nuclei or destruction of the

FIG. 148.

MUSCULAR FIBRES FROM A CASE OF ADVANCED INFANTILE PARALYSIS WITHDRAWN BY LEECH'S TROCAR. (YOUNG.)

a, Muscular fibres presenting a more or less healthy appearance; *b*, muscular fibres, somewhat atrophied, and with granular contents; *c*, muscular fibres greatly atrophied, but presenting faint traces of transverse striation, and having their surfaces thickly studded with nuclei.

fibres. In Landry's paralysis, also, the muscular fibres may undergo some degree of wasting, but they have not been found much altered in their structure. In the acute atrophic spinal paralysis of infants and adults the muscles undergo essentially the same changes as after severe injury of a peripheral nerve. When a portion of a muscle which has undergone advanced atrophy is withdrawn by means of Leech's trocar, it will be found, as shown in Fig. 148, that some of the fibres are

more or less healthy (*a*); that others have lost their normal striation and present granular contents, but are not much diminished in size (*b*); that a large number are reduced to slender, transparent filaments, while still others retain a faint degree of their transverse striation, but have their surfaces thickly studded with nuclei (*c*). The nuclei may be observed to project distinctly from the surface of its atrophied fibre, and it is, therefore, probable that they have been derived from either the nuclei of the sarcolemma or of the endomysium. In chronic atrophic spinal paralysis the changes in the muscles are never so well marked as they are in the acute variety, while in periependymal myelitis the muscles have not been carefully examined.

In progressive muscular atrophy the muscles have been found variously altered. The affected muscles are generally of a pale red or rose color, while at other times they may be buff or ochre, and streaks of adipose tissue may be seen to run in lines between the fibres. The muscles are wasted in various degrees, and different parts of the same muscle may present differences in the degree to which the atrophy is carried, a portion being sometimes normal while the rest is reduced to a fibrous band.

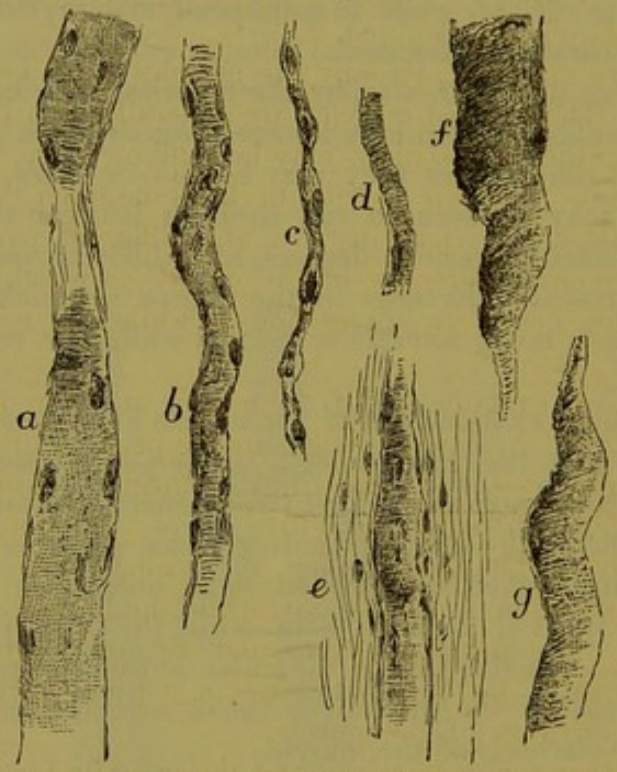
A microscopical examination of the muscle at different stages of the atrophy shows that the changes begin in the perimysium internum as a hyperplastic growth of the interstitial connective tissue in its finest ramification among the single primitive fibres. The muscle corpuscles are increased in number, and swollen, their nuclei are multiplied, and the transverse striation becomes faint, while the fibres present a granular cloudiness. Wasting of the muscular substance goes on side by side with increase of the interstitial tissue, a process which ultimately leads to a true cirrhosis of the muscle.

In pseudo-hypertrophic paralysis and the juvenile form of progressive muscular atrophy the hypertrophied muscles appear as yellow fatty masses of tissue, with scarcely a trace of muscular structure left, while those which have undergone extreme atrophy may appear as fibrous bands.

The first muscular change which takes place in this disease consists of an increase of the connective tissue which separates the muscular bundles from one another, so that the sheaths of the muscular bundles become greatly thickened. There is also a corresponding increase of the connective tissue which passes between the fibres themselves. The comparatively thick masses of tissue which now separate the fibres from one another consist of fibres arranged parallel to the long axes of the muscular bundles, mixed with a considerable number of embryonic cells (Fig. 149, *e*). In this early stage the muscular fibres themselves

do not undergo any manifest changes, except that, according to Duchenne, their transverse striation becomes fainter, while their longitudinal striation becomes more marked (Fig. 149, *a*). The second stage of the change consists of the development of fat cells in the connective tissue and also in the newly formed fibrous tissue, whereby the muscular fibres become widely separated from one another. The muscular fibres now become atrophied and begin to disappear. They become narrower, and, indeed, a single fibre varies in diameter at

FIG. 149.



MUSCULAR FIBRES IN VARIOUS STAGES OF DEGENERATION, FROM A CASE OF PSEUDO-HYPERTROPHIC PARALYSIS. (YOUNG.)

a, Muscular fibres only slightly changed, showing increase of the muscle corpuscles, and indistinctness of the transverse striation in certain parts of its length; *b*, the same as *a*, but more atrophied; *c*, muscular fibre greatly atrophied, and presenting nuclei at intervals; *d*, atrophied muscular fibre, with its transverse striation unusually distinct; *e*, atrophied fibre surrounded by a fibrillated connective tissue rich in nuclei; *f* and *g*, muscular fibres from the erector spinæ, which manifested the greatest changes to the naked eye. These fibres appear to have undergone a hyaline change, but their transverse striation is still faintly visible. The fibres often tapered to a point, sometimes at one and sometimes at both ends.

different points in its length. The transverse striation may disappear in the narrower fibres and be replaced by granules distributed uniformly through them. The fibrous tissue surrounding the fibres contains oat-shaped nuclei which are supposed by some pathologists to be derived from the empty sheaths of muscular fibres. After a time both the muscular fibres and the newly formed fibrous tissue completely disappear, and the entire muscle is represented by fat cells like those of an

ordinary lipomata, but may subsequently be absorbed, and all that is left of the muscle is connective tissue with, perhaps, a few traces of muscular fibres.

The fact that this disease is accompanied by a progressive paralysis and atrophy of muscles has led authors to believe that the essential morbid changes would be found in the anterior gray horns of the spinal cord, and slight changes have been discovered in some of the cases which have been examined post mortem. These changes have not always been the same in different cases and are likewise so slight in degree that they are not likely to be essential to the disease. A considerable number of cases are now reported in which the spinal cord and peripheral nerves were found quite healthy after being examined by the most competent observers and with the aid of the most approved methods of research. Under these circumstances it must be assumed that the muscular atrophy is either primary, or results from disease of the intramuscular nerve fibres and endings, and whatever changes are met with in the cord are either secondary to the muscular disease, or accidental. And if the cases which have been described by Erb under the name of "the juvenile form of progressive muscular atrophy" be transferred from the group of progressive muscular atrophy, in which many of them have hitherto been included, to that of pseudo-hypertrophic paralysis then we have on the one hand a primary disease of the ganglion cells of the spinal cord, and on the other a primary disease of the muscles or of the intramuscular nerve endings.

The *tendons, joints, and bones* are liable to undergo morbid changes in some of the forms of atrophic paralysis. These changes are probably best marked in the acute atrophic paralysis of infants. In this affection the *tendons* appear as thin, narrow bands, and are found to be much atrophied when compared with corresponding healthy structures. The *bones* are retarded in their growth when the disease occurs in childhood; the protuberances and processes are little developed, and the epiphyses are shortened. The external lamella of the long bones is thin and friable, while the medullary portion is increased, and its fatty contents more abundant. The *ligaments* are thin and loose, while the articular extremities of the bones are atrophied, eroded, and their cartilages attenuated. The *arteries* are said to be diminished in calibre. The skin and the circulatory and digestive organs are normal or only show changes which have no necessary connection with the spinal disease. Essentially similar changes occur in disease of the peripheral nerves.

Morbid Physiology.—The ganglion cells of the anterior gray horns of the spinal cord are the trophic centres for the efferent fibres of the peripheral nerves and muscles, and they also appear to produce an

influence upon the nutrition of the bones, tendons, and joints. A destructive lesion of a peripheral nerve severs the ganglion cells of the anterior horns from the distal portion of the nerve and from the tissues to which the efferent fibres are distributed, and consequently the nutrition of tissues becomes impaired. In reflex paralysis an irritation is conducted through afferent fibres to the motor ganglion cells and reflected by them outward to the muscles; or, as suggested by Charcot, the peripheral irritation may exercise an inhibitory influence upon the functions of the ganglion cells, and a diminished functional activity on the part of these cells will lead to a lessened nutrition in the muscles. Ligature of the hilus of the kidney in animals was observed by Brown Séquard to cause spasm of the spinal vessels, and from the results of this experiment he argued that urinary paraplegia is caused by anæmia of the spinal cord. Jaccoud supposes that in such cases the paralysis is caused by the exhaustion which follows over-excitement, while S. Weir Mitchell adopts a view which appears to be a combination of the theory of reflex vascular spasm, and that of exhaustion. It has been found by Lewisson that when the uterus, one kidney, or a fold of the intestine of the hare is strongly compressed, after exposure, a paralysis of the inferior extremities results, which persists so long as the pressure is maintained and disappears immediately on its being removed. This experiment shows that the paralysis is caused by a functional lesion of the cord, consisting most probably of an inhibitory influence exerted upon the motor ganglion cells. When myelitis occurs it is an ascending myelitis.

Acute atrophic spinal paralysis is one of those diseases the morbid anatomy of which has largely contributed to clear up our knowledge of the functions of the anterior gray horns. The multipolar cells probably constitute ganglionic centres both for reflex action and for the transmission of impulses received from the cortex through the pyramidal tracts, and when they are destroyed both reflex actions and voluntary actions are impaired or abolished according as the destruction of the cells is incomplete or complete. Destruction of the ganglion cells is also followed by various trophic changes in the muscles, bones, tendons, and joints. The ganglion cells which constitute the spinal centre for the regulation of the movements of a muscle appear also to form its trophic centre. But most muscles are connected with fundamental and accessory cells and it is only when the connection, between the former and the muscle, is severed that its motor power and nutrition suffer profoundly. The acute nature of the lesion in acute atrophic paralysis is well calculated to sever the muscles from their connections with the fundamental cells, even if the latter were to remain themselves entirely

unaffected, and consequently the clinical features of the paralysis are very similar to the paralysis which results from severe lesions of the peripheral nerves.

The case is wholly different when we have to do with a chronic and progressive affection like progressive muscular atrophy, in which the accessory cells are first attacked, and the disease invades by slow and successive steps the fundamental cells. In such a disease the symptoms of paralysis and atrophy may be expected to pursue a totally different course from that which obtains in acute atrophic spinal paralysis.

In progressive muscular atrophy fibre after fibre of the muscles attacked become diseased in correspondence with a similar progressive invasion of the ganglion cells and their processes, and the consequence is that so long as the healthy fibres predominate over the diseased fibres so long will the muscle give normal reactions to electrical examination. But in acute atrophic spinal paralysis all the fibres of a muscle are more or less simultaneously attacked and consequently the muscle gives the reaction of degeneration at a very early period of the disease.

Treatment.—In the treatment of the various forms of atrophic paralysis the first indication is to remove the exciting cause. In carrying out this indication in cases of paralysis of peripheral nerves considerable scope is afforded for various kinds of surgical interference, such as bringing together by means of suture the ends of divided nerves, the removal of tumors and diseased bones which compress or otherwise injure nerve trunks. When the paralysis is of rheumatic origin antiphlogistic treatment must be adopted in the early stages of the disease, and when it is caused by the presence of lead, alcohol, or other poison, means must be taken for withdrawing the patient from the influence of the deleterious agent, and for eliminating it from the system. If the paralysis is of syphilitic origin, mercury and iodide of potassium must be prescribed either separately or combined. In the early stage of every form of acute spinal atrophic paralysis a mild antiphlogistic treatment must be adopted.

If the nature of the disease be detected at an early period, an ice bag may be applied to the spine. In the early stage of the atrophic paralysis of infancy ergotine has been employed subcutaneously in doses of one-fourth of a grain for a child from one to two years of age, one-third of a grain for one from three to five years, half a grain for children from five to ten years of age, and a grain for patients upwards of ten years of age, the dose being repeated either daily or twice a day. Belladonna has also been employed with apparent benefit in this stage of the affection. After the fever has subsided iodide of potassium may be administered in appropriate doses with the view of promoting the

absorption of effused products, while mild counter-irritants might be applied along the spine.

In the latter stages of acute cases and in all chronic cases of atrophic paralysis stimulating treatment must be adopted. A nutritious and abundant diet should be prescribed along with prolonged sojourn in the open air, mountainous or sea-air being especially useful. The thermal springs of Wildbad, Teplitz, and Gastein, have been specially recommended, and sulphur baths have also been found useful.

Electrical treatment is more useful in the treatment of the various forms of atrophic paralysis than in that of any other variety of paralysis. In the spinal atrophic paralysis the constant current should be made to pass through the diseased area of the cord immediately after the fever has subsided. If the leg alone be affected, the current should be applied over the lumbar enlargement; if an arm only be affected, the cervical enlargement should be acted upon, and if the muscles of the trunk suffer likewise, the whole dorsal region of the cord should be included in the circuit. In order to reach the cord it is better to place one pole on the spine, and to apply the other to the anterior surface of the trunk. The electrodes should be large, the one placed over the back being large enough to cover the entire diseased area. The force of the current should be gentle, and the application continued for from three to ten minutes according to the extent of the lesion. The local application of electricity to the diseased nerves and muscles is useful in all cases in which degenerative changes have occurred in the muscles. So long as the muscles and nerves have not entirely lost their faradic contractility the local application of the faradic current will be of service. But as a rule the constant is superior to the interrupted current, being in most cases the only agent which will evoke muscular response. Appropriate gymnastic exercises of the affected muscles when they are not completely paralyzed, and shampooing and friction, with or without stimulating liniments, may be employed as adjuncts to the electrical treatment. After the chronic stage of atrophic paralysis is fully established internal remedies do not appear to do much good. The most usual remedies for chronic cases are phosphorus with cod-liver oil, arsenic, and tonics like iron and quinine, but these agents probably do good only in so far as they help to improve the general health. The use of strychnine has been advocated in cases of infantile atrophic paralysis, especially in the form of subcutaneous injection, but I have never seen any good results attend its employment. When serious deformities result from paralysis of certain groups of muscles the case should be placed under the care of the orthopædic surgeon.

CHAPTER VIII.

THE SPASMODIC PARALYSES.

I. PARAPLEGIÆ.

1. Primary Lateral Sclerosis (Tabes Dorsalis Spasmodica (Charcot), Spasmodic Spinal Paralysis).

Etiology.—A hereditary tendency to the disease can occasionally be traced. Dr. Morgan records the case of a man suffering from the typical symptoms of lateral sclerosis, whose brother was similarly affected, while a sister of his was said to have died of the same disease. Primary lateral sclerosis appears to occur rather more frequently in males than in females. By far the larger number of cases begin between the ages of thirty and fifty years. A spasmodic paralysis of the lower extremities of congenital origin is frequently met with in children, but the symptoms in these cases are caused most probably by a cerebral lesion.

The exciting causes are unknown, although it is very probable that exposure to cold, injuries of the spine, lead poisoning, and syphilis may coöperate as factors in the production of the disease. It seems to be caused sometimes by the use of a species of vetch—the lathyrus cicera—as a chief article of diet.

Symptoms.—The first symptom is a paresis of the inferior extremities, which may be equal in both or more pronounced in one than in the other, and the only effect of which is to render walking somewhat difficult, especially on getting out of bed in the morning. The patient complains that he is soon fatigued, and that his limbs are heavy, while his gait becomes dragging and difficult. On lying down at night, especially after being fatigued, the legs begin to shake, and as the disease advances the *tremors* may be so extensive and violent as to throw the whole body into a convulsive trembling, which has been named *spinal epilepsy*. These tremors may be readily excited by pushing against the toes so as to produce dorsal flexion of the foot. Muscular tension is developed at an early period of the disease; at first the tension, which is only present when provoked by passive movement of the lower extremities, may be overcome by increasing the pressure, and it

can be considerably diminished by repeated movements, but after a time the muscles become tense during voluntary efforts; the paresis then seems to be greater than it is in reality, and the movements of the affected limbs become stiff and difficult.

As the disease advances the muscular tension increases to permanent rigidity, and a high degree of *contracture* results. The lower extremities are maintained in a position of rigid extension, the thighs are also held rigidly together by contracture of the adductors, the feet are in a position of extreme talipes equino-varus, but the toes are generally strongly flexed. The rigid immobility of the foot is now and then interrupted by attacks of trembling, which are excited by every movement of the extremity.

The Spasmodic Gait or Spastic Walk.—The gait of the patient is rendered quite characteristic by the combined paresis, stiffness, and tremors of the lower extremities. The foot seems to cling to the ground, from which it is detached with difficulty, and it is made to slide forwards, and the toes produce a characteristic scraping noise. The presence of lateral sclerosis may, indeed, be sometimes recognized without seeing the patient by listening to the scraping or shuffling noise which accompanies each step, the "shuffle" in hemiplegia being only heard during alternate steps. Owing to the rigidity of the lower extremities in extension the necessary elevation of the passive leg is usually obtained by an upward rotation of the pelvis, which causes the body to be strongly inclined to the side of the active leg, and the alternate inclination of the body from one side to the other during successive steps gives rise to a "waddling gait." In other cases the necessary elevation of the passive foot is obtained partly or wholly by an unusual degree of elevation of the heel of the active leg, and in these cases the gait has a peculiar hopping character. When the body is inclined from one side to the other in locomotion, the passive foot advances not directly forwards, but forwards and outwards in a semicircular manner, and on being brought to the ground it is dragged over to the opposite side in front of the other leg by the predominant action of the adductors over their antagonists, while the foot is also often inverted by overaction of the inward rotators of the thigh. In long-standing cases the patient experiences great difficulty in disentangling the toes of the foot about to be advanced from the heel of the one which has just been brought to the ground, and in the effort to accomplish this the calves of both legs become strongly contracted, the patient is elevated on tiptoes, and a degree of ankle-clonus may be induced which suffices to give rise to alternate elevations and depressions of the body, and when at last the toes of the posterior are disengaged from the

heel of the anterior foot, and the passive leg is being moved forwards in the semicircular manner already described, the foot is seized with a trembling which may extend to the trunk and be so violent as to agitate the whole body. In aggravated cases of this kind the patient is quite unable to maintain the erect posture unsupported. On standing the patient rests on the tips of his feet, the body is inclined forwards, and the arms are propped up by crutches, or are supported by two sticks which are held well in front of the patient, and with an outward inclination. As the disease gradually extends upwards the abdominal muscles become prominent and tense, and at the same time a kind of lordosis is produced by spasm of the lumbar muscles.

When the upper extremities are affected the paretic condition of the hands manifests itself by the inaptitude of the patient to seize small objects. The digits are at times forcibly flexed into the palm, while a successive invasion of the muscles of the hands, forearms, and arms causes the upper extremity to become rigid and immovable in positions of extension and pronation, and to be strongly drawn to each side of the body.

Although the disease usually begins in the lower extremities and pursues an ascending course, yet occasionally the symptoms are developed in a different order. In some cases the paresis passes from a lower extremity to the upper one of the same side, and this hemiplegic condition may persist for many years before the other lower extremity is attacked. In a few cases the disease begins in the upper extremities and pursues a descending course.

The *tendon and periosteal reactions* are much exaggerated in this disease. The patellar tendon-reaction and ankle-clonus are elicited with undue readiness in the usual way, and the tremors induced may become diffused over the whole body. The quadriceps femoris and the adductors of the thigh may be excited to contract by tapping the broad end of the tibia, and the contractions may extend to the adductors of the opposite thigh. The adductors of the thigh may often be made to contract by tapping over the region of the lumbar vertebræ, and the tibialis posticus, semitendinosus, and other muscles by striking their tendons. The tendon reactions are in like manner increased in the upper extremities when once they are implicated. They can be elicited in the biceps and triceps by striking their tendons, and the former may be made to contract by tapping the lower end of the radius, and the latter by tapping the lower end of the ulna. The posterior portion of the deltoid often contracts along with the triceps when the lower end of the ulna is lightly struck. The flexors of the wrist, the extensors of the wrist, and the supinator longus can each be made to contract by tapping

their tendons at the wrist, while the interossei may sometimes be made to contract by striking the ends of the metacarpal bones.

The cutaneous reflexes appear to be occasionally increased, but they are generally normal or diminished. The electrical excitability of the motor nerves may manifest slight quantitative but never qualitative changes; the excitability of the muscles is slightly diminished to both currents.

Sensory disorders are absent throughout the whole course of the disease; the functions of the bladder, rectum, and sexual organs are unaffected; and there are no vaso-motor disturbances or nutritive affections of the muscles or skin, and no bedsores.

The course of the disease is almost always very chronic, and months or years may elapse before it can even be recognized with certainty. The disease occasionally ends in recovery, but in uncomplicated cases the symptoms slowly and gradually progress until after many years the patient is rendered quite helpless, but even then death generally occurs from accidental causes or intercurrent diseases.

2. Amyotrophic Lateral Sclerosis.

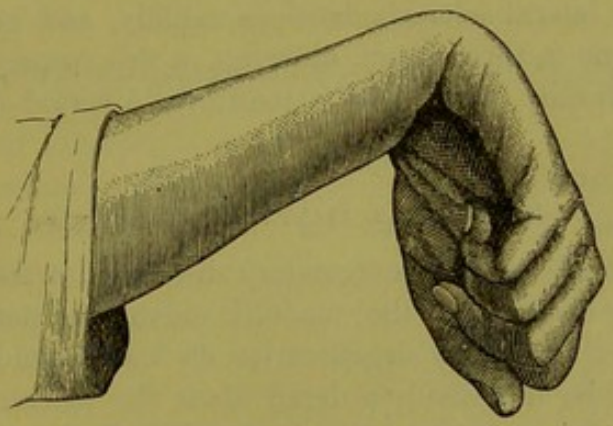
Etiology.—Very little is known with regard to the causation of this disease, but persons of middle age are most frequently attacked. Seeligmüller, however, met with the typical form of the disease in four children of the same family, and in all of them the symptoms appeared for the first time in early childhood.

Symptoms.—In this disease the symptoms of primary lateral sclerosis are complicated by a progressive muscular atrophy. The symptoms are, indeed, more allied to those of the latter than to those of the former disease, but as it would not be possible to understand the significance of the spastic phenomena until the clinical features of primary lateral sclerosis had been described, it has been found convenient to consider amyotrophic lateral sclerosis in this place.

The disease often begins with premonitory symptoms of formication and numbness in the upper extremities, but it is soon observed that these extremities are feeble, and that their muscles are undergoing a rapid and uniformly distributed atrophy, which is accompanied by fibrillary twitchings. Muscular tension and contractures are soon superadded to the paresis and atrophy, and the affected extremities are brought into permanently deformed positions. The arm is applied to the trunk; the forearm is semiflexed and pronated; the hand is flexed on the forearm; and the different segments of the fingers are flexed upon one another

and upon the metacarpal bones (Fig. 150); while any attempt to give passive movements to the limb at any of the articulations provokes muscular tension and resistance. When the patient elevates the arm by a voluntary effort, the extremity becomes agitated by tremors, not unlike those which occur in sclerosis in patches. In advanced cases the thenar and hypothenar eminences become flattened; the palm of the hand becomes excavated; the forearm and arm may be reduced almost to a skeleton, but the spasmodic rigidity becomes less pronounced,

FIG. 150.



ATTITUDE OF THE HAND AND FOREARM IN AMYOTROPHIC LATERAL SCLEROSIS. (After CHARCOT.)

although even now the limbs may maintain the forced attitudes in which they have been held so long.

In some patients the head is fixed by spasmodic rigidity of the muscles of the neck so that it cannot be moved in any direction. The contracture may also extend to the temporal muscles, and then the mouth can only be opened to a limited degree.

After a period of from two to six or nine months the lower extremities become affected by paresis, which may be preceded or accompanied for a longer or shorter period by formication and numbness of the limbs. The paresis of the lower extremities is not at first accompanied by muscular atrophy, but the symptoms are those of a pure lateral sclerosis, and consist of muscular tension, contractures with the limbs held rigid in the position of extension, tremors on voluntary or reflex movements of them, ankle-clonus, and exaggerated tendon-reactions. The rigidity of the muscles of the lower extremities soon become so great that walking is impossible, and the patient becomes helpless and bedridden. After a time even the muscles of the lower extremities undergo a diffused atrophy with fibrillary twitchings, the contractures diminish, and the limbs become flaccid and powerless, and lie immovable in any position in which they may be placed. The third stage of the disease is char-

acterized by the appearance of bulbar paralysis which pursues the usual progressive and fatal course, and the patient ultimately dies from the circulatory and respiratory disorders caused by implication of the nuclei of the pneumogastric nerves.

But although the symptoms usually begin in the upper extremities a few cases are reported in which the phenomena of bulbar paralysis took the precedence, and the muscles of the upper extremities became paralyzed and atrophied at a subsequent period, while those of the lower extremities remained the subjects of a spasmodic paralysis only until the terminal period of the disease.

Amyotrophic lateral sclerosis develops rapidly, and generally causes death in from one to three years, differing in this respect from progressive muscular atrophy, which may extend over a period of from eight to twenty years.

3. Secondary Lateral Sclerosis.

Lateral sclerosis occurs as a secondary disease in transverse myelitis and in various diseases of the medulla oblongata, pons, and brain. The symptoms of secondary degeneration of the pyramidal tract of the spinal cord will be described in detail when the primary lesions from which it results are under discussion. It will suffice at present to say that if, to the motor symptoms of lateral sclerosis already described, any sensory disorders, muscular atrophy, or disturbances of the functions of the bladder and rectum be superadded, then the case is not an example of the primary disease.

II. HEMIPLEGIÆ.

1. Ordinary Hemiplegia.

Hemiplegia consists of paralysis of one half of the body, although many of the muscles are either not implicated or only temporarily weakened. The paralysis is, as a rule, limited to the arm, leg, part of the face, and half of the tongue. The muscles chiefly affected in facial paralysis of cerebral origin are the buccinator, orbicularis oris, and the straight muscles which pass to the angle of the mouth and nose on the paralyzed side; while the occipito-frontalis, corrugator supercilii, and orbicularis oculi remain almost entirely unaffected. Paralysis of the hypoglossal nerve is manifested by a difficulty in executing certain movements of the tongue, and by a deviation of the point to the sound side on protrusion, because the base is dragged further forwards on the healthy than on the paralyzed side. It is often stated that the muscles

of the trunk are unaffected in hemiplegia, but in severe cases the inspiratory muscles act much less freely on the paralyzed than on the healthy side for the first few days of the attack.

2. Hemiplegia and Hemianæsthesia.

It has for some time been observed as a clinical fact that those cases of hemiplegia in which the leg is more paralyzed than the arm are associated with hemianæsthesia. In such cases, however, the special senses are not very seriously implicated.

3. Hemiplegia, Hemianæsthesia, and Hemianopsia.

Some cases of hemiplegia in which the arm is more paralyzed than the leg are accompanied by hemianæsthesia. In these cases the paralyzed limbs are often affected by choreoid movements and the cutaneous sensory disorders are accompanied by disturbances of the special senses. The sense of sight is variously affected; in some there is a restriction of the fields of vision on both sides, but it is most marked on the side of the paralyzed limbs, while in others a well-defined bilateral homonymous hemianopsia is present, the blind portions of the fields of vision being on the side of the paralyzed limbs. In certain cases of hemiplegia and hemianæsthesia which come on with severe apoplectic symptoms, one eyeball may be directed downwards and outwards, and the other upwards and somewhat outwards. Such cases may live for many days or weeks without the patient regaining a sufficient degree of consciousness to render it possible to determine whether hemianopsia is or is not present. Another curious symptom which is sometimes present is that the pupils may be found to dilate to light and to contract when shaded; but as the eyeballs are almost constantly moving, it is probable that the changes in the diameter of the pupils are to be regarded as an associated movement and not as a reflex action. In these cases repeated paroxysms of Cheyne-Stokes breathing may occur, and all of them that have come under my observation have proved fatal.

4. Crossed Hemiplegia.

In crossed hemiplegia the muscles supplied by one or other of the cranial motor nerves are paralyzed on one side and the limbs on the opposite side of the body. The cranial nerves most frequently affected in this form of paralysis are the seventh and the third nerves; when the first of these is affected the face is paralyzed on one side and the

limbs on the other, and when the last of these is attacked the muscles supplied by the oculo-motor nerve are paralyzed on one side and the limbs on the other side of the body.

5. Hemiplegia and Post-hemiplegic Spasms.

In hemiplegia the paralysis is accompanied by spasm of the affected muscles. These may be either *a*, tonic, *b*, combined tonic and clonic, or *c*, clonic spasms. In addition we must specially consider *d*, spasm as it occurs in the various forms of the spasmodic paralysis of infancy.

a. Tonic Spasms.

These spasms may be divided into (1) *early*, and (2) *late rigidity*.

(1) *Early rigidity* consists of a tonic spasm of the paralyzed muscles, which may either occur at the time of the hemorrhage or a few days subsequently during the inflammatory reaction. Early rigidity is sometimes so slight as only to be manifest when passive movement of the paralyzed extremity is made, while at other times it maintains both extremities in fixed positions of flexion, and any attempt to extend the limbs only increases the spasm. This form of rigidity generally disappears soon, but it occasionally persists for weeks or months.

(2) *Late rigidity* corresponds in its essential features to the spasmodic rigidity of primary lateral spinal sclerosis, and, like it, is attended with exaggeration of the tendon-reactions and periosteal reflexes. When the lower extremities are affected the patellar tendon-reaction is in excess, ankle-clonus is readily elicited, and corresponding phenomena may be obtained in the upper extremity when it is the subject of contracture. When the loss of voluntary power is complete the rigidity is more or less constant, although it is in most cases diminished during sleep and increased during voluntary efforts and emotional disturbances. The attitudes assumed by the limbs affected with late rigidity differ considerably in different cases, but on the whole they conform to the rule observed in almost all spasmodic affections; namely, that flexion predominates in the upper, and extension in the lower extremity.

The Hemiplegic Gait.—When the muscles of the paralyzed lower extremity have acquired a certain degree of rigidity, the patient is able to walk by the aid of a stick, even if the voluntary paralysis of the affected side remain complete. The patient on standing leans towards the healthy side, but is prevented from falling over to that side by the support of the stick, while the pelvis and hip-joint of the paralyzed side are elevated by contraction of the abductors of the healthy thigh, so that the weight is taken off the paralyzed extremity. When the

paralyzed lower extremity, say the right, *is the active one*, the line of gravity is carried over to a slight extent to that side, but instead of reaching the centre of the paralyzed foot it remains midway between it and the end of the stick, so that the weight of the body is maintained partly by the paralyzed lower extremity and partly by the healthy arm through the stick. The healthy or left lower extremity is now moved forwards a step, an unusual degree of flexion of the thigh upon the body taking place in order to avoid the necessity of carrying the line of gravity too far to the paralyzed side. The left leg now becomes active and the paralyzed one must be moved forwards. The manner in which this movement is executed depends upon the degree of paralysis and of muscular rigidity present. If the paralysis be almost complete, and the rigidity not great, the extremity is partly swung and partly dragged round mainly by contraction of the inward rotators of the healthy limb. Contraction of these muscles causes the pelvis to rotate forwards on the hip-joint of the healthy side, and consequently the opposite hip-joint, dragging after it the paralyzed leg, is moved forwards.

This forward movement is aided by a further elevation of the right hip-joint caused by contraction of the abductors of the opposite thigh, and sometimes by a slight backward inclination of the trunk by means of which the distance between the points of origin and insertion of the flexors of the thigh on the body is increased. If a high degree of contracture with talipes equinus be present, the paralyzed lower extremity is moved forwards much in the same manner as has already been described in the case of primary lateral spinal sclerosis. When once the weight of the body is taken off the paralyzed extremity the heel becomes elevated, and the toe during the forward movement, which takes place in a semicircular manner, makes a characteristic scraping noise. If tremors or choreoid movements be present in the paralyzed lower extremity, the hemiplegic walk may become modified in such numerous ways as to render it impossible to compare the different varieties which may be presented in a single description.

b. Combined Tonic and Clonic Spasms.

In the combined tonic and clonic forms of post-hemiplegic motor disorders the muscular contractions are at first quite like those which occur in late rigidity, but after a time some of the muscles implicated become the subjects of clonic spasm. The combined tonic and clonic spasms of hemiplegic limbs may be divided into (1) intermittent tremor and (2) choreiform movements.

(1) *Intermittent tremor* is produced when the muscles are put upon the stretch by passive movements or voluntary effort. This kind of

tremor is similar to that described as "spinal epilepsy" in primary lateral spinal sclerosis, and, like the tremor of multiple sclerosis, it is absent during repose. The muscles of hemiplegic limbs are also liable to be affected with fibrillary contractions similar to those which occur in progressive muscular atrophy and amyotrophic lateral sclerosis, but in these cases it is probable that the descending changes of the pyramidal tract have extended to the ganglion cells of the anterior gray horns of the cord.

(2) *Choreiform movements* of the extremities may either precede or follow an attack of hemiplegia, the former being named *pre-hemiplegic chorea*, and the latter *post-hemiplegic chorea*. In *pre-hemiplegic chorea* the patient complains of a feeling of numbness and feebleness of the extremities of one side, his gait becomes hesitating and irregular, and the upper extremity of the affected side is attacked by choreiform movements. These symptoms may continue for some days, when complete hemiplegia, usually associated with hemianæsthesia, is either suddenly or gradually established. *Post-hemiplegic chorea* occurs in partially but never in completely paralyzed limbs, and usually appears simultaneously with a marked diminution of the paralytic symptoms. The clonic spasms become, as a rule, gradually established as motor power returns, but they sometimes appear suddenly during a voluntary effort on the part of the patient to move the paralyzed limb. Clonic spasms appear more frequently in the arm than in the leg, and when they exist in both they are generally more severe in the former than in the latter, while if they exist exclusively in the leg, the arm is completely paralyzed. The movements affected by choreiform spasms are, in their increasing order of frequency, the special movements of the fingers and thumb, pronation and supination of the forearm, extension and flexion at the elbow-joint, and movements at the shoulder-joint. The interossei are particularly liable to be affected by choreiform spasm, and consequently the movements most frequently observed consist of varying degrees of flexion and extension at the metacarpo-phalangeal articulations, associated respectively with extension and flexion at the phalangeal articulation. The movements induced by these spasms are of wider range than those of the hemiplegic tremor, resembling in this respect the movements of chorea rather than the tremors of primary lateral spinal sclerosis. The movements are disorderly and irregular, and may or may not continue during repose; they cease during sleep, and become much aggravated during voluntary efforts to perform a definite movement with the affected limb, such as that of raising a glass of water to the mouth. Post-hemiplegic chorea of adults is always accompanied by some degree of anæsthesia, which extends over the

lateral half of the body on the side affected with spasm, and affects all forms of cutaneous and muscular sensibility as well as the special senses.

c. Clonic Spasms.

The post-hemiplegic motor disorders which consist of clonic spasms unaccompanied by tonic contractions of the muscles, are (1) continuous or remittent tremor, (2) choreiform movements (athetosis), and (3) jerking movements on voluntary effort (hemiataxia).

(1) *Continuous or remittent tremor* is continuous during waking hours, and instead of being exaggerated by voluntary effort like the tremors of lateral spinal sclerosis and that of sclerosis in patches, it may be diminished or arrested for a short time by a voluntary effort, being in this and other respects like the tremor of paralysis agitans. The tendon-reactions are not sensibly exaggerated in this form of tremor.

(2) *Athetosis* is a condition in which the patient is unable to maintain the fingers and toes in fixed positions. The fingers and toes are maintained in continuous slow movement, and are made to assume various distorted positions. In some cases these movements extend to the hand and foot, and occasionally even to the muscles of the neck and face. No motor weakness has been observed, the muscles do not become tense on passive movements, and the tendon-reactions are not sensibly exaggerated. The appearance of the clonic spasm is in almost all cases preceded by a distinct attack of hemiplegia, and when no decided paralysis can be ascertained to have been present, the history of the case shows that the patient has suffered from an attack of convulsions and unconsciousness. Hemianæsthesia is described as having been present in a considerable number of the reported cases, and a certain degree of numbness of the affected side is frequently mentioned, while it is possible that diminution of sensibility would have been found in all cases had special attention been directed to this point. The affected extremity usually presents vaso-motor disturbances, being red or livid, moist, and colder than the corresponding extremity. The affected hand or foot is also frequently smaller than the corresponding part of the opposite extremity, although the muscles affected by spasm may undergo a certain degree of hypertrophy. The electric contractility of the affected muscles varies in different cases, being sometimes normal, at other times enfeebled or increased. The ligaments and joints of the affected extremities may occasionally be found considerably relaxed.

(3) *Hemiataxia* consists of disorderly or incoördinate movements of the limb on voluntary effort, coming on in the absence of paralysis,

permanent rigidity, or spontaneous spasm. The ataxic movements become most marked when the patient is asked to execute a complicated movement with the affected limb when his eyes are closed. This condition is associated with a slight degree of anæsthesia, tactile sensibility being sometimes found diminished, while the sensibility to pain is normal.

III. SPECIAL CONSIDERATION OF POST-HEMIPLEGIC SPASMS AS THEY OCCUR IN INFANCY (THE SPASMODIC PARALYSES OF INFANCY).

The spasmodic paralyses of infancy may, from the clinical standpoint, be divided into (1) paralysis of hemiplegic and (2) paralysis of paraplegic distribution, and in addition we shall consider in this place (3) the cases of tonic spasm of voluntary muscles described by Thomsen. It is possible that some of the paraplegiæ of infancy are of spinal origin, but many of them are probably to be regarded as bilateral hemiplegiæ rather than as true hemiplegiæ.

1. Spastic Hemiplegiæ of Infancy.

The spastic hemiplegiæ of infancy may be divided into *a*, *acquired*, and *b*, *congenital* spastic hemiplegiæ. In addition we shall consider in this place *c*, *double athetosis*.

a. Acquired spastic hemiplegia of infancy dates from birth or begins at any time between that date and four or five years of age. The child is unconscious and convulsed for the first few days after birth (asphyxia neonatorum), or if attacked after birth it is suddenly seized with convulsions and unconsciousness which may last from a few hours to a few days. The convulsions are limited to, or at least most pronounced on one side of the body, and this side becomes subsequently paralyzed. In the cases which recover the hemiplegia pursues the usual course, contractures become established, and choreiform movements may or may not make their appearance, and if these movements once make their appearance they remain permanent. So far, then, these cases present nothing peculiar except that the disease dates from childhood, that it is ushered in by convulsions and profound unconsciousness, and that the motor paralysis is not accompanied by hemianæsthesia. But as the patient advances in age the bones of the paralyzed limbs, and even those of the paralyzed half of the face, are somewhat arrested in their development as compared with the corresponding bones of the healthy side, so that the former are found to be smaller in all their dimensions than the

latter. Each of the long bones of the limbs may be from one-fourth of an inch to one inch shorter than the corresponding bones of the healthy side, and even the clavicle of the paralyzed side may be from one-fourth to one-half of an inch shorter than the opposite clavicle. The diminution in size of the half of the face may extend to all the features, including the eyebrow, eyelids, half of the nose, the cheek, and half the mouth. At from seven to fifteen years of age the patient is attacked with epileptic convulsions, which recur at periodical intervals of greater or less duration during the rest of life. These convulsions are at first limited to the paralyzed half of the body, and they are not always accompanied by decided loss of consciousness. After a time, however, the convulsions become general, but even then they often retain a unilateral character at the beginning of the attack, and the patient usually describes a unilateral aura. The aura is often described as a numbness or other sensation beginning in the paralyzed arm, and ascending along the arm to the shoulder and then to the side of the face, when unconsciousness supervenes. At other times the sensation begins in the leg and ascends successively to the arm and side of the head, or in the side of the face and descends to the arm and leg. The intellectual faculties almost always suffer in this disease, and the patient is usually more or less idiotic, but in some cases this defect of intelligence amounts only to a certain degree of imbecility, which becomes established only after a frequent recurrence of the epileptic attacks.

b. Congenital spastic hemiplegia presents numerous varieties, and all of them are generally associated with imbecility or idiocy. In some cases the paralysis is limited to one-half of the body, while at other times both sides are affected. In these cases there is absence of a history of convulsions and unconsciousness occurring at birth or during childhood, and the spasmodic rigidity of the affected limbs is accompanied, as a rule, by disorders of articulation, wry neck, or strabismus, while the skull is often of comparatively small size and unsymmetrical.

c. Bilateral athetosis is generally of congenital origin, and is always associated with more or less idiocy, and of articulatory disorders, while aphasia is sometimes present. The muscles of the face are more liable to be implicated in the spasm of the bilateral than in the unilateral disease. In cases of bilateral athetosis sensory disorders have not been observed, and there is no history of an apoplectic attack or convulsions having occurred during infancy, and consequently the affection is most probably of congenital origin.

2. The Spasmodic Paraplegiæ of Infancy.

Children are liable to be affected with a spasmodic paraplegia from disease of the vertebral column or from any other lesion which causes a consecutive transverse myelitis; but the cases which are under discussion at present occur at birth, or are of congenital origin. In this affection the muscles of the lower extremities are in a condition of spasmodic rigidity, the patellar tendon-reactions are exaggerated, and the feet are generally maintained in a condition of talipes equinus (Figs. 151 and 152) owing to the predominance of the actions of the

Fig. 151.

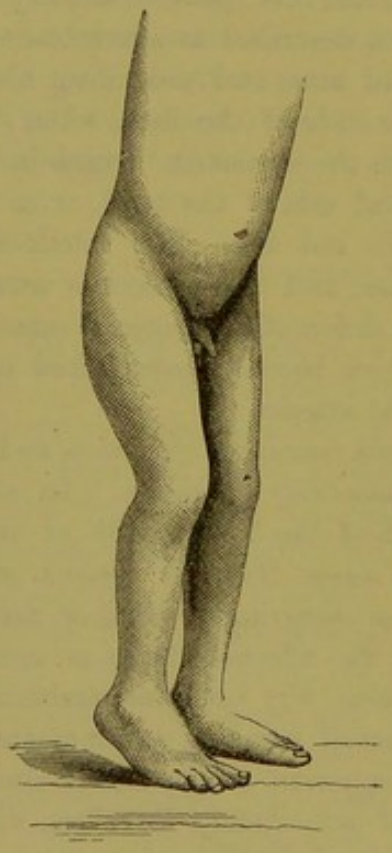
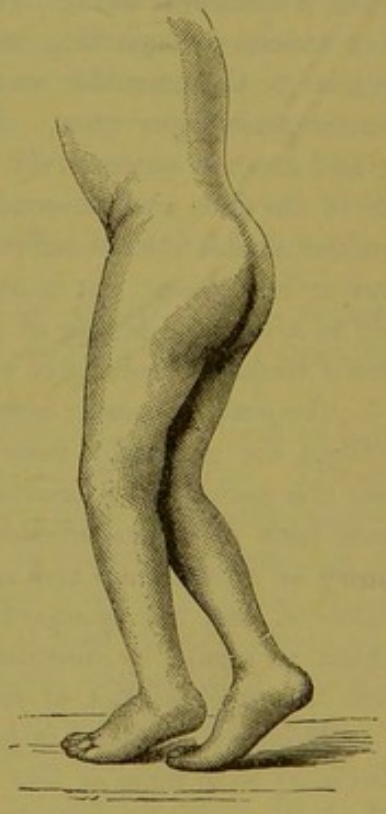


Fig. 152.



muscles of the calves, while the gait of the patient is like that of lateral spinal sclerosis. The upper extremities are not affected, but the patient often suffers from difficulties of articulation, and the head is often rather smaller than is usual in healthy children of the same age. In other cases the head is large with projecting forehead, showing that the patient has suffered in infancy from hydrocephalus or rickets. When the spasm is limited to the lower extremities the patient is often quite bright and

intelligent, and feebleness of intellect is only present in occasional cases, but the patient never appears to suffer from epileptic convulsions.

IV. MONOPLEGIÆ.

The clinical features of the monoplegiæ do not require lengthened description. The paralysis, which is restricted to one limb or to certain groups of muscles on one side of the body, is accompanied by the same kind of muscular tension and excess of the tendon-reactions which are met with in hemiplegia. The clinical varieties most usually observed are crural, brachio-crural, brachial, brachio-facial, facial, labio-glosso, and unilateral oculo-motor monoplegiæ.

The monoplegiæ derive their chief interest not so much from their clinical features as from the localization of the lesions which give rise to them, and consequently they will be more minutely described when we come to discuss the morbid anatomy of the spasmodic paralyses.

Sensory disorders sometimes accompany the paralyses caused by disease of the cortex of the motor area of the brain. The tactile sensibility and the muscular sense are specially liable to be diminished in the paralyzed limbs in cortical disease. Vaso-motor and trophic disorders, consisting of elevation of the temperature of the paralyzed limbs and acute bedsores, have also been observed in cases of disease of the cortex of the brain, but these symptoms do not possess any value in determining the localization of the lesion.

V. PARALYSES FROM FUNCTIONAL DISEASE.

1. Post-epileptic Paralysis.

Attacks of unilateral epilepsy are always followed by a temporary or permanent paralysis of the convulsed limbs, which is attended by some degree of exaggeration of the tendon-reactions, showing that this form of paralysis belongs to the spasmodic or cerebro-spinal variety. The general convulsions of idiopathic epilepsy are followed by a general resolution of the limbs which is not usually spoken of as a paralysis because all the limbs are affected in an equal degree. This muscular feebleness sometimes predominates on one side of the body or in one limb, and then it is recognized to be of a distinctly paralytic nature, and there can be little doubt that the general muscular relaxation which follows an epileptic attack ought also to be regarded as a paralysis.

2. Hysterical Paralysis.

Partial or complete loss of muscular power is a frequent symptom of hysteria. The loss of motor power may begin with mere weakness and heaviness of the limbs, and these symptoms may gradually increase until complete paralysis is established, but at other times complete paralysis is suddenly established after a hysterical attack. The paralysis may assume the form of paraplegia, hemiplegia, monoplegia, or it may be limited to one or more motor nerves or to particular branches of a nerve.

Hysterical hemiplegia often supervenes after an attack of convulsions with partial loss of consciousness, so that in its mode of onset as well as in its clinical features it resembles the hemiplegia which results from an apoplectic seizure, and the fact that *contractures* become developed in the former as well as in the latter, renders the similarity between them still more striking.

In some cases the contracture appears simultaneously with the paralysis, while in other cases a paralysis with flaccidity is established and continues for some time and then contracture develops gradually or suddenly after a fresh attack. In the upper extremity the forearm, hand, and fingers are spasmodically flexed, and the muscles may be so rigid that it is impossible to obtain complete extension or to increase the flexion. The lower extremity is strongly extended upon the pelvis and the leg upon the thigh, while the foot assumes the position of extreme talipes equino-varus. In hysterical paraplegia both lower extremities assume the position just described, and the knees are drawn inwards and strongly pressed against each other by contraction of the adductors of the thighs. In hysterical paralysis with contracture, tremor is sometimes observed on attempting any movement. The patellar tendon-reactions are often exaggerated and even ankle-clonus may sometimes be elicited, these being the symptoms which are so characteristic of the paralysis which results from sclerosis of the lateral columns of the cord. Hysterical hemiplegia differs from hemiplegia due to organic disease of the brain in the following respects: It is usually accompanied by hemi-anæsthesia, and by emotional disturbances, and other well-marked hysterical phenomena; the paralysis is scarcely ever complete, the face and tongue almost never being affected; the leg is generally more paralyzed than the arm, and the diminution of motor power is liable to sudden variations of intensity under the influence of different emotions. Hysterical paralysis may be distinguished from atrophic paralyses by the facts that in it the electrical reactions of the muscles remain normal, and the muscles do not undergo active wasting. These tests, however,

fail in distinguishing hysterical paralysis from Landry's paralysis, and as a matter of history the latter has frequently been mistaken for hysterical paralysis. The most trustworthy diagnostic sign between the two affections is afforded by the patellar tendon-reaction, which is generally exaggerated in hysterical paralysis and absent in Landry's paralysis.

For purposes of diagnosis the student should pay particular attention to the features which distinguish the gait of a person suffering from hysterical paralysis and that of one suffering from a spasmodic paralysis caused by organic disease. In organic spasmodic paralysis, whether of the hemiplegic or paraplegic variety, when the affected extremity is to be moved forward in walking the heel becomes strongly elevated and the toes depressed; the necessary elevation to clear the toes off the ground is obtained chiefly by elevation of that side of the pelvis; and the foot is projected forward by a movement of circumduction, the toes making a scraping noise on the ground. In hysterical paralysis there is little or no elevation of the heel or depression of the toes during the forward movement of the foot; the necessary elevation to clear the foot off the ground is obtained by flexion of the limb at the hip- and knee-joints, and consequently rotation of the pelvis is not a marked feature of the walk; there is no movement of circumduction, and the toes do not scrape the ground. In hysterical paralysis the foot is maintained throughout the whole forward movement at right angles with the leg, and as it is made to glide forwards the sole is held close to and almost parallel with the ground.

The relative feebleness of the extensors of the toes in comparison with those of the heels in spastic paralysis from organic disease, may be tested in another way. If the patient be placed with her back against a wall, so as to prevent her from inclining the body forwards, it will be found that when she attempts to elevate the toes a spasm of the muscles of the calf supervenes, which fixes the anterior part of the foot still further to the ground, but in hysterical paralysis the patient may often, although not always, be coaxed to elevate her toes just like a healthy person, and even when she refuses to make the necessary effort it will be found that the anterior part of the foot does not become so strongly fixed to the ground as in organic paralysis. In organic spasmodic paralysis of the lower extremities the reflex of the sole is exaggerated, while it is often absent when the paralysis is hysterical. In spasmodic paralysis of the lower extremities from organic disease the patient fixes her eyes steadily on the ground, but the hysterical patient either looks about her, or, while pretending to look at the ground, she casts stealthy

glances from the corners of her eyes to see what effect she is producing on the surrounding observers.

Of the paralyses which occur in the region of distribution of particular nerves, hysterical aphonia is probably the most frequently met with. In hysterical aphonia the loss of voice appears suddenly after some mental excitement, and may disappear with equal celerity, and it is not attended by a persistent cough like laryngitis. A laryngoscopic examination reveals the signs of paralysis of the glottis, sometimes on one and at other times on both sides. Difficulty of moving the tongue is sometimes associated with the laryngeal paralysis, and the patient is no longer able to articulate in a whisper, but has to resort to pantomime in order to make herself understood. Paralysis of the muscles of the pharynx and œsophagus is not an uncommon symptom of hysteria, and swallowing then becomes difficult or impossible. In such a case the œsophageal tube passes into the stomach without encountering any obstruction. Inspiratory dyspnœa is a frequent symptom of hysteria, and it is possible that it is sometimes caused by a temporary paralysis of the diaphragm. Paralysis of the muscular coat of the stomach is partly the cause and partly the effect of the general tympanites which is so frequently met with in hysterical patients. The obstinate constipation which is so frequent a symptom of hysteria is probably caused by paralysis of the muscular coat of the bowel. Retention of urine is common in hysteria, but it is not often of paralytic origin. Hysterical women have been known to drop down dead after a great shock, and in these cases death is doubtless caused by paralysis of the heart. Various phenomena occur in hysterical patients from paralysis of the vaso-motor nerves. The excessive menstruation, and even the hæmoptysis and hæmatemesis which are occasionally observed are most probably caused by paralysis of vaso-motor nerves.

3. Toxic Paralyses.

Various poisons give rise, as we have already seen, to paralysis by causing organic disease of the nervous system. Chronic poisoning by lead and by alcohol, as well as the poison of diphtheria, cause a paralysis which we have reasons for believing is due to a progressive multiple neuritis, and it is very probable that arsenic and many other metallic salts act in a similar manner. Other poisons cause paralysis by inducing a molecular or functional change in peripheral nerves, such as curara, which acts upon the intramuscular nerve-endings, and atropine, which paralyzes the terminations of the vagi and the nerves which

supply the ciliary muscle and the sphincter of the iris. Some of the methyl compounds of conium produce paralysis by depressing the irritability of the gray matter of the spinal cord and of the motor nerves. This subject is, however, so wide that were it to be fully discussed we should have to go over nearly the whole field of toxicology.

Some poisons appear to act on the fibres of the pyramidal tracts. Cases of spasmodic spinal paralysis investigated by Dr. Brunelli render it probable that the symptoms were caused by a species of vetch—lathyrus cicera—which was used by the patients as a chief article of diet.

A case I saw a short time ago along with my friend, Dr. Niven, would seem to show that the symptoms of spastic spinal paralysis may be caused by malaria. The patient came home an invalid from South America some weeks before I saw him. When I examined him he was suffering from the usual symptoms of a primary lateral sclerosis, consisting of stiffness and feebleness of the lower extremities, muscular tension, exaggerated patellar tendon-reaction, ankle-clonus, and decided spastic gait. Iodide of potassium was ordered, but the symptoms did not improve much. Dr. Niven now ascertained that the patient was suffering from well-marked attacks of ague, and he was accordingly treated with large doses of quinine, and not only did the febrile symptoms disappear, but the spinal symptoms also improved rapidly, and the patient left for South America, two months afterwards, with scarcely a trace of the stiffness or exaggerated tendon-reactions of the lower extremities to be detected.

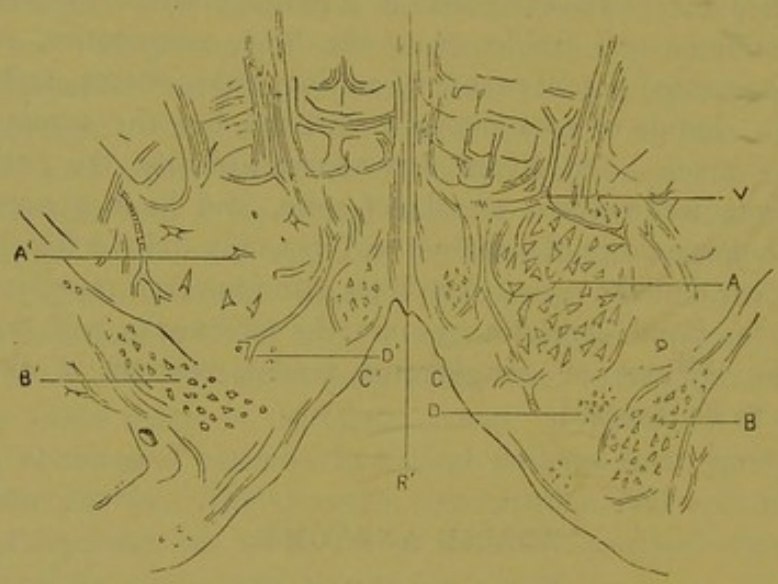
MORBID ANATOMY.

I. Paraplegia.

Some pathologists doubt whether such a disease as a primary symmetrical sclerosis of the lateral columns has yet been proved to exist. Cases have been reported in which the symptoms attributed to primary lateral sclerosis were present during life, but which proved on post-mortem examination to have been examples of sclerosis in patches, or tumor of the medulla oblongata, or of the cerebellum. The most notable case of primary lateral sclerosis so far reported is that of a man under the care of my colleague, Dr. Morgan, in the Manchester Royal Infirmary. The patient died from some intercurrent disease, and Dr. Dreschfeld, who conducted the microscopical examination, found symmetrical sclerosis of the pyramidal tracts of the spinal cord

from the medulla oblongata to the conus medullaris, and, with the exception of slight atrophy of the ganglion cells of the anterior gray horns, an entire absence of any other lesion. The presence of some atrophy in the ganglion cells in this case has led Westphal to suggest that it was an example of amyotrophic lateral sclerosis, but having watched the symptoms during life, and examined sections of the spinal cord, I do not concur in this view. A secondary symmetrical sclerosis of the lateral columns is caused by all transverse lesions of the spinal cord. Transverse and compressive myelitis are followed by a descending sclerosis of the antero-lateral pyramidal tracts below the level of the lesion, and an ascending sclerosis of the columns of Goll and direct cerebellar tracts, but these conditions have already been sufficiently described.

Fig. 153.



Transverse Section of the Medulla Oblongata on a level with the middle of the Nucleus of the Hypoglossal. (From Charcot.)

R, R', median raphé; A, B, represents the normal condition, and A', B', the parts as they appear in amyotrophic lateral sclerosis; C, C', the floor of the fourth ventricle; V, a vessel which bounds the nucleus of the hypoglossal anteriorly and externally; D, fasciculus teres? and D', the corresponding part on the diseased side; A, healthy nucleus of the hypoglossal; and A', the diseased nucleus. B, the healthy nucleus of the pneumogastric nerves; and B', the nucleus on the diseased side, which is seen not to be much affected.

Morbid Anatomy.—In amyotrophic lateral sclerosis Charcot and others have proved the presence of symmetrical sclerosis of the pyramidal tracts of the antero-lateral columns of the cord, and of the anterior pyramids of the medulla oblongata, along with degenerative atrophy of the anterior gray horns and of the motor nuclei in the medulla oblongata, and loss of the motor ganglion cells. The condition of the bulbar nuclei is represented in Fig. 153, borrowed from

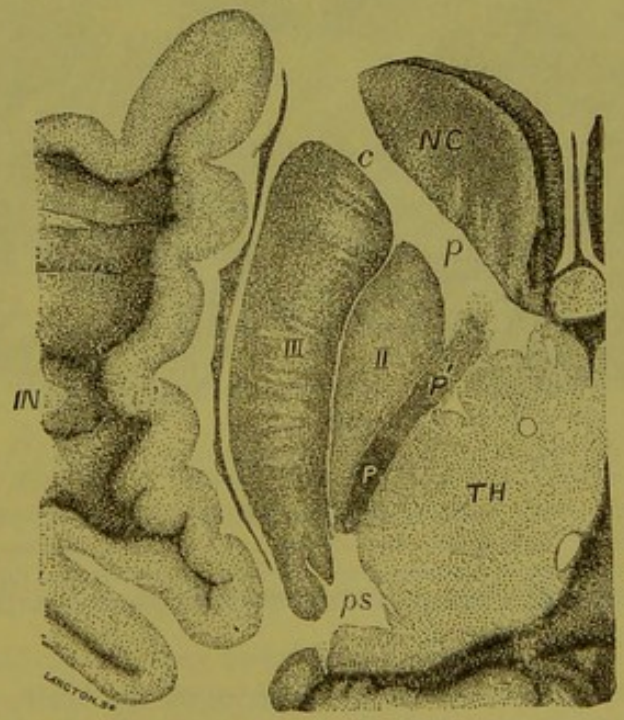
Charcot, the diseased nuclei being shown to the left of a fictitious line (R R′), and the healthy one for the sake of comparison to the right of that line. The part which Charcot calls the fasciculis teres really consists of a group of small cells which I have called the external accessory nucleus of the facial. It is seen to be diseased on the left side (D′) of the figure.

II. Hemiplegia.

1. *Ordinary Hemiplegia.*

The fibres which issue from the motor ganglion cells of the cortex form separate bundles in the centrum semiovale, and descending in the corona radiata come together in the internal capsule. Speaking somewhat broadly, it may be said that the fibres of the middle third of the posterior segment of the internal capsule (Fig. 154, *P*) are concerned

Fig. 154.



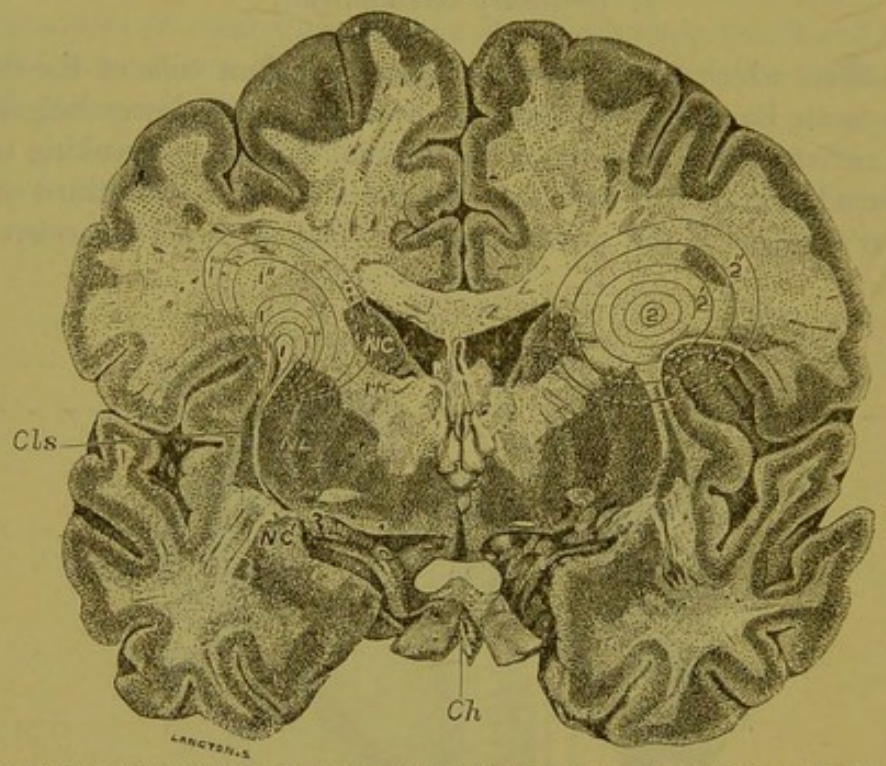
Horizontal Section of the Basal Ganglia and Internal Capsule in an embryo of nine months.

NC, Caudate nucleus; *TH*, Optic thalamus; *IN*, Island of Reil; *II*, *III*, Second and third segments of the lenticular nucleus; *ps*, Sensory peduncular tract; *P*, Fundamental, and *P′*, Mixed portion, and *p*, Geniculate fasciculus of the pyramidal tract; *c*, Anterior segment of the internal capsule.

in regulating the actions of the trunk, lower extremities, and probably the general actions of the upper extremities; that the fibres of the anterior third of the posterior segment (Fig. 154, *P′*) are concerned in

regulating the special movements of the hand as an organ of prehension, and probably also the movements of the rotation of the head and neck along with the associated ocular movements; and that the fibres of the knee (Fig 154, *P*) and the adjoining part of the anterior segment of the capsule are concerned in the regulation of the movements of facial expression, articulation, and the most special movements of the hand, such as those of writing. The posterior third of the posterior segment of the capsule contains the sensory conducting paths, and injury of these

FIG. 155.



VERTICAL SECTION OF THE BRAIN A LITTLE BEHIND THE KNEE OF THE INTERNAL CAPSULE, SHOWING THE EFFECTS OF RUPTURE OF THE LENTICULO-STRIATE ARTERY. (Modified from CHARCOT.)

NC, Head, and *NC'*, Tail of the caudate nucleus; *Ch*, Chiasma, *NL*, Lenticular nucleus; *IK*, Internal capsule; *Cls*, Claustrum; 1, The most frequent position in which the lenticulo-striate artery is ruptured; 1′, 1″, 1‴, Progressive extension of the hemorrhage producing compression and rupture of the fibres of the pyramidal tract (hemiplegia); 2, Primary focus in the internal capsule; 2′, 2″, 2‴, Successive extension of the clot.

causes hemianæsthesia. Of all the arteries of the brain, the lenticulo-striate artery is the one most liable to rupture. If the hemorrhage be small, it may lodge between the external capsule and the lenticular nucleus and give rise to no symptoms. The vessel, however, is a comparatively large one, and the hemorrhage usually extends beyond these limits. It is sometimes directed upwards between the external capsule and the lenticular nucleus, and it may then extend for a considerable distance with the centrum ovale, the fibres of the internal capsule become rup-

tured at the point where they form the foot of the corona radiata, and the hemorrhage may then be so extensive as to extend upwards to the summits of the ascending frontal and parietal convolutions, and outwards so as to compress the Island of Reil, but the external capsule is only rarely ruptured. At other times the hemorrhage is directed inwards through the gray matter of the lenticular nucleus, and if it remain limited to the nucleus the patient will recover almost completely from the hemiplegia which follows the attack. But if the clot is large enough to rupture the internal capsule (Fig. 155, 1″), the fibres below the seat of the lesion undergo descending sclerosis, and the paralysis remains more or less permanent. In descending sclerosis the morbid changes are observed in the middle third of the crusta, the longitudinal bundles of the pons, the anterior pyramid of the medulla oblongata and the column of Türck on the side of the lesion, and in the lateral column of the cord on the side opposite to the lesion. In some cases a large hemorrhage may rupture into the lateral ventricle and then death takes place in a few hours.

Rupture of the lenticulo-striate artery gives rise to a hemiplegia in which the arm is more paralyzed than the leg or face. If one of the anterior branches of the artery is the seat of rupture the anterior segment of the capsule may be injured to a greater degree than the posterior segment, and then paralysis of the face predominates. Hemorrhage of the caudate nucleus may, by pressing on the internal capsule, cause a hemiplegia in which the face is more paralyzed than the arm or leg.

2. *Hemiplegia and Hemianæsthesia.*

Hemorrhage of the lenticulo-optic artery (Fig. 154) impinges against the posterior part of the posterior segment of the capsule, and causes hemiplegia in which the leg is more paralyzed than the arm or face, and which is accompanied by hemianæsthesia.

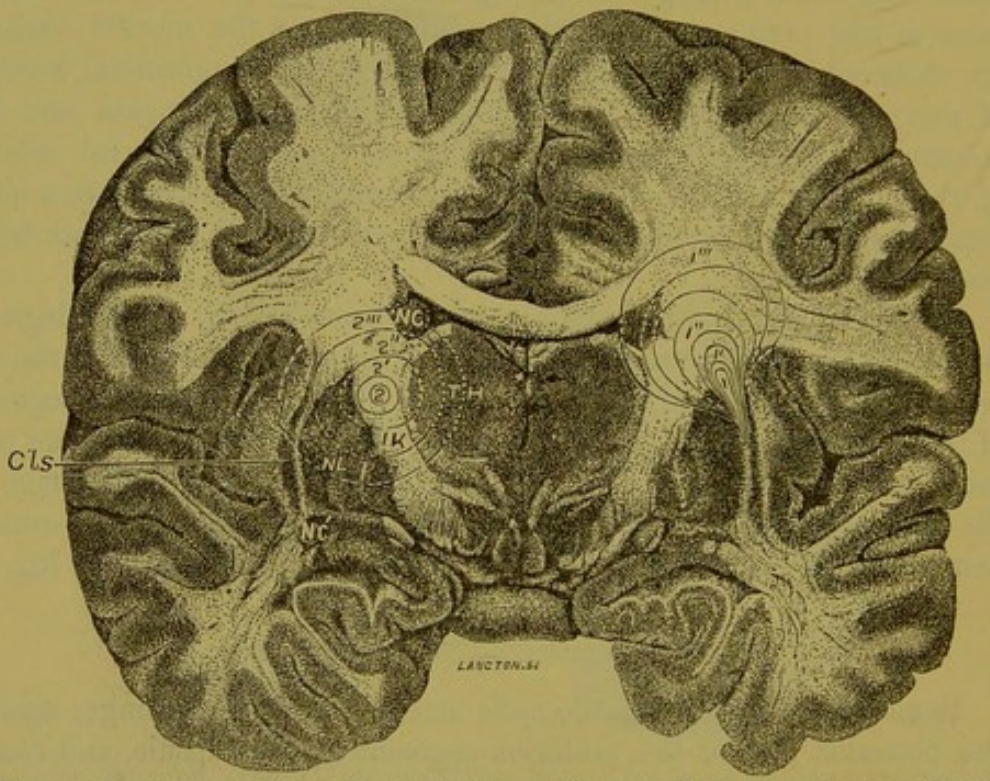
3. *Hemiplegia, Hemianæsthesia, and Hemianopsia.*

The internal capsule may be injured by lesions of the optic thalamus. Hemorrhage of the posterior internal optic artery, if small, does not give rise to any definite symptoms, but if large it ruptures into the lateral ventricles and causes death in a short time. Hemorrhage from the posterior external optic artery does not usually give rise to pronounced hemiplegia, but it causes hemianæsthesia of the opposite side from injury of the sensory peduncular fibres of the optic radiations of Gratiolet. When the lesion occurs in the pulvinar the external geniculate body is apt to be implicated and then bilateral hemianopsia of the

opposite side results along with hemiplegia and hemianæsthesia, and the same symptoms have occasionally been observed when the lesion is situated too far forwards to injure the external geniculate body.

When a large hemorrhage occurs in the area of distribution of the posterior external optic artery, the effused blood ruptures the posterior part of the internal capsule, either wholly or partially, and makes its way inwards and backwards so as to tear up a considerable portion of the anterior tubercle of the corpora quadrigemina. It is in such cases

FIG. 156.



VERTICAL SECTION OF THE BRAIN ON A LEVEL WITH THE POSTERIOR PART OF THE INTERNAL CAPSULE, SHOWING THE EFFECTS OF RUPTURE OF THE LENTICULO-OPTIC ARTERY (HEMIANÆSTHESIA). (Modified from CHARCOT.)

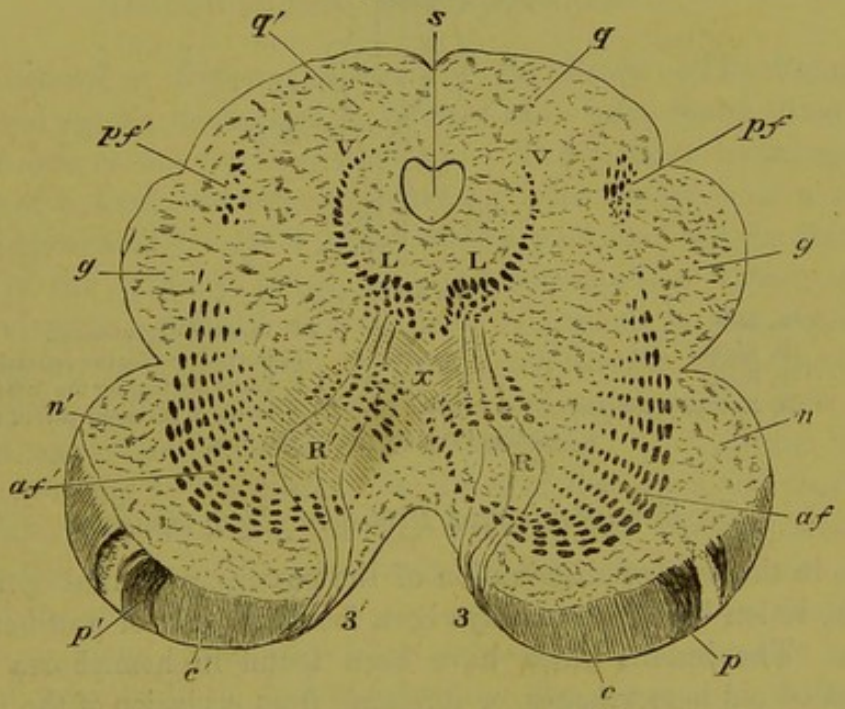
NC, NC', Head and Tail of the caudate nucleus; *NL*, Lenticular nucleus; *TH*, Optic thalamus; *Cls*, Claustrum; 1, Primary focus in the posterior part of the external capsule (hemianæsthesia); 1', 1'', 1''', Progressive extension of the primary focus causing compression or destruction of the internal capsule; 2, Primary focus in the internal capsule (hemianæsthesia); 2', 2'', 2''', Successive extension of the focus.

that the hemiplegia and hemianæsthesia with the peculiar disorder of the movements of the eyeballs already described have been observed. The clot in these cases is only separated from the general ventricular cavity by a thin layer of tissue, consisting chiefly of the ependyma of the ventricle, and under such circumstances it is very probable that some serous exudation will take place into the ventricles. The passage of this fluid through the aqueduct of Sylvius into the fourth ventricle may possibly explain the presence of Cheyne-Stokes respiration in these cases.

4. *Crossed or Alternate Hemiplegia.*

When the lesion is situated in the crus cerebri the fibres of the third nerve in their passage from the nucleus to their point of emergence are damaged, while the fibres of the pyramidal tract in the middle third of the crusta are ruptured or compressed (Fig. 157, *p*). The consequences of a lesion in this position are that the muscles innervated by the third nerve are paralyzed on the side of the lesion, while the limbs

Fig. 157.



Crura Cerebri. Transverse Section of the Crura Cerebri on a level with the anterior pair of the Corpora Quadrigemina: from a nine-months human embryo. The dark portions represent Medullated Fibres.

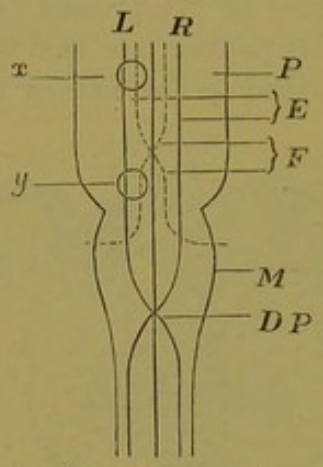
s, Aqueduct of Sylvius; *q*, *q'*, Anterior pair of Corpora Quadrigemina; *pf*, *pf'*, Fasciculi of Medullated Fibres proceeding to the anterior pair of Corpora Quadrigemina; L, L', Posterior Longitudinal Fasciculi; V, V', portions of these Fasciculi which join the posterior commissure of the third ventricle; *g*, *g'*, External Geniculate Bodies; *af*, *af'*, Anterior portion of Fillet; *n*, *n'*, Substantia Nigra; R, R', Red Nuclei; *p*, *p'*, Pyramidal Tract; *c*, *c'*, Crustæ; 3, 3', Third pair of nerves; *x*, Decussation in front of the Aqueduct of Sylvius, which is part of the interlacement of the Tegmentum.

are paralyzed on the opposite side of the body, because the pyramidal tract is injured above the crossing of the fibres of the two sides in the medulla oblongata.

If the lesion is situated in the lower part of the lateral half of the pons there is well-marked facial paralysis on the side of the lesion, and a more or less complete motor and sensory paralysis of the limbs on the opposite side. In order to account for these phenomena it is necessary

to assume that the fibres of the pyramidal tract, which connect the cortex of the opposite hemisphere with the nucleus of the facial nerve in the upper part of the medulla, cross over about the middle of the pons, as represented in Fig. 158.

FIG. 158.



(From NOTHNAGEL.)

L, Left.
R, Right.
P, Pons.
M, Medulla oblongata.
DP, Decussatio pyramidum.
E, Nerve fibres for the extremities.
F, Fibres destined for the facial nerve.
x, Lesion in the upper part of the pons.
y, Lesion in the lower part of the pons.

5. *Hemiplegic and Post-hemiplegic Spasms.*

It is in the area of distribution of the posterior external optic artery that the lesion has almost always been found in cases of post-hemiplegic chorea. The lesions which have been found in hemichorea are the remains of old hemorrhages, or softening from occlusion of the posterior external optic artery, although choreiform movements have occasionally been observed during the growth of tumors in this region. It is evident, therefore, that the symptoms depend not upon the nature of the lesion, but upon its localization. In three cases of unilateral athetosis observed by Charcot the lesion was situated in the posterior extremity of the optic thalamus in one, the posterior extremity of the caudate nucleus in the second, and the posterior part of the corona radiata in a third. In a case observed by Landouzy an old focus of softening was found in the portion of the lenticular nucleus which adjoins the internal capsule, and in one reported by Ringer a focus of softening was found in the posterior part of the lenticular nucleus near to the internal capsule. In a case observed by Gnauck the coexistence of sensory disturbances in the region of distribution of the fifth nerve on the side opposite to the spasmodic movements rendered it probable that the lesion was situated

in the lateral half of the pons. It is, therefore, probable that the lesion in athetosis may occupy different positions in the vicinity of the pyramidal tract. In all the cases hitherto examined after death the positions occupied by the lesion render it probable that the fibres of the pyramidal tract are never completely ruptured and that there are no descending changes in the cord, but the fibres of the tract are likely to have suffered partial damage.

III. Special Consideration of Lesions of the Pyramidal Tracts as they Occur in Infants.

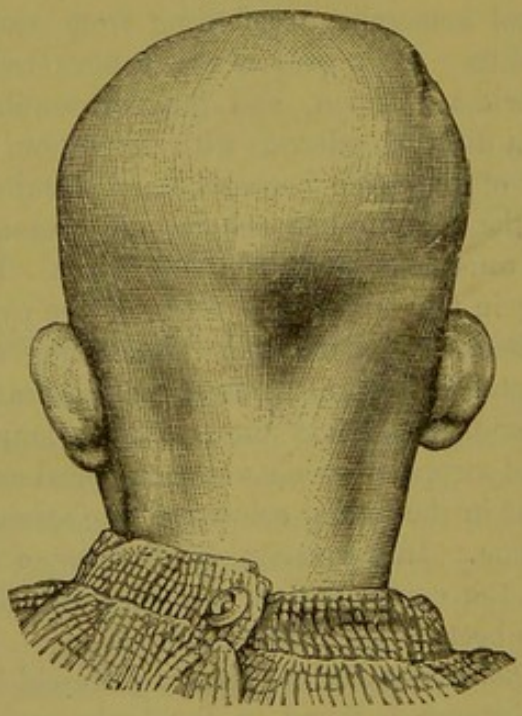
1. *Unilateral or Diffused Atrophy of the Brain.*—The lesions in the acquired spastic hemiplegia of infancy have been found situated in the motor area of the cortex of the brain. The primary lesion appears to consist of a local hemorrhage, softening from occlusion of a vessel, or a local encephalitis. As a general rule a puckered cicatrix forms at the seat of the primary lesion, and the surrounding portion of the cortex undergoes a diffused sclerosis with retraction, so that the motor area of the cortex of that side becomes much smaller than the corresponding area of the opposite hemisphere, and consequently such cases have been named *unilateral atrophy of the brain.* In addition to the changes occurring in the cortex, the fibres of the pyramidal tract connected with the diseased area undergo a descending sclerosis, and consequently the crusta of the crus cerebri, the longitudinal fibres of the pons, and the anterior pyramid of the medulla oblongata on the side of the lesion are found atrophied, while a microscopical examination reveals a patch of sclerosis in the lateral column of the spinal cord on the side opposite to the lesion. In a considerable number of the reported cases the lateral lobe of the cerebellum on the opposite side to that of the cerebral lesion has been found atrophied.

2. *Parencephalus* is a name first given by Heschl to the condition in which a loss of substance is found in the brain, and which is usually filled with serous fluid. The cavity is generally situated on the surface of the cerebral hemisphere; on one side it may open into the arachnoid sac, or be separated from it by the visceral arachnoid, and on the other side it may penetrate the whole extent of the medullary substance so as to reach the ependyma or to communicate freely with the lateral ventricle. The defect may be only of small extent, but it is sometimes associated with hydrocephalus, and it may then extend over the whole mantle of the hemispheres as far as the basal ganglia, while all intermediate sizes may be found between these extremes. The

cerebral defect may be acquired or congenital. The acquired varieties may occur at all ages, and they are chiefly caused by embolism or hemorrhage, although they may occasionally result from injury. The lesion may be situated in any area of the cerebral hemisphere, but in the majority of cases it occupies the area of the Sylvian artery, and it is then accompanied by descending degeneration of the corresponding pyramidal tract. Congenital parencephalus may be caused either by a destructive lesion or by an arrest of development.

When the cavity has been caused by a destructive lesion its walls are formed of cicatricial tissue, and it is probable that evidences of sclerosis of the pyramidal tracts of the cord will be found on microscopical examination, but this point has not been fully investigated in the recorded cases. In the congenital variety, caused by arrest of

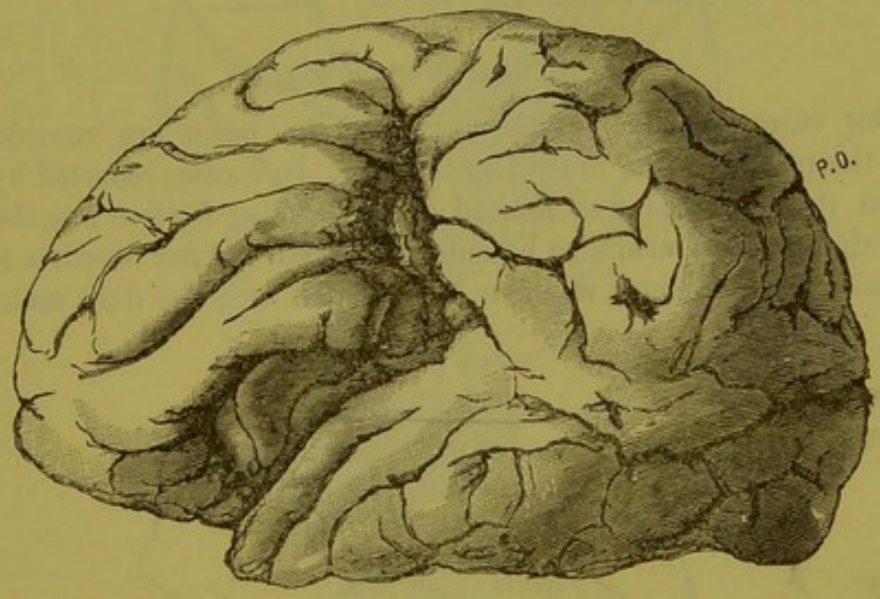
FIG. 159.



development of the brain, the walls of the cavity are formed by the ependyma of the lateral ventricles, and by the gray matter, which to the naked eye has all the appearance of the healthy cortex, but showing deficiency of the motor ganglion cells on microscopical examination. In some cases a defect in the parietal bone is present immediately over the defect in the brain. A case of this kind was examined at a post-mortem by Meschede, and I have recorded a case of defect of the parietal bone (Fig. 159) associated with congenital hemiplegia of the opposite half of the body, and in which a parencephalous defect most probably existed

immediately underlying the osseous defect. In parencephalus from arrest of development a cavity often exists at symmetrical points in each hemisphere, and the antero-lateral columns of the cord, instead of being in a state of sclerosis, are small in size and defective in development, as in a case recorded by myself (Fig. 160). When the parencephalous defect is unilateral the anterior column of the cord on the same side and the lateral column on the opposite side are much less in size than the corresponding columns the fibres of which are derived

FIG. 160.



from the healthy side. This fact was well illustrated in a case of parencephalus shown by Dr. Shuttleworth, of the Albert Asylum, at a meeting of the Manchester Medical Society. It is very likely that bilateral athetosis, and other cases of infantile motor disorders associated with idiocy, are caused by a parencephalous defect of one or both hemispheres of the brain.

Very little is known with regard to the morbid anatomy of the spastic paraplegiæ of infancy. In most of these cases the spastic condition of the extremities is congenital, and generally associated with difficulties of articulation and other evidences of disordered cerebral function, and consequently it is probable that the symptoms are caused by a small parencephalous defect in each hemisphere of the brain.

IV. MONOPLEGIA.

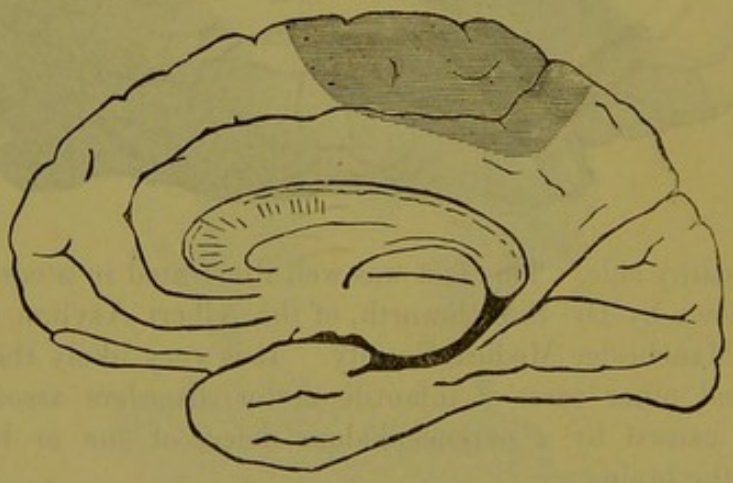
It has just been seen that ordinary hemiplegia is caused by destructive lesions in or near the fibres of the pyramidal tract, as they lie

near together in the internal capsule. In the corona radiata these fibres form more or less separate bundles corresponding to the motor centres in the cortex, so that destructive lesions of the centrum semi-ovale or of the cortex, provided the whole thickness is destroyed, may injure one of these bundles whilst leaving the others intact, and consequently localized lesions in these positions give rise to a monoplegia. It must, however, be remembered that extensive lesions of the cortex of the motor area of the brain will cause a complete hemiplegia which differs scarcely in any respect from the hemiplegia which results from disease of the internal capsule.

1. *Crural Monoplegia.*

A few cases of disease of the cortex of the brain are recorded in which the paralysis was limited to the leg. In a case reported by Dr. Huddon the paralysis was limited to the left leg for five months, but after a time the left arm also became paralyzed. After death a tumor three inches in diameter was found connected with the dura mater; it

FIG. 161.



was situated to the right of the middle line and compressed the subjacent hemisphere, and destroyed the upper extremities of the ascending frontal and parietal convolutions and the postero-parietal lobule (Fig. 162) as well as the paracentral lobule (Fig. 161).

2. *Brachio-crural Monoplegia.*

Paralysis of the leg and arm are frequently associated in disease of the cortex. A case of paralysis, with rigidity of the leg and arm, of three years' duration, is reported by Charcot and Pitres, in which a patch of softening was found at the upper extremity of the fissure of Rolando on the convex surface of the right hemisphere. Pitres de-

scribes a case of paralysis with unilateral convulsions of the left extremities, in which a focus of softening was found not in the cortex, but

FIG. 162.

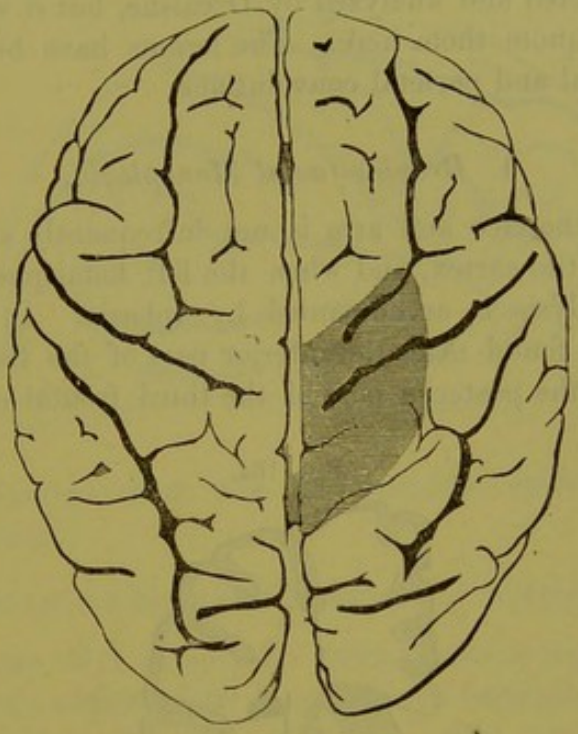
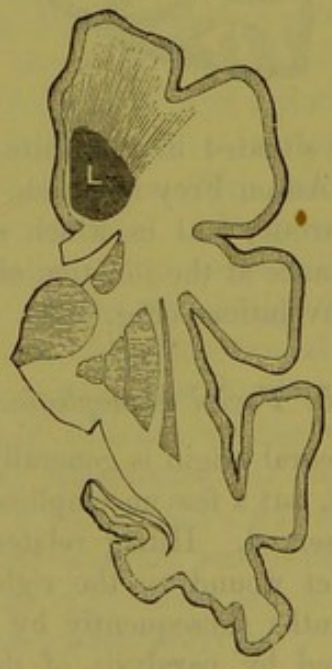


FIG. 163.



in the centrum ovale, immediately beneath the posterior extremity of the first frontal convolution (Fig. 163), and extending backwards underneath the superior parietal lobule.

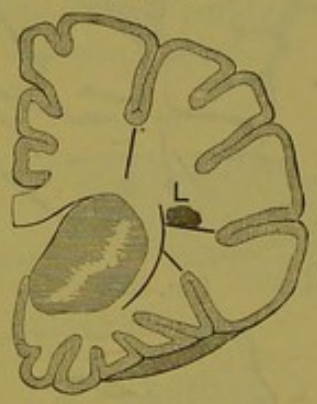
3. *Brachial Monoplegia.*

A large number of cases of brachial monoplegia from cortical disease have been collected and analyzed by Decaisne, but it would occupy too much space to quote them here. The lesions have been found in the ascending frontal and parietal convolutions.

4. *Brachio-facial Monoplegia.*

Paralysis of the face and arm is not infrequently associated in disease in or near the cortex, and when the left hemisphere is the seat of lesion the paralysis is accompanied by aphasia. In these cases the lesion has been found near the inferior part of the fissure of Rolando on a level with the posterior part of the third frontal convolution. In

FIG. 164.



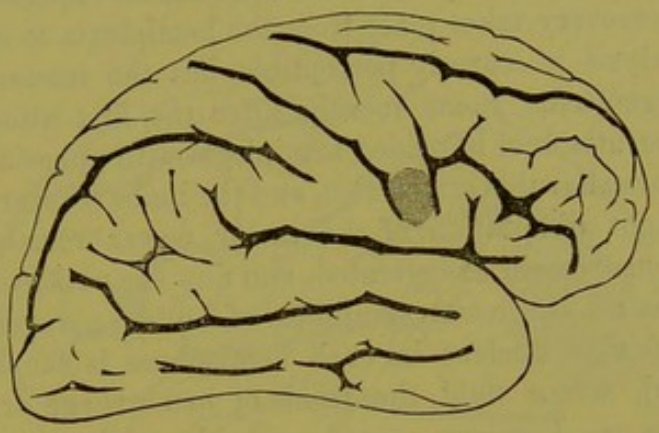
other cases the lesion is situated in the white substance. A case, for example, is reported by Anton Frey in which the left arm and the left side of the face were paretic, and in which a focus of softening was found in the white substance at the junction of the middle frontal with the ascending frontal convolutions (Fig. 164).

5. *Facial Monoplegia.*

Facial paralysis of cerebral origin is generally complicated by aphasia and paralysis of the arm, but a few uncomplicated cases of this form of paralysis have been observed. Hitzig relates the case of a soldier who had received a bullet wound on the right side of the head, and became affected two months subsequently by clonic spasm of the left side of the face, followed by paralysis of those muscles and of the left half of the tongue. After death an abscess was found corresponding to the seat of the injury in the ascending frontal convolution be-

tween the paracentral fissure and the fissure of Rolando (Fig. 165). This case and others show that the lesion in facial monoplegia of corti-

FIG. 165.



cal origin is situated in or near the posterior extremity of the third frontal convolution.

6. *Labio-glosso-laryngeal Paralysis of Cerebral Origin.*

The symptoms of a progressive labio-glosso-laryngeal paralysis are sometimes closely simulated by lesions of the hemispheres of the brain. Dr. Barlow reports the case of a boy, aged ten years, who was suffering from aortic regurgitation, and had an attack of right hemiplegia and aphasia, from which he made a good recovery. Four months afterwards he had an attack of left hemiplegia with aphasia, and in addition the muscles of mastication and articulation and those concerned in the first act of deglutition, were paralyzed. The patient died of the aortic disease, and at the autopsy evidence of an embolus was found in both Sylvian arteries. A focus of softening, about the size of a shilling, was found in the cortex of each hemisphere, involving the inferior extremity of the ascending frontal, and the posterior extremities of the second and third frontal convolutions. In a case reported by Magnus and quoted by Romberg, not only were the symptoms of bulbar paralysis present, but the upper facial muscles were likewise paralyzed, and after death only one hemorrhagic cyst, which was about the size of a small walnut, was found, the situation of this cyst evidently being the centrum semiovale of the right hemisphere immediately underlying the posterior part of the third frontal convolution. Several cases of bulbar paralysis of cerebral origin have now been reported in which the lesions were found in the lenticular nucleus of both hemispheres. Bulbar paralysis occurring in sclerosis in patches is sometimes caused by a patch of sclerosis in each lenticular nucleus,

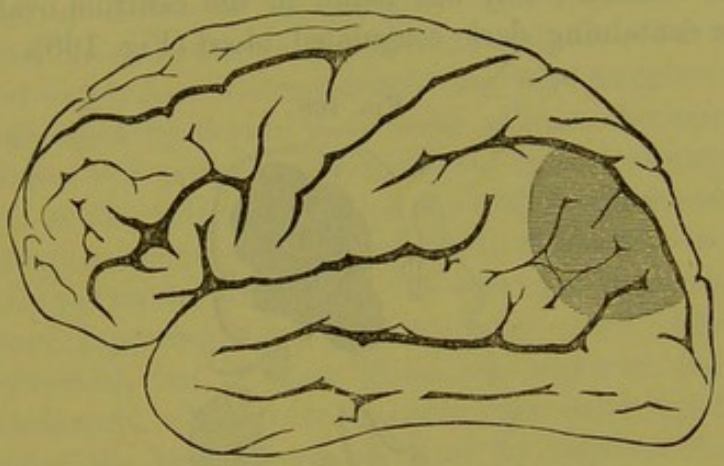
but these symptoms when of cerebral origin usually occur in those who are suffering from granular kidney, or from syphilis of the cerebral vessels. The general history of such cases is that the patient has an attack of right-sided hemiplegia with aphasia, the aphasia rapidly disappears and recovery takes place from the hemiplegia to such an extent that the paralysis is scarcely perceptible, but the tendon-reactions are slightly exaggerated. Some months after the first attack the patient suffers from an attack of left-sided hemiplegia with aphasia; the aphasia soon disappears after this attack also, and the limbs recover, so that there is only a slight diminution of the motor power remaining, but the tendon-reactions remain exaggerated, and now the patient is found to be suffering from all the usual symptoms of bulbar paralysis. In these cases the lenticular nucleus in each hemisphere is found to contain a cyst filled with serous fluid, the result of hemorrhage or of softening. The symptoms are, however, caused, probably not by the disease of the lenticular nuclei themselves, but by partial damage of the fibres of the pyramidal tract which pass on each side through the knees of the internal capsule and which connect the posterior part of the third frontal convolution with the bulbar motor nuclei.

7. *Unilateral Oculo-motor Monoplegia.*

It has already been mentioned that conjugate deviation of the eyes and rotation of the head and neck are frequent symptoms both of convulsions and hemiplegia, and that the deviation in the former is directed away from and in the latter towards the hemisphere in which the lesion is situated. In the brain of the monkey Ferrier localizes a centre in the posterior extremity of the second frontal convolution, irritation of which causes elevation of the eyelids, dilatation of the pupils, conjugate deviation of the eyes and turning of the head to the opposite side; and he also found that extensive movements of the eyeballs, along with associated movements of the head and neck result from irritation of the supramarginal and angular gyri. One or two cases are recorded which render it probable that lesion of the posterior extremity of the second frontal convolution will cause conjugate deviation of the eyes and rotation of the head and neck in man, but the recorded cases are not conclusive upon this point. Numerous cases, however, have been recorded in which this symptom was caused by a lesion in or near the angular gyrus. Grasset has collected much evidence upon this point and he has recorded a case of his own in which the patient suffered from left-sided hemiplegia and a conjugate deviation of the eyeballs with rotation of the head directed to the right, and in which the disease

consisted of lesion of the *pli courb* of the right hemisphere (Fig. 166). Many cases are also reported in which conjugate deviation of the eyes was caused by disease of the centrum ovale and in these cases the lesion was, as a rule, situated between the internal capsule and the supra-

Fig. 166.

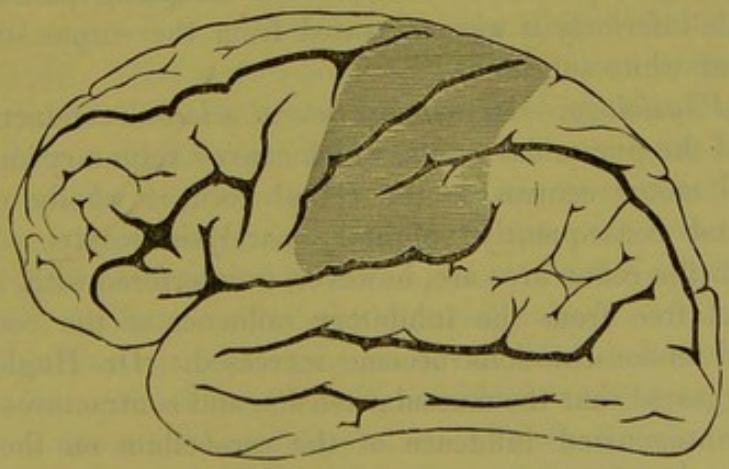


marginal and angular gyri. It would also appear that disease in the neighborhood of the angular gyrus and supramarginal lobule produces at times paralysis of the levator palpebræ superioris of the opposite side, without the other muscles supplied by the third nerve being implicated.

8. *Hemiplegia from Cortical Disease.*

A case of complete hemiplegia of the right side, of six years' duration is described by Lépine in which there was total destruction, caused

Fig. 167.



by yellow softening, of the ascending parietal convolution, and partial destruction of the Island of Reil, ascending frontal convolution, and the

anterior part of the superior and inferior parietal lobules of the left hemisphere (Fig. 167). Secondary degeneration was traced in the left half of the pons and in the pyramid of the medulla. Several similar cases are reported by others. Dussaussay describes a case, quoted by Pitres, of right hemiplegia with conjugate deviation of the eyes to the left, and in which a cavity was found in the centrum ovale of the left hemisphere containing dark coagulated blood (Fig. 168). This cavity

Fig. 168.

was limited internally by the gray substance of the paracentral lobule; superiorly and externally by the gray substance of the ascending frontal and parietal convolutions; in front it extended to the præcentral fissure; and behind to the posterior border of the ascending parietal convolutions; while inferiorly it was separated from the corpus striatum by a thin layer of white substance.

Morbid Physiology.—In *primary lateral sclerosis* conduction through the fibres of the pyramidal tracts, which convey voluntary impulses from the cortical motor centres to the spinal cord, is wholly or partially arrested, and consequently voluntary paralysis results. The spinal centres with the reflex arcs are, however, uninterfered with, and as they are now set free from the inhibitory influence of the cerebrum the reflexes and tendon-reactions become increased. Dr. Hughlings-Jackson has suggested that the muscular tension and contractures are caused by the unantagonized influence of the cerebellum on the paralyzed muscles, but this theory is not free from difficulty.

Amyotrophic lateral sclerosis is caused, according to Charcot, by a primary lateral sclerosis to which disease of the anterior gray horns

becomes superadded, the lesion beginning in the cervical region and subsequently extending upwards to the medulla oblongata and downwards to the lumbar region. The presence of lateral sclerosis explains the facts that the paralysis often precedes the atrophy, and is associated with muscular tension, contractures, and exaggerated tendon-reactions; while the presence of disease of the ganglion cells explains the muscular atrophy which becomes associated with the paralysis. But the paralysis of the upper extremities is sometimes of the pure atrophic variety, and I cannot help thinking that in some cases at least the lesion begins in the gray matter and extends outwards to the lateral columns.

In disease of the substance of the cerebral hemispheres and of the basal ganglia, hemiplegia is caused by arrest of conduction through the fibres of the pyramidal tract. If the clot simply compresses these fibres without rupturing them, recovery takes place when the effused blood is absorbed, but if these fibres are ruptured the hemiplegia becomes permanent. It is very probable that early rigidity occurring at the time of the hemorrhage is caused by rupture of these fibres, while the rigidity which begins a few days after the attack is caused by an inflammation of the fibres excited by the presence of the clot, which acts as a foreign body. Late rigidity comes on about the same time as the descending changes in the pyramidal tract, and is produced by the same mechanism as the spastic rigidity of the limbs in primary spinal lateral sclerosis. It is not easy to give reasons why choreiform movements are so liable to occur when the lesion is situated in the area of the posterior external optic artery. Two probable explanations of these clonic spasms suggest themselves to my mind. The first is that fibres connecting the cerebrum with the cerebellum are injured by these lesions, so that the normal proportion between the outgoing discharges which regulate the tonic (cerebellar) and the clonic (cerebral) actions of the body is lost. The second is that the spasms are caused by partial injury of the fibres of the pyramidal tract which regulate the fundamental actions. The fundamental actions are regulated from the convolutions near the longitudinal fissure, and the special actions from the convolutions which border the Sylvian fissure, and it is manifest that the fibres which descend in the corona radiata from the former will pass on the optic thalamus side of the internal capsule, while those which descend from the latter will pass on the lenticular nucleus side of it. Injury of the optic thalamus will, therefore, be likely to damage the fundamental fibres of the pyramidal tract, while leaving the accessory fibres unaffected. The effect of impairment of the fundamental actions might well be to cause the voluntary and special actions to be effected with irregularity and want of harmony. The only interpretation which

the spastic paralyses of infancy admit of, is that the cerebral motor mechanism in one or both hemispheres is either destroyed by a lesion after its development, or that the development of the motor mechanism is defective from the first or completely arrested.

The morbid physiology of the functional paralyses is somewhat obscure. Post-epileptic paralysis is most probably induced by the exhaustion of the motor cortical centres caused by a previous excessive discharge, just as the sciatic nerve of a frog is paralyzed for a time by passing a strong faradic current through it. Hysterical paralysis is also best explained by supposing that the molecules of the cells of the motor centres of the cortex are in a state of stable equilibrium, and consequently impulses from the cortex fail to be conducted outwards to the muscles through the fibres of the pyramidal tract. It is still more difficult to give an explanation of the toxic paralyses. It is impossible for us to tell why curara acts on the intramuscular nerve endings, atropine on the vagi, and lead on certain peripheral nerves; we must accept the facts as they are disclosed by experiment and observation and generalize them as best we can.

Treatment.—The treatment of the spasmodic paralyses will depend upon the primary lesion which causes the sclerosis of the lateral columns which underlies the paralysis. Primary sclerosis of the lateral columns of the cord must be treated internally like other cases of chronic myelitis by iodide of potassium, while the galvanic current is the most trustworthy local remedy. A carefully conducted hydropathic treatment may sometimes be beneficial, and gaseous thermal springs have been employed with advantage. Local stimulating treatment by means of the faradic current and massage must be avoided in all forms of spasmodic paralysis. Amyotrophic lateral sclerosis must be treated on the same general principles as other forms of chronic myelitis. In secondary sclerosis of the lateral columns of the cord the case must be carefully investigated with a view to discover whether the primary lesion is situated in the vertebræ, membranes, or cord itself, and then the nature of the lesion, whether inflammatory, scrofulous, or syphilitic, should be determined, and the treatment must be conducted accordingly. In hemiplegia and monoplegia the locality of the lesion must first be investigated and then an endeavor must be made to determine whether the lesion is likely to be a hemorrhage, embolism, thrombosis, or a tumor. The treatment of these morbid conditions will be subsequently described. The local treatment of the paralyzed limbs consists of the application of the galvanic current, and a moderate degree of rubbing of the surface, but deep kneading of the muscles, and the application of the faradic current or any other stimulating measures, must be avoided.

CHAPTER IX.

DISORDERS OF MOTOR COÖRDINATION.

1. MÉNIERE'S DISEASE (AURAL OR LABYRINTHINE VERTIGO).

Etiology.—Aural vertigo, as described by Ménière, comes on suddenly and without apparent cause. Acute and chronic catarrh of the middle ear and inflammation of the labyrinth are often accompanied by deafness and severe vertigo, and transient vertigo may be caused by the impulsion of air or fluids through the Eustachian tube into the tympanic cavity, by external pressure on the drum-head, by wax or other accumulation in the meatus, or by the passage of a galvanic current through the ears. Aural vertigo with deafness is sometimes one of the earliest symptoms of locomotor ataxia.

Symptoms.—The characteristic symptoms of Ménière's disease are sometimes preceded by partial deafness, earache, and other indications of a local disease, but in other cases the patient is suddenly attacked with deafness, noises in the ears, and a feeling of giddiness, which is attended by fainting, nausea and vomiting. The attack passes off in a few seconds or minutes, but recurs after a variable period, the paroxysms becoming more aggravated, and more frequently repeated as the disease advances. The noise is sometimes heard in both ears, but is probably always more pronounced on one side than the other; it is compared to the loud whistling of a steam engine or to a succession of explosions, or is described as a continuous humming or buzzing. The noise ceases with the attack in recent or slight cases, but continues to distress the patient during the interval in long-standing and severe cases.

The attack of vertigo varies in duration and intensity. In slight cases it consists of a momentary feeling of swimming in the head, but in severe cases each paroxysm may extend over a period of ten or more minutes, while in still more aggravated cases the feeling of uncertainty and giddiness is never absent during waking hours, and every effort on the part of the patient to assume the erect posture determines a paroxysm of vertigo, nausea, and vomiting. During the paroxysm the patient feels as if he were rotating round a vertical or horizontal axis, or as if he were falling forwards, backwards, or laterally; he staggers

and clutches at surrounding objects, or actually falls in the direction in which his abnormal sensations would lead him to expect. The patient also feels faint, and occasionally actual syncope may occur; the skin then becomes pallid, cold, and covered with sweat, pulse is feeble and flickering, the patient suffers from confusion of ideas, and in aggravated cases there may be a transitory loss of consciousness, and there is an intense feeling of nausea which often terminates in vomiting, when the attack usually comes to an end. Oscillatory movements of the eyeballs have in some cases been observed during the attack, and in these cases there was an apparent displacement of objects with reference to the patient. When one ear is exclusively affected with noises and deafness the patient may experience a constant tendency to walk to the opposite side. It was shown by Hinton that perception of musical notes is often faulty in this disease; in one case observed by him *G* of the third octave was heard as *C* of the octave below, or twelve notes below the normal pitch, and more or less similar results were obtained with other notes.

The course of the disease is a steadily progressive one until complete deafness is established, when, fortunately, all the distressing symptoms of the disease cease. The paroxysms of vertigo come on at first at irregular intervals; they increase gradually in frequency and intensity, and in aggravated cases the patient suffers continuously from some degree of vertigo, while he is liable to paroxysmal exacerbations of great severity. The noises in the ears may cease at first during the intervals, but after a time they are constant during waking hours until the patient is completely deaf.

2. PRIMARY SCLEROSIS OF THE COLUMNS OF GOLL.

Only a few cases which can be regarded as a primary affection of the columns of Goll are recorded. In a case recorded by Pierret the symptoms consisted of a girdle sensation passing round the body, lightning pains and various other sensory disorders in the lower extremities, and inability to maintain the erect posture with closed eyes, while on wishing to advance the patient felt as if she were drawn backwards, but once started she was impelled forwards by an uncontrollable force. The patient died at the age of thirty years from an attack of acute pneumonia, and at the autopsy Pierret discovered the presence of sclerosis of the columns of Goll. The posterior root-zones were, however, implicated in the lesion to some slight extent in the dorsal region, and, judging from the drawings which accompany the report of the case, in

the lumbar region also. The case cannot, therefore, be regarded as a typical example of sclerosis of the columns of Goll. A case of primary sclerosis of the columns of Goll has been reported by DuCastel and another by Gowers, but in neither were there symptoms during life which could with probability be attributed to disease of these columns.

3. SECONDARY SCLEROSIS OF THE COLUMNS OF GOLL AND OF THE DIRECT CEREBELLAR TRACTS.

These affections are only of anatomical interest inasmuch as they do not give rise to any recognizable symtoms.

4. PROGRESSIVE LOCOMOTOR ATAXIA (TABES DORSALIS, GRAY DEGENERATION OR SCLEROSIS OF THE POSTERIOR COLUMNS OF THE SPINAL CORD).

Etiology.—Locomotor ataxia often occurs in individuals whose nearest relatives have suffered from other nervous diseases, such as monomania, hypochondriasis, epilepsy, migraine, mental disease, or violent fits of anger and drunkenness. In other cases the heredity is direct from parent to offspring. Friedreich met with three different families in which several brothers and sisters were attacked with the disease about the same age, while the parents themselves were healthy, and Carré was informed by an ataxic patient that eighteen other members of her family were affected by the same disease. Locomotor ataxia, according to the statistics of Eulenburg, attacks males in the proportion of one hundred and twenty-eight to twenty-one females. It is essentially a disease of youth and middle age, by far the larger number of cases occurring between the ages of twenty and fifty years, although it has been observed occasionally in children.

The exciting causes of locomotor ataxia are exposure to cold and damp, severe bodily and mental exertion, emotional disturbances, sexual excesses, and injuries of various kinds, such as a fracture of the thigh or concussion of the spinal cord. It is sometimes the sequel of acute disease, such as typhus, articular rheumatism, acute pneumonia, and diphtheria, and of repeated abortions and the puerperal state.

But of all the antecedents of locomotor ataxia, syphilis is the most frequent and most important factor in its genesis. Fournier stated, in 1876, that syphilis had preceded locomotor ataxia in twenty-four out of thirty cases observed by him, and soon afterwards Vulpian asserted

that about seventy-five per cent. of patients suffering from locomotor ataxia are old syphilitic subjects. In the more recent statistics of Erb, Fournier, and others, the frequency with which ataxia attacks syphilitic subjects is placed as high as from eighty-nine to ninety-one per cent. of all cases, while Fischer, Westphal, and others do not find the proportion higher than from fourteen to twenty per cent. My own experience agrees with the higher rather than the lower percentages. Locomotor ataxia is one of the tertiary manifestations of syphilis, but Fournier met with a case in which the symptoms of tabes began twenty-two months after the primary infection, and during the secondary symptoms; in other cases an interval of upwards of twenty years elapsed between the infection and the first appearance of the tabetic symptoms, but the majority of cases of syphilitic ataxia occur between the sixth and twelfth years after infection. It is important to notice that locomotor ataxia succeeds with much greater frequency to the milder than to the severer forms of syphilitic infection, and Fournier has shown that an efficient mercurial treatment during the early period of syphilis greatly diminishes the liability to ataxia.

Symptoms.—The course of locomotor ataxia may, for the purposes of description, be divided into three stages: (1) The premonitory or preataxic stage; (2) the ataxic stage; and (3) the paralytic stage, or the period of the generalization of the ataxia.

(1) The Preataxic Stage.

The symptoms of the preataxic stage may be subdivided into disorders of (*a*) general sensibility; (*b*) the general motor functions apart from ataxia; (*c*) the special senses; (*d*) the cranial motor and mixed nerves with the cilio-spinal region of the cord; (*f*) the nutritive functions; and (*g*) the cerebral and psychical functions.

(*a*) *Disorders of General Sensibility.*—The most constant of the premonitory symptoms of locomotor ataxia are *pains* of a peculiar and often distressing kind, which may precede by many years all other symptoms, and generally last throughout the whole course of the disease. These pains are compared by the patient to forked lightning darting through the body, and are consequently named lightning-like or lancinating pains. The lightning pains are, as a rule, not very urgent at first and are believed by the patient to be of rheumatic origin. They consist of a single dart which shoots suddenly into some part of the lower extremities, or occasionally of the back of the head or side of the face, and disappears as suddenly as it came, but only to return at no distant

date. As the disease advances the shooting pains recur at shorter intervals, and after a time a number of them follow each other so quickly as to constitute a paroxysm of almost continuous pain. The paroxysm may last only a few minutes at first, but by and bye its duration is prolonged to some hours or even days, during which the patient suffers the most excruciating agony. The duration of the interval between the paroxysms is also very variable; in some cases the paroxysm recurs every few weeks, but in the interval the patient is comparatively free from pain, while in other cases the patients never know what it is to have a perfectly free interval from pain, and they are liable to suffer every few weeks from exacerbations of great severity. The character of the pain also varies; it is sometimes described as burning, gnawing, or dragging, and during the paroxysm the pain is said to shoot like lightning through the limbs, or to be like that which would be caused by a sharp knife or red-hot iron passing quickly through the tissues. The lancinating pains are usually deep-seated in the soft tissues or in the bones, but in addition to these, true neuralgic pains, which are limited to the area of distribution of a particular nerve, may supervene in the course of the disease. These neuralgic pains sometimes shift from one nerve territory to another, and do not invade the same region during successive paroxysms of pain, but at other times the pain becomes localized for a long time in a particular nerve like the sciatic, and obstinately resists all treatment.

Pain along the vertebral column, which is generally situated in the loins or between the shoulders, is another symptom of tabes. This pain is dull, aching, and more or less continuous, but presents alternating periods of remission and exacerbation, and it is only on rare occasions that it rises to any great intensity, and then it may be suspected that the disease is complicated by a spinal meningitis.

The girdle pain which is so frequently met with in many spinal affections is an almost constant symptom of locomotor ataxia, and the patient feels as if a rope were drawn tight around his body, or as if it were encircled by an unyielding iron ring. This sensation is usually situated on a level with the lower thoracic or upper abdominal region, although it may occasionally be felt at the lower part of the body and it may then reach to the neck of the bladder. A pain of similar character may likewise be felt around the joints of the lower extremities, and patients then describe it as a feeling like that caused by a garter tied tightly below the knee.

Cutaneous hyperæsthesia is not an uncommon symptom of the early stage of tabes, and it may be present in several forms. At times there may be hyperæsthesia to impressions of temperature and anæsthesia to

touch, while at other times anæsthesia to touch may be accompanied by a high degree of hyperæsthesia to impressions of pain. The hyperæsthetic condition is distributed in patches, which are often found on the lower extremities of patients suffering from severe paroxysms of lancinating pains. Circumscribed areas of skin then feel exquisitively sensitive to the slightest contact, such as a light touch or the rubbing of the clothes, and a slight touch on one of these spots appears to be capable of determining an attack of the lancinating pains. These hyperæsthetic areas are also subject to attacks of spontaneous pain, which is generally of a scalding or burning character; a patient once compared this pain to that which might be caused by rubbing into the skin a burning vesuvian match.

Various paræsthesiæ are also experienced in the preataxic stage of the disease. The patient suffers from the sensation popularly known as "the fidgets"; in other cases, from a feeling of heat which may be so intense as to compel him to uncover his feet at night; and in still other cases he is troubled with cold feet from vaso-motor spasm. The patient often complains of numbness and tingling of the extremities, the soles of the feet being, as a rule, the parts first attacked. Patients feel as if they were walking on wool, cork, or felt soles, or upon bladders of water, and in aggravated cases they have a sensation as if the ground were passing from under their feet, or as if they were walking on air. When their hands are affected they feel as if their fingers were covered with gloves, and are then unable to discriminate the primary qualities of objects which they grasp.

(*b*) *Disorders of the General Motor Functions apart from Ataxia.*—The patellar tendon-reactions are usually absent at a very early period of the premonitory stage of locomotor ataxia, and whenever these reactions are wanting in a person who has for some time been suffering from recurring lancinating pains, and who is not the subject of an atrophic paralysis of the quadriceps femoris muscle or of diabetes, there is a very strong presumption that he has already entered upon the first stage of this progressive disease. It is possible that these reactions may be absent in a few persons who are otherwise healthy, while, on the other hand, they may be present or exaggerated in a very few cases, in which other symptoms strongly point to locomotor ataxia; but with these slight reservations absence of the patellar tendon-reaction is one of the most certain and valuable signs of the early stage of locomotor ataxia. The cutaneous reflexes are usually unaffected in the early stage of locomotor ataxia, but as the disease advances the normal interval between the cutaneous excitation and the resulting contraction may become greatly prolonged. In a few cases the cutaneous reflexes are greatly

exaggerated even at an advanced period of the disease. Paradoxical contraction has been observed by Westphal as a symptom of tabes, and its presence may possibly be taken as a sign that the lesion of the posterior columns is extending to the lateral columns, and that the paralytic stage of the disease is approaching.

Transitory paresis of some muscular groups is an occasional symptom of the early stage of tabes; whilst walking, the legs may suddenly give way at the knees so that the patient falls to the ground. This sudden failure of motor power appears to be comparable to the transitory diplopia which is so common a symptom of the preataxic stage. In other cases the disease is ushered in by the occurrence of a sudden paraplegia, and it is only after the patient has recovered from the paralytic symptoms that the motor incoördination becomes apparent. It is probable that in these cases the primary lesion had extended transversely so as to have affected the lateral columns, and that under treatment it has receded so as to become limited again to the posterior columns. Uncertainty of gait and station when the eyes are closed, or when in the dark, is a motor symptom frequently observed before true ataxia is established. This is, indeed, frequently the first symptom to attract the patient's attention. When he puts his hands over his face whilst washing, in the morning, he may feel as though he were swaying from side to side, or he may totter, and he may be compelled to steady himself by propping his knees against the washstand, while his uncertainty of gait becomes very troublesome when he ascends a flight of stairs in the dark, or enters an unlighted chamber. On careful observation of the patient it may be noticed that he has a slight tendency to stagger when he rises suddenly after sitting for some time, or when he is asked to turn suddenly round or to walk backwards, but so long as he walks straight forwards with open eyes and in daylight there is no staggering nor uncertainty of gait. The patient is also troubled at this period with attacks of vertigo, which is the subjective correlative either of diplopia or of the uncertainty of gait. Vertigo is a prominent symptom of the cases of congenital ataxia described by Friedreich.

(*c*) *Affections of the Special Senses.*—Amaurosis is a frequent and distressing symptom of locomotor ataxia. Interference with sight is indicated by a slowly or rapidly advancing diminution in the acuteness of vision, and by a restriction of the field of vision, first, for the perception of colors, and finally for the recognition of objects; the patient may become blind at the end of a few weeks, or years may elapse before the loss of sight is complete, and occasionally the affection ceases to progress after it has lasted for a comparatively long time. The blindness

is caused by white atrophy of the optic disks, which is said to appear in about thirty per cent. of all cases of tabes.

Hemiopia has been met as a transitory symptom of the preataxic stage, but its occurrence is rare. Deafness is not an infrequent accompaniment of locomotor ataxia, but it is only in a certain number of these cases that the loss of hearing can be regarded as forming a part of the disease, and in such cases the deafness is probably caused by a parenchymatous atrophy of the auditory nerve analogous to that of the optic nerve. Deafness, with vertigo and the other symptoms of Ménière's disease, has occasionally been observed in the early stage of tabes.

The *olfactory* sense is also occasionally lost, a case being described by Althaus in which the patient complained at first of a subjective smell of phosphorus, and ultimately lost all sense of smell.

The *gustatory* sense has not, so far as I know, been found affected, although slight disturbances of the sense of taste may be present when the trigeminus is implicated.

(*d*) *Disorders of the Cranial Motor and Mixed Nerves, and of the Cilio-spinal Region of the Cord. Paralysis of the Ocular Muscles.*— The symptoms of locomotor ataxia are frequently ushered in by paralysis of one or more of the ocular muscles, the paralysis being at first generally transient. After a longer or shorter time, however, the paralysis recurs and becomes permanent towards the later stages of the disease. The transient paralyses of the first stage are generally so slight that they declare themselves not by the presence of a perceptible squint, but by the sudden appearance of diplopia, which is either constantly present or only when the eyes are turned in particular directions. Distinct squint and ptosis are, however, occasionally present in the early stage, and very frequently in the later stages of the disease. Out of sixty-four cases of tabes collected by Eulenburg twenty-five had strabismus; of these twenty-five cases nineteen had divergent strabismus, and four had in addition paralytic ptosis, while six had convergent strabismus.

Disorders of the pupils are frequently present in a very early stage of locomotor ataxia. Of the sixty-four cases collected by Eulenburg nine had mydriasis, the dilatation being double in three, single in four, and accompanied by myosis of the other in two cases, while twenty-eight had myosis, the contraction being double in twenty-one and single in seven cases. Inequality of the pupils is common in the early stage of the disease, and on the side on which the contraction of the pupil is the more marked there may be redness of the cheek, congestion of the conjunctiva, local elevation of temperature, and relative diminution of

the palpebral aperture, while in a few cases unilateral hyperidrosis of the side of the face has been observed.

The *Argyll-Robertson pupil* is frequently present in the early stage of locomotor ataxia, and it is generally, although not invariably associated with myosis.

Nystagmus is a very rare symptom of locomotor ataxia, and is probably never present except in the hereditary form of Friedreich.

The *trigeminus* is not unfrequently implicated in tabes. The most prominent symptoms in the area of distribution of this nerve are pains, which may either be neuralgiform in character or of the same kind as the lancinating pains of the lower extremities. Both kinds of pain may be felt in the forehead, face, parietal region, external auditory meatus, and the lancinating pains are particularly liable to be felt in and around the orbit. Paroxysms of these pains may sometimes be mistaken for hemicrania, more especially as the attack is often accompanied by redness of the conjunctiva, photophobia, and an increased flow of tears. The attacks are usually followed by cutaneous hyperæsthesia, but as the disease advances the increased sensibility gives place to anæsthesia which is said to occupy most frequently the conjunctivæ and the nasal fossæ, but may implicate the mucous membrane of the mouth, tongue, and pharynx on one or both sides.

The motor branch of the trigeminus is not so frequently affected as the sensory branches, but occasionally cases have been observed in which the masticatory muscles were the subjects of ataxia, or were so feeble that the patient was obliged to live upon pulpy food.

The *facial nerve* is also occasionally implicated in the disease, and then the face may be contorted by grimaces when the patient speaks, from ataxia of the facial muscles. The muscles of the face, tongue, and larynx may be simultaneously affected with motor incoördination, and then the speech becomes ataxic; the articulation is hurried, stuttering, and becomes so indistinct in advanced cases that the patient's speech is almost incomprehensible. One side of the face may become suddenly more or less paralyzed, and paralysis of the veil of the palate, implicating the muscles supplied by either the facial or glosso-pharyngeal nerves, has occasionally been observed, while the symptoms of bulbar paralysis have been known to occur suddenly in the early stage of ataxia. These paralyses are, like the corresponding affections of the ocular muscles, generally of a transitory character.

The disorders in the area of distribution of the *tenth* and *eleventh* nerves are very numerous, and give to the disease some of its characteristic features.

Laryngeal crises, consisting of paroxysms of spasmodic cough like

those of whooping cough, are liable to complicate the early stage of tabes, while paralysis of the adductors, or more rarely of the abductors, of the vocal cord is occasionally present.

Cardiac crises, consisting of paroxysms of angina with irregular and frequent pulse, are occasionally present, and Charcot believes that in ataxia the pulse is permanently accelerated, often beating from 100 to 130 in a minute. Aortic regurgitation has so frequently been found associated with locomotor ataxia that some authors have been led to believe that the two diseases are related by some causal connection.

The *twelfth nerve* is only rarely affected in locomotor ataxia, but it is implicated with the facial and laryngeal nerves when there is ataxic speech. Several cases of tabes are recorded in which the lateral half of the tongue was paralyzed and atrophied, this symptom being generally associated with some degree of atrophy of the muscles of the hand.

(*e*) *Disorders of the Visceral Functions.—Gastralgic attacks* or *gastric crises* are frequently observed in the premonitory stage of tabes. The attack begins suddenly during a paroxysm of the lancinating pains. It consists of severe epigastric pain, which radiates in different directions, and is accompanied by distressing vomiting of a large quantity of watery mucus, which ultimately becomes mixed with bile and blood. The patient suffers during the attack from a profound malaise and vertigo, while in addition to the gastric pains the lancinating pains in the extremities are unusually severe and agonizing. These attacks may last for two or three days, and may recur every two weeks, but usually the interval between them is not less than a month.

Enteralgic attacks are occasionally present in tabes, but much less frequently than the gastric crises. These attacks consist of paroxysms of unusually severe colic, which are often accompanied by diarrhœic crises, or painful discharges from the bowels of large quantities of watery mucus often mixed with blood.

Nephralgic attacks, the symptoms of which are almost in every respect similar to those of renal colic have been observed in tabes in the absence of calculi, gravel, or blood in the urine.

Affections of the Bladder and Rectum.—During the early stage of the disease the patient often suffers from frequent and painful micturition along with neuralgic pains in the depth of the pelvis, the perineum, the neck of the bladder, or along the urethra. At other times the bladder manifests great tolerance of its contents, most probably from some degree of anæsthesia of the mucous membrane, and the patient may then go a whole day without feeling any desire to evacuate his bladder, and when he empties it at night as a matter of routine he is surprised at the large quantity it contains. At a later stage of the

disease paresis of the bladder makes its appearance, so that emptying the bladder takes an unusually long time, and there is some dribbling afterwards, or there may be a moderate degree of incontinence, but complete paralysis of the bladder only occurs in the last stage of the disease. Dr. Buzzard has suggested that a phosphatic calculus may readily form in tabes with bladder troubles, and he believes that many cases of atony of the bladder, for which surgeons are consulted, are examples of tabes.

Patients complain in the early stages of tabes of peculiar sensations in the rectum; they sometimes feel as if a large foreign body were being forcibly introduced in the anus, and this sensation is accompanied by a strong desire to evacuate the contents of the bowel, while occasionally there may be an involuntary discharge of fecal matters. Anæsthesia of the anus and paralysis of the sphincter are, however, only present in the later stages of the disease.

The *disorders of the sexual functions* consist at first of the phenomena of irritation, but even from the beginning the voluptuous sensations are often accompanied by slight dribbling of urine, spermatorrhœa, and involuntary seminal emissions, while the erections are often imperfect and accompanied by premature ejaculation. Locomotor ataxia in women is sometimes ushered in by symptoms of genital irritation arising in the absence of the usual sexual excitants, and consisting of erections of the clitoris followed by a discharge of mucus.

(*f*) *Vaso-motor and Trophic Disturbances.*—Vaso-motor disorders are not prominent features of locomotor ataxia. They are manifested by the presence of hot or more frequently cold feet, a tendency to the formation of *cutis anserina*, mottling of the skin, increased or diminished cutaneous secretion, and the occurrence of patches of ecchymoses on the lower extremities during paroxysms of severe lancinating pains.

The most common trophic affections are cutaneous eruptions, such as herpes, lichen, or bullæ like those of pemphigus, these eruptions sometimes appearing during or after every aggravated attack of lancinating pains. The nails of the great toes are liable to fall off spontaneously during the course of tabes, and are replaced in a few weeks by new and perfectly normal nails, which may in their turn be shed a few months later, and this process may be repeated several times in succession. All the teeth have been known to fall out within a period of a few weeks in the course of tabes. Perforating ulcer of the foot is most commonly met with in subjects who are suffering from the earlier symptoms of tabes. The nutrition of the muscles is usually preserved until a late period of the disease, but occasionally some muscular groups may undergo active atrophy in the preataxic stage. The intrinsic muscles of the

hand are particularly liable to be attacked, and then the hand presents the well-known claw-form, but the muscular atrophy which complicates tabes does not pursue the progressively invading course of that observed in progressive muscular atrophy. Atrophy of the muscles of the hand in tabes has several times been found associated with hemiatrophy of the tongue.

Disorders in the Nutrition of Joints and Bones.—The *arthropathies des ataxiques* of Charcot is the most remarkable trophic affection which is met with in tabes. The joint affections always appear in the lower extremities during the early stage of tabes, and also in the upper extremities during the early invasion of the symptoms in them, although the symptoms may be long established in the lower extremities, and they also begin, almost always, during an unusually severe paroxysm of lancinating pains. The knee-joint is the one most frequently affected, and then in a descending order of frequency the shoulder, elbow, hip-joint, wrist, and the small joints of the feet and hands. Various luxations of the joints ensue, which produce notable deformities which need not be described here. The bones sometimes become abnormally friable, so that spontaneous fractures are liable to occur.

(*g*) *Cerebral and Psychical Disorders.*—*Vertigo* is a common symptom of the early stage of ataxia. This symptom is sometimes the subjective correlative of diplopia, or is associated with aural troubles, but occasionally it comes on in the absence of apparent cause. Essential vertigo is frequently accompanied by confusion of mind, an involuntary turning of the head and eyes, and an embarrassment of speech which sometimes consists of a difficulty of articulation and at other times of a decided aphasia. These attacks are doubtless of the nature of minor attacks of epilepsy, and we shall now see that locomotor ataxia is occasionally ushered in by more decided epileptiform attacks.

Epileptiform Attacks.—The symptoms of tabes may be ushered in by unilateral epileptiform attacks with subsequent transitory paralysis of the convulsed limbs, while at other times the seizures are like the *petit mal* or occasionally like the *haut mal* of idiopathic epilepsy.

Apoplectiform Attacks.—Locomotor ataxia may also be preceded or accompanied by apoplectiform attacks similar, in every respect, to those which are met with in sclerosis in patches and general paralysis of the insane. In some of these attacks there is only a transitory loss of consciousness from which the patient recovers without any other symptom; in other cases the seizure is accompanied by embarrassment of speech, facial paralysis, brachial monoplegia, or complete hemiplegia with or without hemianæsthesia. In these cases the recovery takes place in a few hours or at most in a few weeks, the transitory nature of the paral-

ysis showing that it must have resulted from a functional lesion and not from a hemorrhage or other gross lesion of the brain. Locomotor ataxia may also be complicated with a permanent hemiplegia, and in such cases care should be taken lest the ataxia may not be entirely overlooked.

Psychical disorders are occasionally observed in the early stage of locomotor ataxia; the patient becomes irritable and moody and towards the end of the disease he may suffer from hallucinations, illusions, and weakness of memory. At other times his moral character undergoes a great change; he is sometimes grossly indecent, and may be guilty of making lascivious gestures, or leaving his clothes unbuttoned in the presence of his family. The symptoms of progressive paralysis of the insane are frequently associated with those of tabes, and sometimes those of the former and sometimes those of the latter take the precedence in the order of development.

(2) The Ataxic Stage.

After one or more of the premonitory symptoms which have just been described have been in existence for some time, the phenomena which characterize the ataxic period of the disease make their appearance. The duration of the preataxic stage varies greatly in different cases; in exceptional cases the ataxic gait begins at once, while in other cases lightning pains, amaurosis, absence of patellar tendon-reactions, and other symptoms of the disease may be in existence for a period of from twenty to thirty years before the motor incoördination is fully established.

Static Ataxia.—The motor disorders of tabes almost always begin in the lower extremities, and are at first so slight that they can only be recognized by the application of special tests. If the patient be asked to stand up and keep his feet closely applied along their inner edges, he may manage to maintain the erect posture with moderate steadiness when his eyes are open, but when they are closed the patient immediately oscillates from side to side, and will fall unless he is supported or open his eyes. A still severer test is afforded by asking the patient to stand on one leg, first with eyes open and then closed.

As the muscular incoördination increases standing without support, even when the eyes are open, becomes increasingly difficult and finally impossible. When the patient now stands with the aid of two sticks it may be observed that all the extensor muscles of the body are in a state of powerful muscular contraction. The muscles of the calf are strongly contracted, and extend the leg upon the foot so that they form

an obtuse angle with one another, the extensors of the thigh are contracted and extend the thigh upon the leg, the flexors of the thigh are also contracted, and the foot being fixed they tend to extend the trunk on the thigh, this tendency being greatly increased by contraction of the gluteal muscles and of the erector spinæ. The tendency of the patient to fall backwards, caused by contraction of the extensor muscles, appears to be counteracted by a voluntary contraction of the muscles which flex the body upon the thighs. The body is thus bent forwards so that the line of gravity falls in front of the line joining the centres of the arches of the feet, and the tendency to fall forwards is counteracted by the support of the two sticks. The attitude assumed by the patient under these circumstances is characteristic. The feet are kept widely apart in order to widen the base, the legs are drawn backwards so as to form an obtuse angle with the feet, the thighs are extended on the legs, and a plumb-line let fall from each trochanter falls considerably behind the heel, the body is inclined well forwards in order to keep the line of gravity in front of the arches of the feet, and the buttocks project well backwards.

Ataxic Gait.—In the early stage of the disease the patient may continue to walk, when once he has started, without the slightest difficulty or sign of motor disorder, but he may be observed to stagger a little on getting up suddenly from a chair, especially after sitting for some time. A little uncertainty of gait may also be observed when he is asked to stop walking at a given signal, and he may manifest a decided stagger when in the course of a brisk walk he is asked to turn abruptly around. The uncertainty of gait is also increased when the patient walks in the dark or closes his eyes, when he is made to ascend or descend stairs, or when he walks backwards.

But when the ataxia becomes more pronounced the gait is so characteristic that it can be readily recognized without the application of any special tests. The patient has now to direct his eyes to the ground and to his feet while walking, and were he to close them the movements of the legs would become disorderly, and walking would be impossible. The attitude already described, with the tonic contractions of the extensors of the body, renders it impossible for the foot to advance with the usual pendulum movement of healthy locomotion; the passive foot is flung forwards and outwards in one piece with a rapid jerk by a simultaneous contraction of the flexors of the thigh on the body and the abductors of the thigh, and it is subsequently brought to the ground with a thump. During the advance of the foot the heel is said by some observers to be the last to leave the ground and the first to touch it as the forward movement is being completed, but this mode of pro-

gression must be rare if it ever occurs. When the foot is about to be advanced a certain degree of flexion occurs at the knee-joint and the foot is raised off the ground; the foot is then suddenly projected forwards and outwards, and the heel is afterwards brought down with a thump in the manner already described. In order to enable the passive leg to clear the ground during its forward movement, the abductor muscles of the thigh on the side of the active leg enter into strong contraction, and the pelvis on the side of the passive leg is thus well elevated. The elevation of the pelvis on the side of the passive leg is indeed so well marked that the patient is in some danger of carrying his centre of gravity too far to the side of the active leg. In order to counteract this tendency the upper part of the body is curved to the opposite side by contraction of the erector spinæ, and when the patient is able to walk without the aid of sticks the arm on the side of the passive leg is thrust out laterally, and, in order to maintain his equilibrium during the transference of the line of gravity from one foot to the other in walking, the trunk is strongly inclined from one side to the other, and the arms are flung about like those of a rope-dancer. When the patient walks by the aid of sticks, the tendency to too great a displacement of the line of gravity towards the side of the active leg is instinctively counteracted by an outward inclination being given to the sticks, so that they afford a lateral support.

Patients have been known to walk long distances after being affected with a high degree of ataxia, but after a time the incoördination becomes so great that the maintenance of the erect position and walking become impossible. If the patient be now supported by two persons under the arms whilst he tries to walk, the legs are thrust backwards and forwards, to the right and to the left in the utmost disorder, and are incapable of giving the least support to the body; they move, as Trousseau remarks, like those of a puppet or a marionette. The muscles of the trunk now become affected, and the body makes irregular, swaying movements when the patient sits in a chair, but even in these advanced cases the patient when laid down may be able to resist passive movements of the limbs, and to perform the simple movements of flexion and extension with scarcely diminished power. When the patient now attempts to touch an object with the tip of the foot, the line of motion is irregular and zigzag, and disturbed by lateral movements, while he is quite incapable of exerting more complicated movements, such, for instance, as are required in describing an imaginary circle with the tip of the great toe.

At a late period the ataxia appears in the upper extremities; in the cases of hereditary ataxia described by Friedreich, the motor inco-

ordination appears in the upper simultaneously with, or soon after its first manifestation in the lower extremities, but in the more usual form of the disease ataxia of the upper extremities is rare and belongs to the later manifestations of the disease. In ataxia of the upper extremities, complicated and special movements, such as those required for writing or playing the piano, are uncertain, and the irregularity becomes greater if an attempt is made to perform them with closed eyes. The slighter degrees of ataxia of the upper extremities may be tested by instructing the patient to touch with closed eyes some part of the surface of the body, such as the tip of the nose or lobule of one ear with the point of the forefinger of each hand alternately, when the ataxia declares itself by the inability of the patient to touch the intended spot until after repeated trials. A similar uncertainty is observed when the patient is asked to transfer a small object from one hand to the other. At a later period of the disease the simpler movements become irregular and ataxic, the patient fumbles in buttoning and unbuttoning his clothes, the act of grasping is performed in an uncertain and spasmodic manner, and the patient can only reach the object in a roundabout way and with jerky interruptions. Static ataxia may be revealed in the upper extremities by the inability of the patient to hold out his arms horizontally, especially when the eyes are closed. In a still more advanced stage of the disease patients can no longer dress or feed themselves, inasmuch as they cannot perform such simple movements as are requisite for carrying a spoon to the mouth, but even under these circumstances they may be able to exert great muscular power in resisting passive movements. The ataxy may invade, as we have seen, the muscles of the trunk, and ultimately the muscles of the neck may also be implicated, and the head is the subject of irregular and shaking movements.

The electrical reactions of the muscles are not of much value in the prognosis or diagnosis of locomotor ataxia. It is probable that in the earlier stages of the disease there is an increase and in the later stages a diminution of the electric contractility.

Sensory Disorders.—The girdle pains and the lightning pains of the premonitory period are continued, as a rule, through the ataxic stage, and often, indeed, become increased in severity as the disease advances. The hyperæsthetic spots, however, now give place to anæsthesia, and anæsthetic patches may be found on the soles of the feet, the toes, and the back of the feet, and they may become widely diffused over the thighs and trunk before the patient is aware of their presence. After a time, however, the patient finds that he no longer feels the floor distinctly, that all articles which he touches have a velvety feel, and that he cannot hold small objects in his fingers. Every variety of

paralysis of sensation and every combination of these are met with in the late stages of the affection, but probably analgesia is the most frequent. Occasionally, however, the sensibility to pain is retained, or even increased, while there is a diminution of sensibility to some or all varieties of touch, and again, partial paralysis of the sense of touch may be combined with analgesia and hyperalgesia, or with hyperæsthesia towards impressions of temperature. At a late period of the disease a distinct retardation of the conduction of sensations, especially of impressions of pain, is observed, and this may also be found in the earlier stages. The prick of a pin frequently gives rise to a double sensation, the first of touch and the second of pain, and the sense of temperature may also be retarded. The sensation of pain continues for a relatively long time, even when its cause has only been of momentary duration, and the highest degree is not reached until several seconds after the pain has begun. In circumscribed areas of the skin of the foot the patient may feel two points when only one is touching the skin, and when two points are in contact four or five may be felt. In some cases the patient is not sure which side of the body is touched when the eyes are closed, even although the cutaneous sensibility is more or less completely retained. In the early stage of the disease the disorders of the muscular sensibility and muscular sense declare themselves by a feeling of fatigue and the sensation termed the "fidgets," but as the disease advances the muscular sense becomes diminished, and consequently the power of recognizing what muscles are thrown into action is lessened in a corresponding degree. After a time the patient, when his eyes are closed, cannot tell the position of his lower extremities, and has lost the power of recognizing small weights, and of discriminating the differences of weights.

Visceral Disorders.—The gastralgiæ are seldom so distressing in the second as in the first stage of the disease, but the genito-urinary symptoms become much more urgent and troublesome. The sexual weakness of the early stage now advances to complete impotence, and the dribbling of urine, which was at first an occasional symptom, now becomes habitual, while in some cases there is so much anæsthesia of the urethra that the urine may pass in a full stream without the patient's knowledge. In other cases the patient is unable to evacuate the bladder, which becomes so distended as to require the use of the catheter. The tenesmus and neuralgiform pains about the rectum, which were present in the first stage of the disease, now give place to anæsthesia, and then there may be an unconscious passage of the stools, which, indeed, would be a much more frequent symptom if it was not for the high degree of constipation which is usually present.

Trophic disorders are not liable to occur in this stage of the disease, but the skin of the lower extremities may become dry and covered by furfuraceous scales, and patches of ichthyosis may form over the toes and metatarsal bones, and the muscles of the lower extremities may now undergo diffused wasting.

(3) The Paralytic Stage.

In the terminal period of locomotor ataxia the motor incoördination has increased to such an extent that the patient is quite unable to walk, and he is consequently obliged to sit propped up in an armchair, or to lie helplessly in bed; but even when he is quite unable to stand, the individual movements of the lower extremities may be performed with scarcely diminished power. But after a time even the power of performing separate movements is diminished, and the muscles undergo diffused atrophy, are soft and flaccid, and no tension is provoked on passive movements of the limbs. The anæsthesia to pain and touch is now complete, the patient is quite unable to tell the position of his limbs in bed without the aid of sight; but the sense of temperature may still remain nearly normal. The patient also suffers from incontinence of urine; the stools pass unconsciously; bedsores appear on the sacrum; chronic cystitis supervenes; and the patient dies in a state of great marasmus, as in other forms of myelitis. In other cases the disease may progress upwards and implicate the medulla oblongata, and the patient dies from respiratory troubles, or difficulties of deglutition. At other times cerebral symptoms may supervene, and the patient, after being delirious for a few days, dies comatose. But the disease runs its full course only on rare occasions, and in the majority of cases the patient dies from an intercurrent disease such as acute pneumonia, phthisis, or cardiac complication. The *course* of locomotor ataxia is usually slow and chronic; it often extends over a period of many years, and even the initial stage, with lancinating pains, may last more than twenty years. In the majority of typical cases the average duration of life appears to be from eight to twelve years, but a few cases run a rapid course, these having been described under the name of *acute ataxia*.

Varieties.—The following clinical varieties of locomotor ataxia may be distinguished:

(1) The *typical form of tabes*, which begins with lancinating and girdle pains, loss of patellar tendon-reactions, and disorders of the oculomotor nerves, and runs through the preataxic, ataxic, and paralytic stages which have been described.

(2) *Hereditary Tabes.*—This form of tabes was first described by Friedreich. The disease begins between the ages of twelve and eighteen years; it is usually remarkably protracted, extending sometimes over a period of thirty years; several members of the same family are liable to be affected, and the female members appear to be attacked by preference. Lancinating pains and other sensory disorders are never well marked and are often absent; the ataxia begins at an early period and attacks the upper extremities simultaneously with or soon after the lower extremities. Disorders of articulation, and ataxic nystagmus are generally present, and the tendon-reactions are lost; but static ataxia, bedsores, psychical disorders, tremor, and amaurosis are generally absent; while muscular atrophy, paresis, contractures, and weakness of the bladder appear only in the last stage. The symptoms of this group are very variable and the cases included in it are probably more allied to sclerosis in patches than to locomotor ataxia.

(3) *Anomalous Form.*—This variety comprises the form in which are present arthropathies, muscular atrophy, tremors of the upper extremities, psychical disorders, or any other prominent symptom which does not belong to the typical disease.

(4) *Paraplegic Form.*—In some cases the symptoms of tabes are obscured by the presence of paraplegia. The presence of paralysis with muscular tension may lead the observer to believe that the case is one of ordinary paraplegia, but in such cases the patellar tendon-reactions are usually absent, instead of being exaggerated, as in ordinary paraplegia. These cases will be subsequently described under the name of posterior and lateral sclerosis.

(5) *Neuralgic Form.*—The lancinating pains are very violent and distressing in this form, and these pains and the absence of the patellar tendon-reactions may be the only symptoms present for upwards of twenty years, and consequently this variety has been named "*tabes dolorosa*" or "abortive locomotor ataxia." *Amaurotic tabes* belongs to this variety, inasmuch as white atrophy with blindness may exist for very many years without any other symptom except absence of the patellar tendon-reactions.

(6) *Hemiataxia.*—The ataxic symptoms may in rare cases be much more pronounced on one side of the body than on the other.

(7) *Meningitic Form.*—When locomotor ataxia is complicated with spinal meningitis the typical symptoms of tabes are apt to be complicated by pain in the back, and along the vertebral column, spinal tenderness, and diffused or circumscribed cutaneous hyperæsthesia.

5. COMBINED SCLEROSIS OF THE POSTERIOR AND LATERAL COLUMNS.

Symptoms.—The symptoms of locomotor ataxia and of primary lateral sclerosis may be present in every possible combination, those of the former disease predominating at one time and those of the latter at another.

The symptoms which indicate that the lateral columns are being gradually invaded in cases of locomotor ataxia are spontaneous jerkings in the lower extremities, gradual loss of power to perform simple movements of extension and flexion, muscular tension, and contractures. When the symptoms of lateral sclerosis predominate, the signs by which a complication of locomotor ataxia may be suspected are the presence of lancinating pains, girdle pains, and other sensory disturbances, vesical weakness, slight swaying on closing the eyes, and, above all, the absence of the patellar tendon-reaction and ankle-clonus. Two cases came under my observation in which the symptoms of pure lateral sclerosis with exaggerated tendon-reactions present in the lower extremities were accompanied by extreme myosis of both pupils, and it is probable that there was a combination of sclerosis of the lateral columns, and of the posterior columns in the upper segment of the cord.

6. DISEASES OF THE CEREBELLUM.

The diseases of the cerebellum consist of hemorrhage, abscess, and tumors.

Symptoms.—The general symptoms of cerebellar disease consist of headache, which is more frequently situated in the occiput and only occasionally in the forehead, vomiting, vertigo, unsteadiness on standing which increases when the eyes are closed, and a reeling gait. In hemorrhage the symptoms come on suddenly, and when a large clot forms the patient suffers from apoplectic symptoms, and death may occur rapidly from pressure on the pons and medulla oblongata. Abscess of the cerebellum generally results from disease of the middle ear, while a tumor declares itself by the gradual onset of the symptoms or by the presence of double optic neuritis, which is frequently accompanied by amblyopia or even complete blindness.

Stationary lesions in the cerebellum may be of large size without giving rise to any recognizable symptoms. Progressive lesions cause different symptoms according to the situation of the lesion. When the lesion is situated in the upper part of the middle lobe the patient has a

staggering gait, and manifests a tendency to fall backwards, and when it is situated in the inferior part of the middle lobe it is probable that the tendency is to fall forwards, or to revolve round a horizontal axis. Tumors situated near the upper part of the middle lobe give rise to a tonic contraction of the muscles of the neck which causes retraction of the head. The muscles of the trunk and extremities may likewise be implicated in the spasm, and the patient then suffers from tetaniform seizures, during which the head is retracted, the body arched backwards, the lower extremities extended, and the upper strongly flexed as in tetanus. All lesions attended with an increase of size, like abscesses and tumors, must exert pressure forwards on the pons, inasmuch as the organ is limited above by the unyielding tentorium and behind and below by the occiptal bones. The consequence is that abscesses and tumors of the cerebellum are often attended by slight weakness of the limbs with increase of the tendon-reactions. The long course of the sixth nerves in front of the pons to reach the cavernous sinus renders one or other of them liable to be paralyzed in tumors of the middle lobe. Cases of tumor of the middle lobe are met with in which the patellar tendon-reactions are absent without any disease being found in the spinal cord. It is possible that in these cases there is an increase of the spinal fluid, owing to obliteration of the foramen of Magendie, which forms the communication between the spinal and cerebral subarachanoid spaces. Tumors of the cerebellum are also sometimes attended by numbness and tingling of the trunk and extremities, but only rarely by anæsthesia, and one or more of the cranial nerves may be more or less paralyzed. Movements of the eyeballs are frequently observed in cases of tumor of the middle lobe of the cerebellum. These movements may be vertical, horizontal, or oblique, and are generally parallel. At times they are only observed during a tetaniform seizure, but when there is permanent rigidity the eyes may be rotated in one direction and fixed, or slight parallel oscillatory movements may be present. When the lesion is situated in one of the lateral lobes or in one of the middle peduncles, there is a tendency to fall towards the side of the lesion. If a tumor is growing slowly, the tendency to fall to one side is counteracted by cerebral action to such an extent that this symptom is not readily elicited, but when the patient turns suddenly round, especially with closed eyes, a slight stagger may be observed, or the statement of the patient may indicate to which side he has a tendency to fall. A tumor of one lateral lobe may also encroach upon the middle lobe, and then the tendency to stagger to one side is accompanied by a tendency to fall backwards, so that the patient now manifests an inclination to fall backwards and to one side in a diagonal direction. If the tumor grows

forwards so as to encroach upon the lateral half of the pons, some of the cranial nerves become paralyzed, while the sensory and motor tracts may be more or less compressed. In such a case paralysis of the sensory and motor branches of the trigeminus may be present either separately or combined, along with more or less hemiplegia of the opposite side. Much more frequently the patient becomes completely deaf on the side of the lesion from compression of the auditory nerve, facial paralysis is then established, and the external rectus of the eyeball is the last to be affected. If the growth begins in the pons the sixth or seventh nerves are the first to be affected, and the auditory nerve last. If the growth encroaches upon the pyramidal tract hemiplegia of the opposite side, with or without some degree of anæsthesia, accompanies deafness and peripheral facial paralysis on the same side as the lesion. The paralytic phenomena may be preceded by spasmodic contractions of the masticatory, facial, or ocular muscles. Instead of paralysis of the external rectus of the eyeball on the side of the lesion, there may be a conjugate deviation of the eyes, which is directed towards the diseased side when the lesion is irritative, and away from it when it is destructive.

The symptoms caused by lesion of the inferior peduncle of the cerebellum are not well ascertained. In a case in which I suspected a hemorrhage of the restiform body where it joins the inferior peduncle, the symptoms were paralytic myosis and diminution of the palpebral fissure, and partial anæsthesia of the face on the side of the lesion, and partial hemiplegia with well-marked hemianæsthesia on the opposite side. Although the patient could move the partially paralyzed limbs pretty freely in bed, he was totally unable to stand without support; he had, indeed, to be supported on both sides in order to be kept erect, and it seemed as if he had an equal tendency to fall backwards, forwards, and to either side. On attempting to walk the head was drawn slightly backwards, and the eyes were directed upwards, the body was inclined to the paralyzed side until it was at an angle of about sixty degrees to the floor, and as each foot was moved forwards it was raised about eighteen inches from the ground, and the patient had the greatest difficulty in disentangling it from the leg which was planted on the ground. Sometimes he raised the feet alternately in the most ridiculous manner without moving forwards at all. When he attempted to lie down, after being placed sitting on the edge of the bed, his head became rotated towards the side of the lesion, and by the time his body reached the bed his face was directed towards the pillow. On some occasions the patient on making a semi-revolution round his longitudinal axis, worked his way back, but not without considerable difficulty, until he got on his back, while at other times he only got on his back after making a com-

plete revolution. He never manifested a tendency to revolve on his longitudinal axis when once laid on his back.

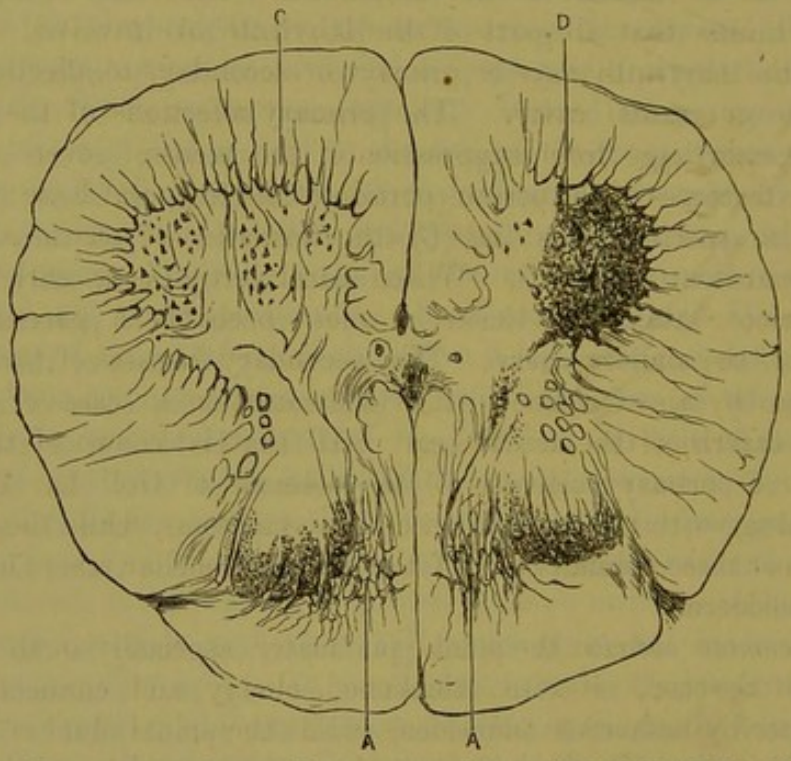
The symptoms caused by lesion of the superior peduncle of the cerebellum are also not well known. The proximity of these peduncles to the corpora quadrigemina renders it probable that they would give rise to unilateral or bilateral disorders of vision, and it is not unlikely that the eyeballs might be found on different levels, the one being directed a little upwards and outwards, and the other a little downwards and outwards. Lesions in this situation might also be expected to give rise to disorders of the third nerve.

Morbid Anatomy.—In aural vertigo the lesion was thought by Ménière to be limited to the semicircular canal and vestibule, but Knapp thinks that all parts of the labyrinth are involved. The disease of the labyrinth may be primary or secondary to affections of the tympanic or cranial cavity. The primary affections of the labyrinth are (1) hemorrhage from suppression of the menses, severe attacks of fever, or fractures of the petrous portion of the temporal bone; (2) serous effusion in specific forms; and (3) the formation of pus as a complication of purulent meningitis. When aural vertigo is an early symptom of locomotor ataxia the lesion is most probably a parenchymatous neuritis of the auditory nerve. The secondary diseases of the labyrinth are set up by an extension to it of inflammation in cases of acute and chronic catarrh of the middle ear. All that is known of the morbid anatomy of primary sclerosis of the columns of Goll has been mentioned along with the description of the symptoms, while the secondary sclerosis of these columns and of the direct cerebellar tracts has already been considered.

In *locomotor ataxia* the spinal pia mater, especially in the posterior aspect of the cord, is often thickened, cloudy, and connected to the dura mater by numerous adhesions, while the spinal fluid is increased. The cord is generally of an increased consistence, although it has occasionally been found softened, and it is often flattened from before backwards owing to the atrophy of the posterior columns, while a grayish-yellow discoloration may be observed extending the whole length of the cord along the posterior median fissure. On making transverse section the posterior columns are seen to be of a grayish color and diminished in size, and the posterior gray horns and posterior roots have also been found atrophied. The degeneration is not, as a rule, uniformly distributed over the whole transverse area of the posterior columns. The intensity and extent of the lesion, in long-standing cases, are greatest in the upper lumbar and dorsal regions, when it occupies the whole transverse section of the posterior columns. In the inferior

portion of the lumbar region there is frequently only a slight gray discoloration in the external half of the posterior columns, while in the upper dorsal and cervical regions the area of the lesion becomes again restricted, and in the upper cervical region the columns of Goll are alone affected. In long-standing cases the posterior gray horns may be found shrivelled and distorted, and the posterior roots wasted, while the direct cerebellar tracts may likewise be affected, and in a few cases the anterior gray horns have been implicated. In very protracted cases the whole transverse area of certain portions of the cord may be found, on section, transformed into a gray translucent mass. But when a

FIG. 169.



TRANSVERSE SECTION OF THE LUMBAR REGION, FROM A CASE OF LOCOMOTOR ATAXIA, COMPLICATED WITH MUSCULAR ATROPHY. (CHARCOT and PIERRET.)

A, A, Sclerosis of the posterior root-zone; C, Left anterior gray horn, healthy; D, Right anterior gray horn in a state of atrophy

patient, suffering from lancinating pains and other symptoms of abortive tabes, dies from some intercurrent disease, the sclerosis is limited to the root-zones of the posterior columns, and the columns of Goll are free from disease (Fig. 169).

On microscopical examination of the portions of the cord which have undergone sclerosis, the interstitial tissue is found increased in amount, the nuclei and Deiter's cells are increased in number, the walls of the vessels are thickened, and corpora amylacea and granule cells are scat-

tered throughout the tissue, the nerve fibres are thin and wasted, and many of them have disappeared, while in the later stages of the disease the tissue is converted into a firm fibrillary, often wavy connective tissue, in which only a few traces of nerve fibres are left.

The ganglion cells of the posterior gray horns are often found pigmented, but the ganglion cells in Clarke's columns are unaffected although a number of fine, apparently degenerated, nerve fibres may be seen to pass through and around them. The degenerative process sometimes extends to the anterior gray horns, and the motor ganglion cells and anterior roots are then seen to be atrophied. Degenerative changes have likewise been discovered in the peripheral nerves and the posterior roots, and cases have recently been recorded which render it probable that the morbid changes of tabes begin in the afferent fibres of the peripheral nerves. The optic nerves are sometimes found atrophied, and morbid changes have occasionally been found in the ocular motor and hypo-glossal nerves, while the roots of the pneumogastric nerves have been found atrophied in cases of tabes with laryngeal crises. The nuclei of origin of the cranial nerves on the floor of the fourth ventricle and the floor of the aqueduct of Sylvius are occasionally involved in the disease. When arthropathies form part of the disease the articular cartilages of the affected joints are destroyed, while the articular ends of some of the bones are eroded. In cases of combined lateral and posterior sclerosis the lateral as well as the posterior columns are in a state of sclerosis. In some cases the posterior sclerosis does not reach downwards as far as the lumbar region and in these the patellar tendon-reactions are exaggerated.

The diseases of *the cerebellum* consist of hemorrhage, softening, abscesses, and morbid growths; but inasmuch as their morbid anatomy is the same as that of other parts of the brain it is unnecessary to consider them further at present. The tumors most frequently met with, especially in young people, are tubercular growths and glioma.

Morbid Physiology.—Ménière's disease is caused by a primary or secondary disease of the semicircular canals, and the most consistent theory of their functions is that they are peripheral end-organs of afferent fibres, whose central end-organ is the cerebellum. As we have seen, the cerebellum is an organ for adjusting the body to space relations, and disease of the peripheral end-organs throws the whole of this mechanism into disorder, and consequently the patient is unable to effect those delicately coördinated muscular contractions which are required for maintaining various attitudes in space, and vertigo is the subjective correlative of this objective fact.

The case of primary sclerosis of the columns of Goll, recorded by

Pierret would seem to indicate that these columns take some part in the function of equilibration in space. The fact that these columns and the direct cerebellar tracts undergo an ascending sclerosis proves that they are centripetal conducting paths, and the fact that the former terminates indirectly and the latter directly in the cerebellum appears to show that both tracts consist of cerebello-afferent fibres. It is, therefore, likely that the columns of Goll and the direct cerebellar tracts conduct centripetal impulses to the cerebellum, but when sclerosis of these tracts is caused by a transverse myelitis, the symptoms which might be caused by disease of them are obscured by the paralysis which is caused by arrest of conduction through the pyramidal tract.

In *locomotor ataxia* the primary lesion is most probably situated in the posterior root-zones of the spinal cord, and in the homologues of these tracts in the medulla oblongata, pons, and crura cerebri; namely, the fasciculus rotundus, and the ascending and descending roots of the fifth nerves. It is, indeed, supposed by Dejerine that the primary changes begin in the peripheral nerves and ascend through the posterior roots to the spinal cord, and it is at least certain that the nerves of special sense are primarily affected in this peripheral manner. But even if the primary changes begin in the peripheral nerves, there can be little doubt that those portions of the posterior columns through which the afferent fibres pass to reach the posterior gray horns are involved in the disease at a very early period. As the disease of the posterior root-zones extends horizontally towards the posterior median fissure, the columns of Goll become implicated, and when once the fibres of these columns are interrupted in any part of their course the portions above the seat of the lesion undergo degeneration, and in advanced cases these columns are always found in a state of sclerosis throughout their entire length. The direct cerebellar tracts are also found in a state of degeneration in long-standing cases, and in some cases the morbid process is observed to have extended to the posterior gray horns, the anterior gray horns, and the lateral columns. The lancinating pains may be explained by the irritation of the fibres of the posterior roots in their passage through the posterior columns, while the subsequent anæsthesia may be supposed to be caused by destruction of these fibres. Irritation of the posterior gray horns causes the cutaneous trophic changes, while destructive lesions of these horns causes retardation of painful impressions and analgesia. Extension of the lesion to the lateral columns causes the paraplegia which sometimes complicates tabes, while implication of the anterior gray horns causes the muscular atrophy and probably also the arthropathies of locomotor ataxia. Disease of the automatic centres in the lumbar region causes the vesical and sexual

disorders. Parenchymatous atrophy of the optic nerves causes blindness and white atrophy of the optic disks, and it is probable that the disorders of the other special senses, occasionally observed, are caused by degeneration of the respective nerves. Absence of the patellar tendon-reaction is caused by disease of the afferent portion of the reflex arc in its passage through the posterior columns; while reflex pupillary immobility is caused by lesion of the fibres of the reflex loop which connect the afferent fibres in the optic tract with the efferent fibres in the third nerve. The fibres of this reflex loop proceed from the anterior tubercle of the corpora quadrigemina to the nucleus of the third nerve, and in their course they pass through or near the descending root of the fifth nerve, which is as we have seen the homologue of the posterior root-zone of the cord. The other symptoms which are observed in the region of the cranial motor nerves may be explained by sclerosis of the fasciculus rotundus and ascending root of the fifth nerve and by an extension of the morbid process from these bundles of nerve fibres to the nuclei themselves. The sympathetic oculo-pupillary symptoms are caused by irritation, or more frequently by paralysis, of the fibres which proceed from the dilator nucleus through the cervical portion of the cord to emerge with the eighth cervical and first dorsal anterior nerve-roots. These fibres descend most probably in the posterier root-zones of the cervical region of the cord, and are consequently liable to be implicated in the disease at an early period.

It now remains for us to explain the phenomena of static and dynamic ataxia. Leyden first suggested that the ataxia was the result of the disorders of sensibility which are present in this disease, but this theory fails to account for the well-ascertained clinical fact that, on the one hand, a slight degree of ataxia may be present in the almost entire absence of sensory disorders, while, on the other, there may be an absence of ataxia with a high degree of anæsthesia. It was suggested by Erb and others that the ataxia was caused by disease of motor or centrifugal paths, but this view is not supported by any well-ascertained physiological or pathological facts. It seems probable that the ataxia is caused by disease of afferent subconscious paths. These paths consist of afferent spinal, afferent cerebellar, and afferent subconscious cerebral tracts. The posterior root-zones themselves constitute, most probably, afferent spinal paths for coördinating various reflex movements, but it is probable that static ataxia is never present until the columns of Goll, or the direct cerebellar tracts are implicated in the disease. It has been suggested by Althaus that dynamic ataxia is caused by disease of the afferent conducting paths of the basal cerebral ganglia, but the tension of the extensors of the body which is present in tabes, when the patient

assumes the erect posture, would seem to take an active part in the production of the ataxic gait. This tension is caused most likely by irritation of cerebello-afferent paths and consequently irritation of these paths is likely to be one of the main causes of the ataxic gait. When the patient is unable to maintain the erect posture the extensors of the body have lost their tension, and it may then be presumed that these paths are more or less destroyed and that cerebellar paralysis is established for lack of communication with the external world. All that can be said with regard to the manner in which disease of the cerebellum causes the symptoms, is implied in what has already been said respecting the physiology of the organ.

Treatment.—When Ménière's disease depends upon morbid changes in the middle ear the patient should be placed under the care of the aural surgeon, and when the local disease is not accessible to treatment relief may be obtained by rest in the recumbent posture. The administration of quinine in doses of from ten to twelve grains three times a day for some days appears to have produced great amelioration of the symptoms in some cases. At first the subjective aural sounds caused by the quinine are superadded to those of the disease, and the symptoms are intensified, but if the quinine be omitted for eight or ten days the sounds and the vertigo undergo notable diminution. If the quinine be readministered for a period of eight or ten days the symptoms are again aggravated, to be followed by a second amelioration on its cessation, and after a time the attacks of vertigo may be completely arrested. Large doses of salicylate of sodium have also proven useful in the treatment of the disease.

Primary sclerosis of the columns of Goll requires to be treated by rest in the recumbent posture along with the remedies used for other forms of chronic myelitis, while secondary sclerosis of these columns and of the direct cerebellar tracts does not admit of treatment apart from the primary disease of the cord which has caused the sclerosis.

Locomotor ataxia is so frequently preceded by syphilitic infection that in all cases of the disease the most careful investigation should be instituted with the view of discovering any indication of the presence of this poison. In every case in which a history of syphilis can be detected, and in all doubtful cases, iodide of potassium in ten grain doses three times a day should be given, and if there are any active syphilitic symptoms present other than the ataxia, the patient should have a mercurial course. Cases of locomotor ataxia which are complicated by spinal meningitis are also benefited by the administration of iodide of potassium. But even when locomotor ataxia is of syphilitic origin the nervous disease pursues, after a time, an independent course, and consequently when the patient has been subjected to a thorough

antisyphilitic treatment, or when the absence of syphilis has been proved from the first, other methods of treatment must be adopted.

Of internal remedies nitrate of silver, given in doses of one-sixth to one-third of a grain three times a day is probably the most generally useful. It may be continued for three weeks at a time, and then after an interruption of two weeks its use may be resumed for another three weeks. The use of the remedy may be continued in this way with repeated interruptions for many months without producing deleterious effects. If the skin shows the slightest discoloration, the medicine must, of course, be interrupted for a longer period. Belladonna, arsenic, chloride of gold and sodium, and chloride of barium have also been employed in the treatment of tabes, but not apparently with much success. The administration of ergot is sometimes beneficial, but it ought to be given with caution, as diseased rye is known to cause sclerosis of the posterior columns of the cord. Phosphorus, either alone or with cod-liver oil, has sometimes been found useful. Strychnine is always useless and dangerous.

Thermal baths may be soothing to the patient during severe attacks of lancinating pains, but they do not appear to exercise a favorable influence over the course of the disease.

Saline thermal baths appear to act favorably, and those of Rheims and Nanheim have been favorably reported upon. *Sulphur*, *chalybeate*, and *mud* baths have been employed, but it is doubtful whether they do any good except by improving the general health of the patient.

Hydrotherapeutics, as carried out in well-conducted hydropathic establishments, is a very useful adjunct in the treatment of many cases of tabes. The institution should be situated in an elevated region, so that the patient may have the advantage of a pure mountain air. This treatment answers best in the summer season. The wet pack should be used with caution, beginning with a temperature of 88° F. and going down to 77° F. Wet rubbings are useful, beginning with a temperature of 77° F. and going down to 65° F.

Electrical treatment exercises a favorable influence on the progress of the disease. The constant current is usually employed, and both electrodes should be placed on the vertebral column—one in the lumbar region and the other at the nucha. One pole, say the lower, should be fixed and the other slowly moved along the back so as to come in contact with every part of the vertebral column; then the upper pole should be fixed, and the lower one slowly moved along the back. The direction of the current is of no consequence. The current should be applied daily, but each sitting should not be for more than from three to six minutes' duration. Strong currents should be avoided, especi-

ally in irritable persons with severe pains. The application of the faradic brush to the surface of the body is useful when there is marked anæsthesia. The positive pole should be placed on the sternum, and the negative pole, with the brush, drawn repeatedly along the vertebral column and then along the extremities.

Favorable results have been obtained by Dr. Mortimer Granville by means of nerve vibration. I have given a trial to this treatment in some cases, and found it very grateful to the patients, although I cannot say that it appeared to be of much use in other respects.

Nerve-stretching has been used as a means of treatment in locomotor ataxia. It is possible that the lancinating pains may be rendered more tolerable by this treatment, but after witnessing the effects of the treatment in a few cases, I am not disposed to hazard further trial of it myself or to recommend it to others.

The treatment of tabes by complete rest in the recumbent position, which was first recommended by Dr. S. Weir Mitchell, appears to be one of the most successful methods which can be adopted, but it is only in exceptional cases that patients can be induced to lie in bed for any considerable period of time so long as they are able to move about at all.

Amongst various remedies which have been used against the lancinating pains may be mentioned sinapisms, blisters, warmth, Priessnitz's cold-water compresses, belladonna plasters, chloroform and other liniments, faradization or galvanization of the hyperæsthetic spots (stabile anode), subcutaneous injections of morphine, and large doses of the bromide of potassium, or of the hydrobromate of quinine, and large doses of iodide of potassium when the symptoms indicate the presence of meningitis. One patient told me that he found most relief from the lancinating pains by hot ironing the painful joints. When anæsthesia is present cutaneous faradization may be employed with benefit.

In vesical weakness faradization of the bladder, either with or without the aid of the bladder electrode, may be usefully employed. Cystitis must be treated in the same way as in chronic myelitis.

Constipation must be combated by mild aperients, enemata, and regulation of diet, or by faradization.

The treatment of the diseases of the cerebellum must depend upon the nature of the lesion. In hemorrhage the treatment is the same as for cerebral hemorrhage, and it seems to me that there may be a possibility of our being able, in some cases, to evacuate the pus through the temporal bone. In tubercular growths, glioma, and other tumors, little can be done beyond palliative treatment, unless, indeed, the growth be of syphilitic origin, when an energetic antisyphilitic treatment may procure partial or, on rare occasions, more or less complete recovery.

CHAPTER X.

VASCULAR DISEASES OF THE SPINAL CORD.

I. ANÆMIA, THROMBOSIS, AND EMBOLISM OF THE SPINAL CORD (MYELOMALACIA, VASCULAR SPINAL SCLEROSIS).

Etiology.—The predisposing causes of spinal anæmia are congenital narrowness of the calibre of the vessels, weakness of the heart, and undue excitability of the vaso-motor nerves giving rise to vascular spasm. Anæmia of the cord is often only an expression of general anæmia caused by hemorrhages, and dyscrasic conditions like chlorosis, pernicious anæmia, and other cachectic diseases. Spinal anæmia occurs much more frequently in the female than in the male sex. In some cases the anæmia is caused by extensive atheromatous changes of the spinal bloodvessels, or by the arterio-capillary fibrosis which accompanies chronic interstitial nephritis. When the vessels are extensively changed by atheroma, thrombosis may take place to such an extent that softening occurs, a condition which has been named *myelomalacia*, but arterio-capillary fibrosis is more likely to give rise to sclerosis of the walls of the vessels and of the tissues in their immediate vicinity, a condition which may be named *diffused vascular sclerosis*. The anastomosis of the spinal vessels is so free that embolism of them is not likely to give rise to extensive softening, but the multiple embolism which occurs in cases of chorea sometimes gives rise to small spots of necrotic softening in the cord. The greatest degree of anæmia of the spinal cord is caused by compression, thrombosis, or embolism of the abdominal aorta above the point of origin of the lumbar arteries.

Symptoms.—When the abdominal aorta is suddenly obliterated by a large embolus the lower extremities become quickly paralyzed, and every form of sensibility is lost in them; the reflex excitability and the tendon-reactions are also abolished, and the bladder and rectum are paralyzed. If the obliteration of the aorta takes place more slowly the symptoms develop gradually and are less severe; they consist of a feeling of numbness in the lower extremities, which also feel weak and easily tired on slight exertion. Nothing is known of the symptoms caused by ischæmia of the cervical region of the spinal cord. The ischæmia caused by vaso-motor spasm is characterized by the same

kind of symptoms as those resulting from aortic obstruction, although they are much less severe in the former than in the latter. In this form of anæmia some source of peripheral irritation can usually be discovered, and the paralytic symptoms generally disappear if the source of irritation be removed.

In the spinal anæmia which results from general causes the most constant symptoms are motor weakness, first in the lower and subsequently in the upper extremities, and tremor on the slightest exertion, and in some cases complete paralysis may be developed. The sensory disorders consist of various paræsthesiæ, pain, hyperæsthesia, and, on rare occasions, a slight degree of anæsthesia. The cutaneous reflexes and tendon-reactions are often exaggerated, and the sphincters are not, as a rule, affected. It is said that the symptoms improve on lying down, and on the contrary are made worse when the patient assumes the erect posture. The spinal symptoms are associated with the usual symptoms of general anæmia or of chlorosis.

In *myelomalacia* the symptoms are very obscure. The patient first complains of numbness and feebleness of the lower extremities, and these symptoms gradually increase in intensity until ultimately anæsthesia and complete paralysis of the lower extremities are fully established. The cutaneous reflexes are also diminished and finally abolished in the lower extremities, but the patellar tendon-reactions may be retained for a long time. As the disease advances the sphincters of the bladder and rectum become gradually paralyzed, and ultimately the patient dies from bedsores, marasmus, and pyæmia. Hyperæsthesia, spasms, or increase of the cutaneous reflexes, are said to be absent throughout the course of the disease.

In *diffused vascular sclerosis* the symptoms pursue a chronic course, and are for a long time like those of a primary lateral sclerosis, with an admixture of indefinite sensory disorders. The sphincters are not affected until a late period of the disease.

II. HYPERÆMIA AND HEMORRHAGE OF THE SPINAL CORD.

1. Spinal Hyperæmia.

Etiology.—Hyperæmia of the cord is produced by excessive functional activity, such as occurs in severe exertion or violent sexual excitement. Congestion being present in the early stages of inflammation, all the causes of myelitis likewise give rise to hyperæmia. It may also be caused by various toxic agents, such as strychnine, carbonic

oxide, and alcoholic excess. Passive hyperæmia is caused by those conditions which favor general venous congestion, such as diseases of the heart and lungs, or convulsive diseases like tetanus, epilepsy, and eclampsia.

Symptoms.—The most prominent symptoms of spinal hyperæmia are pains in the loins, and along the spine, of a dull oppressive character, and which are not increased on pressure. The patient complains of tingling, formication, and tearing pains in the lower extremities, and a slight degree of hyperæsthesia of the skin, a girdle sensation, and a moderate increase of reflex activity may be present. The motor symptoms consist of transitory jerking of the muscles and trembling of the limbs, and Rosenthal says that the electrical excitability is increased. In severer cases the patient complains of numbness in the lower extremities, the limbs feel heavy and feeble, and the patient feels tired on slight exertion, but complete paraplegia probably never occurs. The sensory and motor disorders may occasionally implicate the upper extremities, and in those cases respiration is said to have been disturbed. The symptoms are said by Brown-Séquard to be aggravated by lying on the back with the head and legs raised, while they are relieved by lying on the face, standing or walking, and the patients are said to feel worse before getting up in the morning. If the congestion is sufficiently intense to give rise to serous effusion the patients feel better in the horizontal position. There is no fever, and the pulse may be either quick or slow if the hyperæmia extend to the spinal centres of cardiac innervation. Passive must be distinguished from active congestion by the presence or absence of general venous congestion. Spinal hyperæmia ends in recovery unless the case prove fatal from a cardiac or some other complication.

2. Spinal Apoplexy (Hæmatomyelia).

Etiology.—Spinal apoplexy is more common in youth and middle age than in old age, attaining its greatest frequency between twenty and thirty-five years of age. Men are more subject to the disease than women in the proportion of about four to one. Amongst the exciting causes are traumatic injuries, suppression of accustomed discharges, active congestion of the cord, and the presence of a rapidly growing tumors like glioma.

Symptoms.—The disease may be preceded by obscure premonitory symptoms like those of congestion of the cord, which may last from a few hours to several days, but even in those cases the onset of the

characteristic symptoms is always sudden, and complete paraplegia develops in the course of a few minutes or at most an hour. During the development of the paralysis the patient often complains of a violent pain, which is either localized at a particular spot, extends along the spinal column, or shoots in the form of a girdle round the body; but when the paralysis has become complete the pain usually disappears, although the spinous processes of the vertebræ over the seat of the lesion may remain tender to pressure, or to the cathode of a galvanic current, for a considerable time. When the lower cervical region is implicated the paraplegia extends to the upper extremities, the respiratory muscles are paralyzed, and the breathing is diaphragmatic. The paralyzed muscles are at first flaccid, and the reflexes and tendon-reactions are absent in the lower extremities, from the shock, even in the cases in which the lesion is situated high up.

In the early stage there is more or less complete anæsthesia of every form of sensibility, having in most cases the same distribution as the motor paralysis, but when the lesion is unilateral there may be hyperæsthesia of one of the lower extremities and anæsthesia of the other even although both limbs may be paralyzed. Retention of urine may be present from the first from interference with the conducting paths, or the shock may give rise to a transient incontinence, but permanent incontinence of urine and feces only occurs when the automatic centres of the bladder and rectum are invaded by the hemorrhage or are secondarily implicated by the inflammatory reaction. Priapism is a frequent symptom during the first few days, and the temperature of the paralyzed extremities may be raised from 0.2° to 2° C. above that of the axilla. In the course of a few days the symptoms of secondary myelitis may be superadded to those caused by the primary lesion, and then the patient may suffer from pains of various forms, and twitching movements and jerkings of the extremities. If the myelitis extends downwards so as to reach the level of the upper lumbar region of the cord the sphincters become paralyzed, cystitis and bedsores appear, and the patient dies from septic fever; and if it ascend to the upper cervical region the patient dies from asphyxia.

If the patient survive the stage of secondary myelitis signs of improvement begin to show themselves. In those cases in which there is hyperæsthesia of one lower extremity and anæsthesia of the other, the anæsthetic extremity gradually regains its motor power and it may ultimately be almost completely restored, but the hyperæsthetic extremity becomes the seat of a permanent spasmodic paralysis with exaggerated reflexes and tendon-reactions. If the upper extremities are paralyzed the one on the anæsthetic side may also recover, while the paralyzed

muscles of the opposite upper extremity remain permanently paralyzed and undergo atrophy, being innervated from the level of the lesion.

The sensory disorders also undergo some improvement. The hyperæsthesia may gradually disappear from the lower extremity which is permanently paralyzed, but the anæsthesia often remains more or less permanent in the lower extremity which has recovered motor power, and it also extends over the half of the body on that side up to the area of distribution of the sensory nerves derived from the upper limit of the lesion. On the paralyzed side, although the sensibility has become normal below the level of the lesion, yet a band of anæsthesia extends round the half of the body corresponding to the vertical extent of the lesion.

The immediate and permanent symptoms will, of course, vary according to the extent and situation of hemorrhage, and in cases which make a partial recovery the lesion is generally limited to one-half of the cord. In small hemorrhages it may not be possible to make a certain diagnosis of the nature of the lesion. When, for example, the hemorrhage remains limited to the anterior cornua the chief symptom will be the sudden occurrence of an atrophic paralysis and the case should be regarded as very acute anterior poliomyelitis.

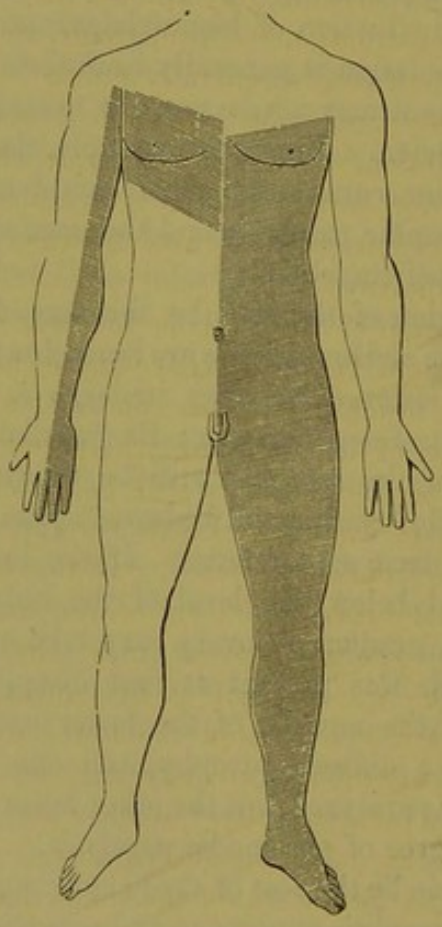
If the *lumbar* region of the cord be the seat of the hemorrhage the symptoms of paralysis and anæsthesia are limited to the lower extremities; the muscles of the lower extremities undergo a diffused atrophy and manifest the reaction of degeneration; the patellar tendon-reactions are lost, but the reflex of the sole may still be retained; the bladder and rectum are paralyzed; cystitis and bedsores appear at an early period, and the patient dies from septic fever. If the lesion is, however, very limited and situated below the level of the automatic centres of the bladder and rectum gradual recovery may take place, the paralysis of the sphincters which was present at first disappears, the sensory disorders improve, but the muscles of the lower extremity on the side of the lesion undergo a diffused atrophy, and one or more groups may remain permanently paralyzed, but the other lower extremity often gives signs of a slight degree of spasmodic paralysis.

If the *dorsal* region be the seat of the hemorrhage the patient recovers, unless a secondary myelitis supervene and prove fatal. The permanent symptoms will be a spasmodic paralysis of the lower extremity on the side of the lesion and an atrophic paralysis of the dorsal, abdominal, or intercostal muscles supplied from the level of the lesion. There will be anæsthesia of the lower extremity and of the half of the body on the opposite side to that of the inner paralysis, and a band of anæsthesia

surrounding the body on the side of the lesion and corresponding to the vertical extent of the lesion.

If the lower cervical or upper dorsal regions are the seat of the hemorrhage the permanent symptoms will be a spasmodic paralysis of the lower extremity and an atrophic paralysis of groups of muscles of the upper extremity and of the upper intercostal muscles on the side of the lesion, hemianæsthesia on the opposite side of all points below the level of the upper limit of the lesion, and on the side of the lesion a band of anæsthesia encircling half the body and an area of anæsthesia of variable distribution in the upper extremity. In addition to the motor and sensory disorders, oculo-pupillary phenomena are found on

FIG. 170.



the side of the lesion. In a case of spinal hemorrhage which has been under my observation for many years the permanent symptoms are a spasmodic paralysis of the right lower extremity and an atrophic paralysis of the intercostal muscles from the ninth upwards, and of the small muscles of the hand and the flexors of the fingers and wrist. The hand assumes the well-known claw-form met with in cervical pachymeningitis

when the lesion is situated on a level with the eighth cervical and first dorsal nerves. On the left side, the lower extremity and the half of the body up to the level of the clavicle are almost completely anæsthetic, the skin of the right lower extremity and of the half of the body up to the level of the tenth rib, which was at first hyperæsthetic, is now normal, but above the tenth rib a band of anæsthesia encircles half the body up to the level of the clavicle, while the anæsthetic area extends down the inner aspect of the arm and forearm, the ulnar half of the palm and back of the hand and two and a half fingers (Fig. 170). The oculo-pupillary symptoms are well marked in the right eye.

If the lesion extends further up in the cervical region other groups of muscles will be paralyzed in the upper extremity and the area of distribution of the anæsthesia will also be different from that of the case just described. If the hemorrhage occur above the origin of the phrenic nerves, rapid death by asphyxia is inevitable.

Morbid Physiology.—Anæmia of the spinal cord had been experimentally produced in animals by Stenon as far back as 1667, and since that time by various other physiologists, but the most complete and conclusive experiments of this kind were performed by Kussmaul and Tenner. These experimentalists first tied the subclavian arteries, and so arrested the circulation through the vertebral arteries. Compression upon the descending portion of the arch of the aorta now caused more or less complete arrest of the circulation in the cord, and after a minute and a half the animals operated upon became paralyzed in their posterior extremities, but the paralysis soon extended upwards, and death resulted from paralysis of respiration. Inert powders have been injected into the aorta of animals by Panum and Vulpian, with the result of obliterating a large number of the small bloodvessels of the abdominal organs and of the lower part of the spinal cord. The operation was almost immediately followed by paralysis of both sensation and motion of the posterior extremities, and after death foci of softening were found in the spinal cord. These experiments show that arrest of the circulation through the cord, either by occlusion of the large vessels from which the cord derives its arterial supply, or of a large number of the vessels of the cord itself, is followed by a cessation of the spinal functions.

Hyperæmia of the spinal cord gives rise at first to slight irritative sensory and motor phenomena, but these soon give place to depressive symptoms. The primary irritative symptoms are caused by the free supply of nourishment conveyed to the cord by the well-filled vessels, but as the vessels become greatly distended the nervous tissues are com-

pressed, the material exchanges are in great part arrested, and the irritation gives place to depressive symptoms.

Hemorrhage of the spinal cord is said by Hayem and Charcot to be always a secondary result of hyperacute myelitis or of some other disease of the spinal cord. It is probable that hemorrhagic infiltration is always the result of a very acute central myelitis, and a hemorrhagic clot sometimes occurs as a grave and generally fatal complication of a chronic spinal disease like progressive muscular atrophy. In other cases, however, the sudden onset of the disease and the absence of all irritative symptoms as well as of power, seem to indicate that the hemorrhage is primary, and that any evidence of myelitis which may be present in the cord after death is caused by a secondary myelitis. The character of the symptoms will depend upon the extent and localization of the hemorrhage, the degree of secondary myelitis, and the tracts of the cord which have been destroyed.

Treatment.—In the treatment of *spinal anæmia* the causes must be removed by a tonic and stimulating treatment. In aggravated cases the patient should be laid on his back and his legs raised, and this position should be maintained during the night and for a considerable portion of the day. Strychnine may be administered in spinal anæmia, and the constant current should be applied daily to the vertebral column, while the faradic current may be applied to the paralyzed muscles if the reflexes are not in excess. Chapman's spinal bag, filled with hot water, or hot sand-bags, may be applied along the back. The diet should be generous and moderately stimulating.

In *spinal hyperæmia* leeches may be applied along the spine, especially when the affection is caused by the suppression of accustomed discharges. A saline purgative may be of use by unloading the vessels and lowering the arterial tension. Ergotine and belladonna are the favorite internal remedies. The patient should be directed to lie on his side, or in the prone position, and Chapman's ice-bag may be applied to the spine. The cold effusion, douches, cold packing, and sea baths may take the place of the ice-bag. The diet should be plain, nourishing, and unstimulating, and everything which might increase the hyperæmia, such as sexual and alcoholic indulgence, must be avoided.

Spinal hemorrhage must be treated at first like spinal hyperæmia, and after a time like an ordinary case of myelitis.

CHAPTER XI.

MYELITIS.

The forms of myelitis which affect exclusively the embryological systems of the spinal cord have already been considered, and consequently there remain for discussion only those kinds in which the gray and white substances are indiscriminately attacked, and in which the symptoms of atrophic and spasmodic paralysis, along with various disorders of sensation and of the functions of the bladder and rectum, are associated in the same case. When inflammation affects several of the embryological systems of the spinal cord it may be named *diffused* or *indiscriminate myelitis.*

Etiology.—Many cases of myelitis begin in the absence of any recognizable exciting or predisposing cause. Males appear to be more liable to myelitis than females, and the majority of cases occur between ten and thirty years. Sexual excess and severe bodily exertion act as predisposing causes.

The most usual exciting causes are wounds, fractures, and contusions of the vertebral column, extension of inflammation to the cord from disease of the membranes or vertebræ, and exposure to cold when the body is overheated. Myelitis may also be caused by an ascending neuritis set up by irritative lesion of the joints and internal organs, especially the genito-urinary organs. It may also occur as a complication or sequel of the acute specific fevers, acute rheumatism, and severe puerperal diseases, and a very acute form of myelitis is liable to occur in syphilitic subjects.

Any condition, indeed, which narrows the spinal canal and leads to compression of the cord may become a cause of myelitis. Of these causes caries of the vertebræ is the most frequent and important. The cord may be subjected to compression by the bodies of diseased vertebræ, displaced fragments of bone, or protruding intervertebral cartilages. More frequently, however, the cause of compression is to be found in the accumulation of inflammatory products around the membranes, or secondary inflammatory thickening of the dura mater itself. Amongst other diseases of the vertebræ which occasionally cause compression of the cord may be mentioned exostosis, syphilitic new formations, dry arthritis of the vertebræ, and thickening of the odontoid

process of the axis. External tumors of all kinds, such as carcinomata, sarcomata, and echinococci may, by effecting an entrance into the vertebral canal, occasion more or less sudden compression of the cord, or even give rise to a secondary myelitis in the absence of compression. Intramedullary, meningeal, and perimeningeal tumors of all kinds give rise to myelitis by compression, and of these it is important to remember that syphilitic deposit is the most frequent.

Symptoms.—The spinal phenomena, in acute cases, are often preceded by premonitory symptoms consisting of general malaise, slight pyrexia, chilliness, headache, general depression, loss of appetite, and aching of the limbs. The spinal symptoms generally manifest themselves by sensory disturbances consisting of girdle pains; numbness, and various other paræsthesiæ, along with shooting, burning, and other sensations in the limbs; by pain in the back over the vertebral column, which is accompanied by tenderness on pressure of the spinous processes, and by superficial sensitiveness that can be elicited by passing a hot sponge or the cathode of the galvanic current over the part. Patients complain at times of painful, dragging sensations in the bladder and rectum, gastralgic attacks, and neuralgic pains in other viscera. As the disease advances the paræsthesiæ and pains give place to anæsthesia, which may be partial or complete, although even when sensation is wholly lost to objective examination the patient may complain of severe pains in the part—*anæsthesia dolorosa.* In most severe cases the anæsthetic lower part of the body is separated from the normal skin by a pretty sharply defined line.

The *motor* symptoms consist at first of twitchings of individual muscles or of entire extremities, and occasionally the spasm may increase to a condition of tetanic rigidity. But the symptoms of motor irritation soon give place to paralysis, and loss of motor power is sometimes developed with so much rapidity that we speak of *apoplectiform* myelitis. When paralysis is rapidly developed the muscles are perfectly flaccid, and when the limbs are raised they fall like inert and lifeless masses. If the patient survive, symptoms of motor irritation may reappear in the affected limbs, consisting of spontaneous twitchings of the paralyzed muscles, which are generally accompanied by severe shooting pains. The muscles now become tense, and the tension is increased by passive movements of the limbs, while contractures become established which fix the lower extremities generally in positions of extension and only occasionally in flexion. The most common forms of paralysis are paraplegia and hemiparaplegia, while complete paralysis of all four extremities occurs occasionally.

The *reflex* excitability and tendon-reactions vary according to the

seat of the disease. When the disease involves the lumbar enlargement both the reflex of the sole and the patellar tendon-reaction are abolished; when it involves the upper end of the lumbar enlargement the reflex of the sole is exaggerated, but the patellar tendon-reaction and the cremasteric reflex are lost; and when the lesion is situated in the dorsal region the reflexes of the sole, of the cremaster, and of the patellar tendon are exaggerated.

The bladder, rectum, and sexual organs are generally affected in acute myelitis. At first there may be spasmodic closure of the sphincter of the bladder, so that a catheter has to be used, but incontinence of urine soon supervenes, vesical paralysis being, indeed, sometimes one of the earliest symptoms of the disease. In severe cases the urine becomes alkaline and sometimes bloody after the seventh or eighth day; it then contains numerous crystals of the triple phosphates and putrefaction bacteria, and there is considerable muco-purulent deposit. The bowels are obstinately constipated, and the abdomen becomes distended with flatus, while soon the stools are passed involuntarily. *Priapism* is not an uncommon symptom of acute myelitis, the erection being usually incomplete, but generally persisting for days.

The *vaso-motor* disturbances are variable. The paralyzed limbs are often œdematous, and generally cold, although occasionally elevation of temperature has been observed in them. The perspiration is sometimes increased, sometimes diminished.

The trophic disorders consist of acute bedsores, which may make their appearance as early as from the second to the fifth day and are situated over the sacrum, trochanters, and other exposed situations; they often pursue a rapid course and prove fatal by pyæmia. When the disease is situated in the lumbar region the paralyzed muscles undergo a diffused atrophy, and then the affected nerves and muscles manifest the "reaction of degeneration." When the lesion is localized in the dorsal region the muscles retain their nutrition and the electrical reactions are normal.

Psychical symptoms are not often present in acute myelitis, although in children the symptoms may be ushered in by headache, delirium, and general convulsions; when cerebral symptoms occur in the adult they are probably due to the accompanying fever.

When the erector muscles of the spine are paralyzed the patient is unable to sit up in bed, and when the abdominal muscles are paralyzed all forcible expiratory acts are rendered feeble, and consequently the air-passages cannot be cleared of mucus, a difficulty which is still further increased when the intercostal muscles are paralyzed. When the cervical portion is implicated some or all of the muscles of the upper

extremities are paralyzed and various sensory disorders appear in them. In addition oculo-pupillary symptoms are present, and double optic neuritis has been observed on rare occasions; while the pulse may either be much accelerated or retarded, and the patient suffers from palpitation, irregularity of the heart's action, and unpleasant sensations in the præcordial region.

As the disease ascends the movements of the diaphragm become feebler, and as inspiration is now carried on chiefly by means of the cervical muscles death by asphyxia is imminent; when the medulla oblongata becomes involved articulation and deglutition are interfered with, and the patient dies from respiratory paralysis.

In acute myelitis the early stage of the disease is accompanied by fever, and the temperature may remain persistently high throughout the whole course of the disease, while there may be hyperpyrexia at death. In other cases the fever is slight and may disappear entirely in the course of the disease, but in the terminal stage a fever of remittent type appears which is caused by septic infection from the cystitis, bedsores, and pyelo-nephritis.

Acute myelitis is always rapidly developed, but when the acute symptoms have subsided the disease may pass into a chronic myelitis, and the symptoms may then remain stationary for months or years. In other cases acute myelitis pursues an ascending course and becomes quickly fatal. When the lumbar region of the cord is the chief seat of the disease it proves fatal after many months from the exhaustion caused by septic fever. In some cases complete recovery may take place and a favorable termination may occasionally result even after the formation of bedsores. In most cases, however, recovery is only partial, and one or more groups of muscles remain permanently paralyzed and atrophied.

Chronic myelitis is sometimes a sequel of acute myelitis, but in most cases the disease begins slowly and is gradually developed. The symptoms often begin by paræsthesia in the lower, and occasionally in the upper extremities, and a girdle sensation, and at the same time the patient complains of a feeling of weakness and heaviness of the limbs, and probably suffers from jerkings of the legs in bed at night. He also complains of a sensation of stiffness of the legs, which is worse in the morning and at starting to walk than when the limbs have been moderately exercised. As the disease advances the stiffness and weakness of the legs become worse, and after a time the paralysis assumes the typical form of spastic paraplegia, or occasionally of a spinal hemiplegia, and now the lower half of the body is also more or less anæsthetic. The course of chronic myelitis is very variable. When the disease reaches a certain degree of intensity it may remain stationary

for many years, or periods of improvement may alternate with relapses and exacerbations. The disease may, indeed, last a lifetime without producing dangerous symptoms. Complete recovery is, however, rare, and the majority of cases terminate fatally either from bedsores, cystitis and pyæmia, occasionally from bulbar paralysis, or more frequently from pneumonia or other intercurrent disease.

VARIETIES OF DIFFUSED MYELITIS.

The following varieties of diffused myelitis may be distinguished: 1, Central myelitis; 2, bulbar myelitis; 3, transverse myelitis; 4, wounds of the spinal cord; 5, compressive myelitis; 6, unilateral myelitis; 7, meningo-myelitis; 8, universal progressive myelitis; and 9, disseminated myelitis. The various inflammations of the spinal cord may be subdivided into *acute* and *chronic* myelitis.

1. Central Myelitis.

Symptoms.—This affection begins suddenly by paræsthesiæ and other sensory disturbances, which are soon followed by complete anæsthesia and paralysis with flaccidity of the lower extremities, and by paralysis of the bladder and rectum. The reflex excitability is abolished, and the paralyzed muscles undergo rapid atrophy, and manifest the "reaction of degeneration" at an early period. The further progress of the disease is marked by acute bedsores, cystitis with ammoniacal urine, œdema of the paralyzed limbs, arthropathies, more or less intense fever, and progressive advance of the paralysis upwards, so that the intercostal muscles and those of the upper extremities become successively paralyzed, and ultimately the muscles of articulation and deglutition are invaded, and the patient dies from respiratory paralysis.

The course of acute central myelitis is very similar to that of acute ascending paralysis, but in the latter the bedsores as well as the vesical and rectal disorders of the former disease are absent. In other cases the symptoms become more or less chronic, and the disease may then be mistaken for chronic atrophic spinal paralysis, but in the latter disease the sensory disorders and the bedsores of the former affection are wanting. In some cases the symptoms develop with almost apoplectiform rapidity, the lower extremities becoming completely paralyzed in the course of a few minutes, or, at most, in the course of an hour or two. In such cases the gray matter of the cord has been found soft-

ened and mixed with red blood-corpuscles, and the disease has consequently been named *hæmatomyelitis*. It may also be named hyperacute central myelitis.

2. Bulbar Myelitis.

Symptoms.—Acute bulbar myelitis begins suddenly with violent headache, giddiness, vomiting, distressing hiccough, various paræsthesiæ, and difficulty of articulation and deglutition, but, contrary to what occurs in an apoplectic attack, consciousness is retained. After a short time the inferior muscles of the face, the muscles of the tongue, soft palate, and pharyngeal muscles become paralyzed in various degrees and combinations, and in some cases the ocular muscles, especially the external recti, may be implicated. Disorders of respiration appear at an early period, the respirations are hurried and irregular, and there may be an intense feeling of oppression, dyspnœa, and cyanosis. The pulse is small, quick, and irregular or intermittent. One, two, or all the extremities may be paralyzed, or paralysis of the limbs may be absent. The patient may complain of pain and formication in the limbs, but there is anæsthesia. The bladder and rectum may become paralyzed towards the terminal period of the disease. The disturbances of circulation and respiration now increase, the patient becomes unconscious, and death from asphyxia results.

Chronic progressive bulbar paralysis has already been described. A chronic form of bulbar paralysis may also result from necrotic processes, and from inflammation set up by tumors and other morbid processes in the neighborhood of the medulla, but this form of bulbar myelitis does not demand separate description.

3. Transverse Myelitis.

Symptoms.—The symptoms of transverse myelitis will vary according as its course is acute or chronic, and according as it is caused by wounds and other injuries of the cord, by the growth of tumors, or by the extension of inflammation from other tissues, or as it begins as an idiopathic affection. In this place, however, we have to do chiefly with the localizing symptoms, and for the purposes of description the affection may be divided into *a*, dorso-lumbar; *b*, dorsal; and *c*, cervical transverse myelitis.

a. Dorso-lumbar transverse myelitis may begin more or less suddenly by fever, paræsthesia of the lower extremities, retention of urine,

and startings of the lower extremities, but these symptoms soon give place to sensory and motor paralysis of the limbs and paralysis of the sphincters of the bladder and rectum. The lower extremities become œdematous, and their muscles may undergo a diffused atrophy, and may manifest the reaction of degeneration. The patellar tendon-reactions, which were possibly at first exaggerated, now become lost, and the cremasteric reflex is also lost, but the reflex of the sole is often exaggerated. The neck of the femur is occasionally the subject of spontaneous fracture, and arthropathies of other joints may appear. In acute cases of this kind the patient generally dies at an early period of the disease from the usual cystitis, bedsores, and septic fever.

The *chronic* form of the disease begins gradually by girdle pains in the region of distribution of the ilio-hypogastric and ilio-inguinal nerves, while the patient complains of shooting pains, and various paræsthesiæ in the lower extremities. The patient suffers from cramps in the calves of the legs, and is soon fatigued, while the movements are stiff and constrained. After a time the lower extremities become rigid by contractures, the cutaneous reflexes and tendon-reactions are exaggerated in them, and the gait is of the spastic variety. As the disease advances the sensory disorders become more profound, and anæsthesia of the lower extremities becomes established, while the patient becomes completely paraplegic. The lesion now usually invades the lumbar enlargement, and when it creeps down to the level of the second or third lumbar nerves the cremasteric reflex is lost, the extensor muscles of the knee undergo atrophy, the patellar tendon-reaction is abolished, and the sphincters are soon afterwards paralyzed. With a still further downward extension of the lesion all the muscles of the lower extremities undergo a diffused wasting and lose their tension; the limbs become œdematous, the reflex of the sole may, after a time, be lost; bedsores, which have, indeed, already appeared over the sacrum, now form over the trochanter and other points subjected to pressure; the urine is alkaline and contains pus; and the patient dies from pyæmia.

b. Dorsal Transverse Myelitis.—In this form of myelitis both the sensory and motor paralysis extend higher than in the dorso-lumbar variety, but the condition of the lower extremities, with regard to paralysis, contractures, spastic gait, exaggerated reflexes, and tendon-reactions, is the same as in the early stage of that disease. When the lesion is situated in the upper part of the dorsal region the muscles of the back and abdomen are liable to be paralyzed, and paralysis of the abdominal muscles renders urination, defecation, and forcible expiratory acts difficult, and consequently the patient is troubled with constipation, and being unable to clear the bronchial tubes thoroughly from mucus

he is liable to succumb to a slight bronchial catarrh. Double optic neuritis has occasionally been observed. The level at which the cord is diseased may be determined by the level of the girdle pains and anæsthesia, and by an examination of the cremasteric, gluteal, abdominal, epigastric, dorsal, and scapular reflexes.

c. Cervical Transverse Myelitis.—This form of myelitis generally begins with pain in the back of the neck, and the muscles of the neck become rigid, while owing to the predominant action of those of the back, often of one side, the head is generally rotated and retracted. The upper extremities now become subject to shooting pains and muscular startings, provided the lesion involves the origin of the brachial plexus, but these symptoms of irritation soon give place to sensory and motor paralysis. The paralysis of the upper extremities is of the atrophic variety, and its distribution, with the consequent deformities, will depend upon the level of the lesion, while it may be accompanied by œdema and arthropathies. The lower extremities become affected with a spasmodic paralysis with exaggerated reflexes and tendon-reaction. There may also be complete or incomplete anæsthesia of the body and lower extremities below the level of the lesion, and of the upper extremities on a level with the lesion, the distribution of the last depending upon the posterior roots which are implicated. The pupils are, in some cases, dilated, in others contracted, or often first dilated and then contracted; the size of the palpebral fissure may be increased or diminished; and the face may be unusually pale or flushed, and it is often covered by perspiration. In some cases oculo-pupillary symptoms are absent. The establishment of paralysis may be preceded by anomalous symptoms like cough, dyspnœa, sweats, wasting of the muscles of the arm and neck, pains between the shoulders or at the back of the neck, severe attacks of vomiting, hiccough, difficulty of deglutition, and great slowness of the pulse. The inflammatory process is liable to ascend to the medulla oblongata, and death results from respiratory paralysis.

4. Wounds of the Spinal Cord.

Symptoms.—The symptoms caused by wounds and injuries of the cord may be divided into (*a*) those caused by comparatively slight injuries of the cord, such as simple incised and punctured wounds, and (*b*) those caused by the more serious lesions, such as sudden compression and tearing of the cord.

a. The symptoms which indicate that an injury by cutting or stabbing in the neighborhood of the spine has penetrated the cord will at first be those caused by loss of conduction to and from the brain

in the portions situated below the seat of the injury. At the moment the injury is received paraplegia, hemiparaplegia, or general paralysis is suddenly developed. If the cord is completely divided, there is complete anæsthesia of the paralyzed parts; but if only one-half of the cord is divided the anæsthesia is situated on the side opposite to the lesion, while girdle pains may be present on a level with the lesion. If the injury be of considerable extent paralysis of the bladder and rectum occurs, and there is also vaso-motor paralysis with increased temperature and redness of the region affected by the motor paralysis. The reflex actions are usually abolished at first, owing to the shock, but if the lesion is situated in the dorsal or cervical region, they may, subsequently be exaggerated. After a time the symptoms of secondary traumatic myelitis complicate those caused by the primary lesion, and then the symptoms will be more or less like those caused by acute idiopathic transverse myelitis.

b. The symptoms which indicate that the cord is crushed or torn in severe injuries of the spine are, in addition to the evidences of local injury of the spine, complete paralysis and anæsthesia of the portion of the body which lies below the seat of the injury. The other symptoms are abolition of the reflex actions, retention of urine and constipation, with meteorism or involuntary evacuations of urine and feces, priapism, in which the organ is turgid but still flexible, and elevation or sometimes depression of the temperature of the body.

When the cervical region is injured the temperature of the body is generally much increased, and often rises still further for some hours after death. In a case of fracture of the fifth and sixth cervical vertebræ, reported by Quincke, the temperature at death was 42.3° C., and eight hours after death it was 43.2° C., and in a case of compression of the cervical region he found the temperature 42.7° C. at death, and 43.6° C. eight hours afterwards. A case of fracture of the spine on a level with the body of the fifth cervical vertebra is recorded by Mr. Jonathan Hutchinson, in which the temperature, which was 99.3° F. in the urethra twenty-four hours after the injury, rose to 106.3° F. at death, thirty-eight hours after the injury, and Mr. F. Churchill has recorded a case of fracture of the same vertebra in which the temperature in the axilla was 110° soon after death. In some cases the temperature undergoes great variations, being in one case as low, at one time, as 94.2° F., and soon afterwards as high as 106.4° F., these variations being apparently independent of bedsores and septic fever. In some cases of fracture of the cervical spine the temperature of the body falls after the injury, and remains low till death.

Another symptom of injury of the cervical region is afforded by a

very feeble, slow, and infrequent pulse, which beats in some cases only forty-eight times in the minute, and it is in these cases, according to Mr. Jonathan Hutchinson, that the temperature falls and the surface is cold to the touch. When the injury is situated in the dorsal or lumbar region the pulse is not affected except by the shock, just as occurs after other severe accidents. The severer forms of injury to the cervical spine are rapidly fatal by paralysis of respiration, but when the patient survives for a few days the symptoms of acute transverse myelitis are superadded to those caused by the injury.

5. Compression Myelitis, Tumors in the Vertebral Canal.

Symptoms.—The symptoms of compression myelitis may be divided into *a*, extrinsic, and *b*, intrinsic symptoms.

a. The extrinsic or prodromal symptoms consist of severe girdle pains, hyperæsthesia of the skin corresponding to the distribution of the pain, eccentric neuralgiform pains generally fixed to one spot, severe pain in the back, local stiffness, and tenderness of the spinous processes. The neuralgic pains are sometimes accompanied by herpetic or bullous eruptions of the skin. Phenomena of motor irritation are superadded to the sensory disturbances in the distribution of the nerves whose roots were first implicated. These consist of twitchings, spasms, and contraction of the muscles supplied by the affected nerves. The irritative motor symptoms are soon followed by weakness and paralysis of single muscles or groups of muscles, the affected muscles undergo a diffused atrophy, the cutaneous reflex actions and the tendon reactions are lost in them, and they may manifest the partial "reaction of degeneration," but not often the severest form of that reaction. As the second stage of the disease approaches the patient complains of paræsthesiæ in the lower half of the body, consisting of tingling, numbness, and sensations of heat or of cold, and after a time more or less anæsthesia is established, but complete anæsthesia is rare in vertebral caries. The motor symptoms consist at this stage of startings of the lower extremities, especially at night, and a sense of weariness and heaviness of the limbs, while the patellar tendon-reactions will often be found exaggerated, unless the lesion is situated in the lower dorsal and lumbar regions, when they may be lost.

When cancer of the vertebral column makes its way into the spinal canal it gives rise to the most agonizing pains. These consist of severe girdle pains, and of pains which radiate along the distribution of certain nerves, such as the crural and sciatic nerves, when the lumbar vertebræ are affected. The skin to which the affected nerves are dis-

tributed is intensely hyperæsthetic, so that the slightest touch is painful. The pains never intermit, but they are liable to paroxysmal exacerbations of great severity, which are difficult to allay even by large doses of narcotics. Patches of anæsthesia may be observed in the skin to which the affected nerves are distributed, but the pain still continues unabated, this condition being named *anæsthesia dolorosa*, and when the symptoms of transverse myelitis are after a time superadded to these sensory disorders, the condition is called paraplegic dolorosa.

In all cases in which the premonitory symptoms of compression or transverse myelitis are present a careful examination must be made to determine the level of the girdle sensations, or the upper limit of the paræsthesia or anæsthesia if any be present, and when once this point has been ascertained the vertebral column on a level with the upper limit of the sensory disorders must be subjected to careful examination. In cases of angular curvature, and in those cases in which an external tumor has made its way into the vertebral canal, decided evidence of the nature of the disease may be at once obtained. And even in those cases in which there are no enlargements or other deformities it may be found that one or two of the spinous processes are tender to pressure or percussion, or sensitive to a hot sponge drawn along the vertebral column, or to the cathode of the galvanic current.

b. The intrinsic symptoms are generally the same as those of transverse myelitis, and are ushered in by paralysis of the lower extremities, which may begin at first as a hemiparaplegia, and afterwards develop into complete paraplegia. The symptoms of acute and chronic transverse myelitis need not be described a second time. In intramedullary tumors the phenomena of atrophic paralysis may predominate, but they are often associated with a spasmodic paralysis with exaggerated reflexes in one of the limbs or in one or more groups of muscles. Multiple tumors are sometimes found in the cord, consisting of sarcoma, tubercle, or syphilis, and the symptoms may then manifest themselves at different elevations, as in acute and chronic disseminated myelitis. In addition to the variable character of the purely spinal symptoms, one or more of the tumors may appear in the medulla oblongata and pons, and the symptoms caused by these tumors may help to clear the diagnosis.

6. Spinal Hemiplegia and Hemiparaplegia (Unilateral Lesion of the Spinal Cord).

Symptoms.—The symptoms of unilateral spinal paralysis may be developed insidiously and gradually, or quite suddenly, the mode of

invasion depending upon whether the lesion consists of a slowly invading disease like the growth of a tumor, or upon a sudden injury like a stab from a sharp-pointed instrument. The most prominent symptoms are a motor paralysis on the side of the lesion which may involve only one leg (hemiparaplegia), or the leg and the arm of the same side (spinal hemiplegia), and a sensory paralysis on the opposite side of the body.

The paralyzed muscles undergo early and rapid atrophy when the lesion is situated in the lumbar enlargement, but when it is situated at a higher level the lower extremity on the side of the lesion undergoes spasmodic paralysis, provided the patient survive sufficiently long for secondary degenerations to occur in the cord, while groups of the muscles of the upper extremity are the subjects of atrophic paralysis if the lesion is on a level with the roots of origin of the brachial plexus. In some cases well-marked ataxia has been observed in the paralyzed leg after return of motor power.

Vaso-motor paralysis is declared, on the side of the lesion, by an elevation of the temperature of the paralyzed limb to the extent of from 1° F. to 3.6° F., but the temperature may be lower than normal when the disease has existed for some time.

Muscular sense and muscular sensibility are also usually diminished on the side of the lesion, but all forms of cutaneous sensibility are increased. Impressions of touch, temperature, and pain are felt with unusual acuteness, and there is an increased power of localizing tactile sensations. At times, however, the hyperæsthesia is limited to a few forms only of cutaneous sensibility. The hyperæsthetic area of the skin is usually bounded by an anæsthetic zone which corresponds with the level and longitudinal extent of the lesion of the spinal cord, and a narrow hyperæsthetic belt which extends to the opposite side may sometimes be detected above the anæsthetic zone (Fig. 171). The reflex actions on the side of the motor paralysis vary, but they are generally increased below the seat of the lesion.

On the side opposite the lesion there are no motor disorders or they are only slight in degree, and both the muscular sense and the electro-muscular sensibility are retained on that side; however, there is more or less complete anæsthesia of the skin, although some forms of sensibility may be involved to a greater extent than others. The anæsthesia extends nearly to the median line of the body and it is often bounded above by a slightly hyperæsthetic region, corresponding to a similar zone on the opposite side. Near the median line anteriorly and posteriorly there is a vertical space of about one inch in breadth in which hyperæsthesia is absent on the side of the lesion, and a similar space on the front and back in which there is an absence of any great degree

of anæsthesia, these phenomena finding a sufficient explanation in the well-known anatomical fact that the sensory nerves of the two sides overlap. There are no vaso-motor disturbances on the anæsthetic side, and reflex action is usually normal, but has occasionally been found increased. The patient sometimes complains of a feeling of constriction and neuralgiform pains on a level with the lesion, and these may at times be more prominent on the anæsthetic and at other times on the hyperæsthetic side. In acute traumatic cases retention or incontinence

FIG. 171.

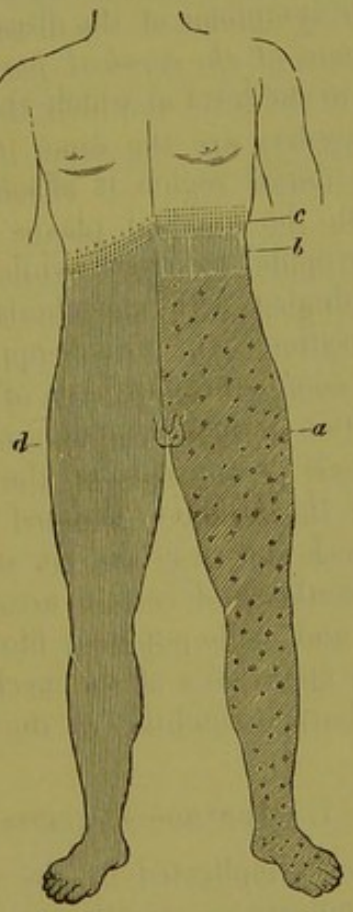


DIAGRAM OF THE CUTANEOUS SYMPTOMS IN UNILATERAL LESION OF THE DORSAL PORTION OF THE SPINAL CORD ON THE LEFT SIDE. (After ERB.)

The diagonal shading (*a*) signifies motor and vaso-motor paralysis; the vertical shading (*b*, *d*) signifies cutaneous anæsthesia; the dotted shading (*a*, *c*) indicates hyperæsthesia of the skin.

is always present at first, but after a time recovery takes place, so that at most only a certain degree of weakness of the sphincters remains. The sexual functions are at times unaffected and at other times more or less weakened.

The trophic disorders consist of acute bedsores which appear on the anæsthetic side, and inflammation of the knee-joint, which appears on the paralyzed side.

Varieties.—The symptoms differ according to the level at which the lesion is situated in the cord.

(1) *Unilateral lesion of the lumbar enlargement* causes an anæsthetic zone, on the paralyzed side, in the area of distribution of one or more of the sensory branches of the sacral or lumbar plexuses, in addition to the other characteristic unilateral symptoms, but there is no belt round the body. The muscles of the paralyzed limb undergo atrophy when the lesion occupies a considerable portion of the lumbar enlargement in vertical extent.

(2) *Unilateral lesions of the dorsal region* of the cord give rise to the most characteristic symptoms of the disease, as already described.

(3) *Unilateral lesions of the cervical portion* give rise to different symptoms according to the level at which the lesion is situated. The motor and sensory disorders are the same in the trunk and lower extremities as when the dorsal region is affected, but when the lesion is situated on a level with the brachial plexus the muscles of the upper extremity undergo atrophic paralysis, while hyperæsthesia of certain parts of the skin is mingled with anæsthesia of other regions, or for certain varieties of sensation. On the side opposite the lesion there is no paralysis, but there is complete anæsthesia of the parts below the level of the lesion. In addition to these symptoms oculo-pupillary phenomena and vaso-motor paralysis of the side of the face are present on the side of the lesion. If the lesion is situated in the upper part of the cervical region, the neck and head on the side of the lesion manifest anæsthesia and hyperæsthesia of certain areas of the skin, and paralysis of the vaso-motor and oculo-pupillary fibres, while on the opposite side there are usually anæsthesia of the neck and a narrow zone of hyperæsthesia, with a normal condition of the face and eye.

7. Meningo-myelitis.

Myelitis is sometimes complicated by the symptoms of meningitis. The symptoms which indicate meningitis are pain and stiffness in the back of the neck, pronounced hyperæsthesia, diffused pains in the back and lower extremities, pain along the vertebral column, and tenderness on pressure of some of the spinous processes of the vertebræ. In some chronic cases the posterior are more involved than the lateral columns, and then ataxic symptoms predominate over those of paralysis.

8. Universal Progressive Myelitis.

This form of myelitis embraces the whole transverse area of the cord, and is characterized by a progressive weakness of the lower extremities

which develops into complete paralysis. The disease usually begins in the lower extremities and pursues an ascending course, but occasionally it begins in the upper extremities and pursues a descending course. The muscles may be in a state of contracture at first but as the gray matter becomes involved they undergo gradual atrophy. The cutaneous reflexes, tendon-reactions, and faradic contractility also gradually diminish and gradually disappear. There may be more or less violent pains in the back, trunk, and extremities, the patient complains of paræsthesiæ and dysæsthesiæ, and after a time there is complete anæsthesia, paraplegia, paralysis of the sphincters, and bedsores.

9. Disseminated Myelitis.

Symptoms.—There are two forms of disseminated myelitis, *a*, the acute; *b*, the chronic.

The symptoms of the *acute* form are very variable, but sensibility is usually more or less impaired, and there is a spasmodic paralysis of the lower extremities with its attendant phenomena, although in some cases one or more groups of muscles may suffer from an atrophic paralysis. The state of the electrical sensibility and of the cutaneous reflexes is variable, but they are generally diminished. The grouping of the symptoms may show that several centres of disease exist, and the diagnosis is rendered more probable if the symptoms of acute myelitis supervene during an attack of variola, or if they develop suddenly in phthisical patients.

Chronic disseminated myelitis is a part of a disease which is widely distributed in the spinal cord and brain, and will be subsequently described under the name of *multiple sclerosis*.

Morbid Anatomy and Physiology.—The morbid anatomy of the various forms of myelitis has already been sufficiently considered, and we shall now make a few remarks to connect the morbid changes with the principal alterations of function observed in this disease.

Acute myelitis gives rise to the various forms of softening of the spinal cord which have already been described. The initial symptoms of irritation, which are present in the early stages of acute myelitis, must be ascribed to the increased irritability of the nerve cells and fibres occasioned by increased nutritive activity during the early stages of the inflammatory process, while the later symptoms of paralysis are caused by the destruction of these elements and their compression by inflammatory exudation. The girdle pains are caused by implication of the posterior roots of the dorsal region in the inflammatory process, while the paræsthesiæ and neuralgic pains of the inferior half of the body

result from irritation of the sensory conducting paths situated in the gray matter and posterior columns of the cord. The motor symptoms are caused first by irritation and subsequently by loss of the function of the motor centres and conducting fibres in the cord. When the anterior gray horns are diseased the resulting paralysis is of the atrophic and when the pyramidal tracts are affected the paralysis is of the spasmodic variety.

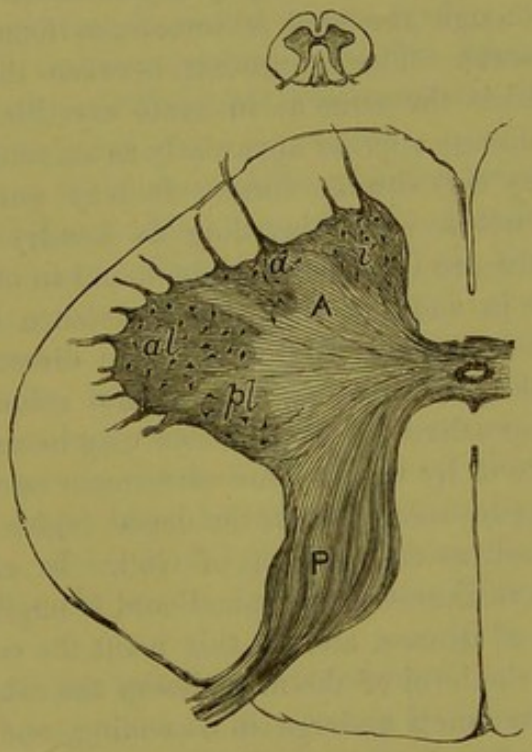
When the myelitis affects the lumbar region the bladder and rectum are involved, the sphincters being first closed by spasm, and subsequently incompetent from paralysis. Priapism must be regarded as a sign of irritation produced either by interference with the conducting paths passing from the brain to the lumbar portion of the cord, or by reflex irritation proceeding from the bladder or skin. The trophic affections of the muscles and bones are caused by lesion of the ganglion cells of the anterior horns, while the cutaneous trophic affections and bedsores appear to be caused by disease of the posterior horns. The vaso-motor disturbances are caused by interference with the centres in the cord, but the spinal mechanisms which preside over these functions are not yet clearly ascertained.

In *acute central myelitis* the softening affects principally the gray substance, which often flows out or becomes depressed under the surface of section when the cord is divided transversely. On microscopic examination the disease is seen to extend in the lumbar region forwards from the central column into the anterior gray horns, the ganglion cells of which may be entirely destroyed and also backwards into the posterior gray horns, while in some cases the white substance, especially the pyramidal tracts, may be implicated in the disease. In the cervical and upper dorsal regions of the cord and in the medulla oblongata the disease is restricted to the central gray column and the accessory cells of the anterior gray horns and their continuations through the medulla, while the fundamental cells in these regions may be entirely unaffected. *Hæmatomyelitis* is like acute central myelitis in every respect except that in the former of the two the central softened mass assumes a hemorrhagic character. In the cases of acute bulbar myelitis observed by Leyden, small indefinite centres of softening were found in the medulla oblongata.

In *acute transverse myelitis* the part of the cord in which the chief lesion is localized is found in various stages of softening, and if the case has assumed a more or less chronic form the usual ascending and descending degenerations are observed above and below the primary seat of the disease. In rapidly fatal cases evidences of inflammation are discovered in the gray matter above and below the seat of the chief

lesion. In the neighborhood of the primary lesion all the ganglion cells of the anterior horns may be destroyed, but in the remote portions of the cord the disease becomes more and more limited to the central gray columns and to the margins of the groups of ganglion cells in the anterior horns. The annexed diagram (Fig. 172) illustrates this condition, and although it was taken from a somewhat protracted case of myelitis, I have observed similar appearances in the cervical region from a case of fracture of the vertebral column in the dorsal region which

FIG. 172.



SECTION OF THE MIDDLE OF THE CERVICAL ENLARGEMENT OF THE SPINAL CORD FROM A CASE OF CENTRAL MYELITIS. (After YOUNG.)

i, The internal; *a*, the anterior; *al*, the antero-lateral; and *pl*, the postero-lateral group. The median area was completely destitute of cells, and a large number of the marginal cells of the different groups of the anterior horn were destroyed or diseased.

proved fatal in five days. The continuation of the central column into the medulla oblongata and its accessory nuclei are generally implicated. In simple incised or punctured wounds of the spinal cord the cut edges project at first beyond the pia and the wound is closed. In a few days the membranes are reddened and covered with fibro-purulent matter, while the neighboring parts of the cord are more or less softened. Crushing of the cord causes softening and disintegration of the cord itself, and congestion and hemorrhage into the membranes. If the patient survive a kind of cicatrix is formed which may enclose cystic cavities containing

clear fluid. In acute myelo-meningitis the usual signs of meningitis will be found along with those of myelitis, and in sections of the cord tinted with carmine a highly stained border is observed which extends more or less deeply into the substance of the cord and consequently this form of disease has been called peripheric or cortical myelitis. *Acute disseminated myelitis* occurs in small spots scattered through the substance of the cord. The morbid change appears to consist chiefly of an increase of the interstitial tissue, which becomes unusually dense and rich in nuclei.

Chronic myelitis gives rise to gray degeneration or sclerosis of the affected parts, although the cord is sometimes found softened in the main focus of disease. The connection between the morbid changes and the symptoms is the same as in acute myelitis. In *chronic central myelitis* the disease appears apparently as an acute central myelitis, and then assumes the chronic form. It may pursue an ascending course, but it is not so surely invading as Landry's paralysis. The morbid appearances are the same as those found in other forms of central myelitis, but in addition the white substance is invaded in the lumbar region, the columns of Goll and the direct cerebellar tracts undergo an ascending sclerosis, and the lateral columns in the lumbar region a descending sclerosis. This disease may be set up by an injury of the sciatic nerve or by the pressure of a tumor on the cauda equina, and then the posterior root-zones in the dorsal region are implicated in the sclerosis as well as the columns of Goll. In *chronic transverse myelitis* the whole thickness of the spinal cord is implicated at the level of the chief focus of disease, and at this point the cord may be found softened. Above the level of the main lesion the columns of Goll and the direct cerebellar tracts undergo an ascending, and below this level the lateral columns undergo a descending sclerosis. Evidences of a central myelitis are also frequently observed, the motor ganglion cells are completely destroyed immediately above and below the main focus, while in the parts which are remote from this lesion the morbid changes are restricted to the central columns and the accessory nuclei. In *compression myelitis* the primary disease may consist of caries, cancer, or some other disease of the vertebral column, but it is unnecessary to describe all these diseased conditions in this place. It may also consist of some forms of meningitis, of an accumulation of pus between the dura mater and vertebræ, or hæmatoma of the spinal dura mater, but these affections will subsequently be described. The following perimeningeal and meningeal tumors have been found in the vertebral canal, namely: *Lipoma*, originating either from the tissue outside the dura mater or from the pia mater; *fibroma* and *fibrosarcoma*, which

may be situated either within or without the sac of the dura mater; *sarcoma*, *myxoma*, and *psammoma*, originating from the pia mater or arachnoid; *enchondroma*, springing from the vertebræ and dura mater; *osteoma*, appearing as disks in the arachnoid, and as a diffused ossification of the dura mater; *multiple fibrous melanoma*, exceptionally found in the spinal canal; *carcinoma*, almost always met with as a secondary growth from the mammæ or vertebræ; *miliary tubercles*, which are always found on the soft membranes; and *syphiloma*, which appears as gummata of the dura or pia mater. *Neuromata*, either true or false, have been found on the roots of the spinal nerves, and are most frequently situated on the cauda equina. Parasitic growths are rarely met with in the vertebral canal, but echinococcus has been occasionally met with, and Westphal has reported a case of cysticercus cellulosæ of the spinal cord.

The following are the *intramedullary* tumors which have been observed, namely: *Glioma*, which may form a rounded tumor or may infiltrate the gray matter throughout the whole length of the spinal cord; *myxoglioma*, which is only a variety of glioma; *gliosarcoma*, *myxosarcoma*, *sarcoma*, and *carcinoma* are rare in the substance of the cord; *nested sarcoma*, with calcareous deposit at parts of the growth forming *psammoma*, may infiltrate considerable portions of the cord and replace the nervous tissues while retaining the form of the cord; *solitary tubercles*, which may be situated either in the gray or white matter; *syphiloma*, which in the form of gumma is only rarely found in the substance of the cord; and the *cystic dilatation* described as syringomyelitis.

The substance of the spinal cord is rendered more or less flat and thin at the point compressed, so that it may be reduced to the size of a small quill, and in chronic cases the usual ascending and descending changes occur above and below the level of the lesion.

In *hemiparaplegia* the chief lesion and the secondary changes are restricted to one-half of the cord. Interruption, with subsequent descending sclerosis of the fibres of the lateral column, produces a spasmodic paralysis of the muscles, vaso-motor paralysis, and loss of the muscular sensibility and the muscular sense of the parts innervated from below the level of the lesion, while destruction of the posterior horns and the posterior columns, causes loss of the sense of touch and temperature, and of common sensation on the side opposite the lesion (Fig. 173). The anæsthetic belt which surrounds the half of the body on a level with and on the same side as the lesion, is caused by implication of the posterior root-fibres, and the narrow hyperæsthetic zone which is frequently observed on both sides above the level of the anæs-

thetic belt, is supposed by Brown-Séquard to be caused by irritation of descending fibres of those posterior roots which are situated immediately above the level of the lesion. In *universal progressive myelitis* the whole of the transverse area of the cord is diseased in the lumbar and lower dorsal regions, but in the upper dorsal and cervical regions, the sclerosis may be limited to the columns of Goll and the direct cere-

FIG. 173.

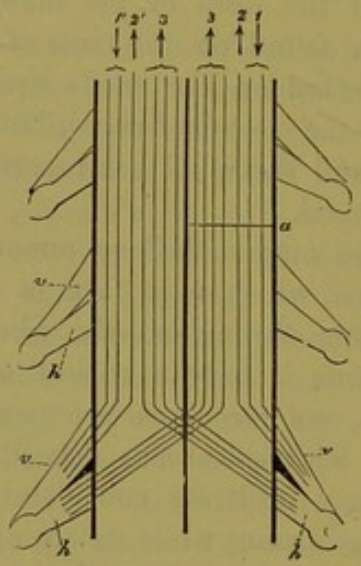


DIAGRAM OF THE COURSE OF THE PRINCIPAL CONDUCTING PATHS WITHIN THE CORD. (After ERB.)

1 and 1′, The motor and vaso-motor tracts, passing through the anterior root (e), and remaining on the same side of the cord; 2 and 2′, Tracts which conduct the muscular sensibility, also passing through the anterior roots, and remaining on the same side of the cord; 3 and 3′, The tracts which conduct sensory impressions of touch, temperature, pain, and tickling. These enter the cord through the posterior roots, and cross to the other side, and pursue their course upwards on that side. Section of the right half of the cord (a) must interrupt conduction through the motor, vaso-motor, and musculo-sensory tracts (1 and 2) on the right side, and the cutaneous sensory tracts on the left side (3′).

bellar tracts, while associated with them may sometimes be found a certain degree of chronic central myelitis.

In *chronic myelo-meningitis* the spinal cord is surrounded by a ring of sclerosed tissue, and consequently this form of myelitis has been named by Vulpian *peripheric* or *cortical myelitis*, or *ring-shaped sclerosis*. Chronic disseminated myelitis will be subsequently described.

Treatment.—In *acute myelitis* active antiphlogistic treatment must be adopted during the febrile stage and Chapman's ice bag should be applied along the spine, while the patient should lie as much as possible on his side or abdomen. It is also desirable to place the patient on a water bed and the parts exposed to pressure should be sponged with brandy or whiskey, and dusted with an absorbent powder in order to prevent, if possible, the formation of bedsores. In the very acute stage

of myelitis a saline mixture may be given in order to maintain the activity of the skin and kidneys, and the bowels should be freely acted on by mineral waters containing sulphate of magnesia, or by senna, rhubarb, or castor oil. Belladonna and ergot are supposed to contract the capillaries of the spinal cord and either one or other may be administered in the early stage of myelitis, and when the fever has subsided iodide of potassium may be given to promote absorption of effused products. When myelitis is developed in the course of syphilis large doses of iodide of potassium should be administered, while mercury should also be given either in combination with the iodide salt, by inunction, or by subcutaneous injection. Counter-irritation and electrical treatment should not be used until the disease has become more or less chronic. Great care should be observed in introducing the catheter for fear of setting up or aggravating cystitis, and every precaution should be taken to prevent the introduction of bacteria into the bladder along with the instrument.

Chronic myelitis must be treated in the earlier stages of the disease on the same general principles as are applicable to the acute disease. The patient should be kept in the recumbent posture, the usual precautions against the appearance of cystitis and bedsores must be taken, and ergot, belladonna, or iodide of potassium should be given internally. Counter-irritation to the vertebral column should only be used with caution, but in transverse myelitis Corrigan's button may be applied at two or three points on each side of the vertebral column opposite the seat of the lesion. The application of a water douche at a temperature of from 98° to 104° F. over the back is sometimes very soothing. When all acute symptoms have subsided and the disease is more or less stationary arsenic or phosphorus with cod-liver oil may be found useful, but strychnine should on no account be administered.

Baths of various kinds are found useful in the treatment of chronic myelitis, and Erb recommended the use of the thermal brine baths with a temperature of from 88° to 78° F., while ordinary brine baths, chalybeate, and mud baths are recommended by others. The temperature in most cases should in my opinion be considerably above that recommended by Erb, but this point must be largely determined by the feelings of the patient.

Hydropathic treatment is also useful, but great care should be taken not to use a too exciting method, and consequently cold douches and slappings should be avoided.

The galvanic current is one of the most useful therapeutic agents for the treatment of chronic myelitis. The poles should be placed over the vertebral column in such positions that the diseased portion of the cord

is included in the circuit, and either a stabile or a slowly labile current may be employed. The currents should not be strong and each application should be of short duration. This treatment should be continued for months, being occasionally interrupted but only to be recommenced after a brief pause.

The patient should avoid all overexertion, or mental excitement, and should lead a very regular life, while the diet should be simple, nutritious, and easy of digestion. Alcoholic drinks, tea, coffee, and tobacco should only be used very sparingly, and sexual intercourse should be confined within the strictest limits or absolutely prohibited. Residence in a mountainous region at a moderate elevation, or at the seaside, will be useful, and it is often advisable for the patient to spend the winter in the South. When completely paraplegic the patient should be allowed to enjoy the fresh air in a wheeled chair, and when bedridden care should be taken that the patient is made to lie alternately on either side or in the prone position in order to avoid as long as possible the formation of bedsores, the appearance of which would be accelerated were the patient permitted to be always in the recumbent posture.

Pain must be relieved by narcotics, such as the subcutaneous injection of morphia. The faradic brush, Preissnitz's compresses, applications of chloroform, and frictions with veratrine ointment and soothing liniments will be found useful in the treatment of many of the sensory disorders which arise in the course of a chronic myelitis, while electrical currents form the best local application for the removal of the paralyses, atrophies, and anæsthesia which persist after the disease has run it course.

CHAPTER XII.

DISEASES OF THE SPINAL MEMBRANES.

I. VASCULAR DISEASES OF THE SPINAL MEMBRANES.

1. Hyperæmia.

Hyperæmia of the spinal membranes has already been considered along with hyperæmia of the spinal cord.

2. Hæmatorrhachis (Meningeal Apoplexy).

Etiology.—Very little is known with regard to the predisposing causes of hæmatorrhachis, but it occurs more frequently in men than in women. The most usual exciting causes are injuries of the spinal column, caries of the vertebræ, excessive bodily exertion, the spasms of convulsive diseases like epilepsy, and the sudden suppression of accustomed discharges. It may also occur in such diseases as scorbutus, purpura hemorrhagica, smallpox, typhoid fever, and other acute infectious diseases. Aneurisms have been known to rupture into the vertebral canal, and blood effused into the brain or cerebral membranes may sometimes pass down into the spinal canal.

Symptoms.—The patient is suddenly attacked with violent pains and falls down, but generally without loss of consciousness. The severest pain is localized at a spot over the spinal column corresponding to the seat of the hemorrhage, but this pain is associated with eccentric sensations such as formication, burning, and tingling, which radiate along the distribution of the nerves the roots of which are first implicated, while an objective examination reveals anæsthesia or hyperæsthesia of these nerve-territories. The early motor symptoms consist of spasmodic jerking and trembling of the extremities, or of a tonic spasm of various groups of muscles which causes the extremities to be forcibly flexed or the head to be retracted or rotated towards one shoulder. The vertebral column is likewise stiff and painful and the patient is unable to raise himself in bed or to assume the sitting posture.

The irritative symptoms give place after a time to those of sensory and motor paralysis, but complete paraplegia is rare, and the anæsthesia

also is seldom complete on objective examination, but the patient complains of subjective sensations of swelling and heaviness of the limbs and trunk, and of feelings of numbness and tingling.

The reflex excitability is said to be depressed in some cases, but probably this only occurs in the region supplied by fibres derived from the nerve roots directly affected; weakness of the bladder and rectum is present only in severe cases, and fever is absent at first, but may occur on the second or third day. The symptoms remain stationary for some time, but when the disease is uncomplicated it often runs a favorable course and a tolerable recovery may take place in a few weeks or months.

Varieties.—(1) If the lumbar region is affected there are pains and stiffness in the loins, tearing pains in the lower extremities, perineum, bladder, and genitals, well-marked paralysis of the lower extremities, loss of the reflexes, and paralysis of the bladder and rectum.

(2) If the *dorsal* region is affected there are pains in the back and abdomen, girdle-pain, stiffness in the dorsal part of the spine, paralysis of the legs and abdominal muscles, and retention of the reflexes in the lower extremities.

(3) If the *cervical* region is affected there are pains in the arms and shoulders, stiffness of the neck, and pain in the occiput. The anæsthesia and paralysis are most marked in the upper extremities, and oculo-pupillary symptoms, difficulty of breathing, and a retarded and feeble pulse may be present. When the upper cervical region is affected sudden death from respiratory paralysis may occur.

II. INFLAMMATION OF THE SPINAL DURA MATER (PACHYMENINGITIS SPINALIS, PERIMENINGITIS).

Pachymeningitis may be divided into inflammation of 1, the external layers of the dura mater and the surrounding cellular tissue, or *external pachymeningitis;* and 2, the internal surface of the membrane with deposition of morbid products between the dura mater and arachnoid, or *internal pachymeningitis.*

1. Pachymeningitis Spinalis Externa (Peripachymeningitis).

Etiology.—Various diseases in and about the vertebral column, such as vertebral caries and bedsores, constitute the most frequent causes of this disease, but it may possibly arise occasionally as an idiopathic affection.

Symptoms.—The symptoms of external spinal pachymeningitis may develop in an acute or chronic form. In the *acute* purulent forms the prominent symptoms are caused by irritation and consist of pain and rigidity of the back, great tenderness on pressure, shooting pains in the lower extremities, and pain in the joints, while there may be considerable elevation of temperature, diarrhœa, and other general symptoms. In the acute form the symptoms of transverse myelitis may be more or less suddenly developed. In the *chronic* fibrinous form of the disease the premonitory symptoms are obscure and the symptoms of slow compression of the cord or of a chronic transverse myelitis with secondary degenerations predominate. These symptoms comprise both sensory and motor paralysis below the level of the lesion, muscular tension, increased cutaneous reflexes and tendon-reactions, and after a time paralysis of the sphincters and bedsores.

2. Pachymeningitis Interna.

Internal pachymeningitis may be divided into *a*, the hypertrophic, and *b*, the hemorrhagic varieties.

a. Pachymeningitis Interna Hypertrophica.

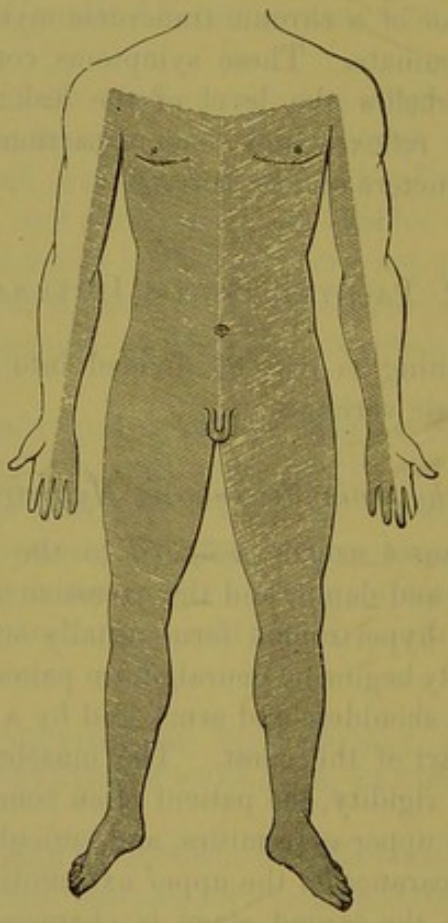
Etiology.—The causes usually assigned to the hypertrophic variety are exposure to cold and damp, and the excessive use of alcohol.

Symptoms.—The hypertrophic form usually occurs in the cervical region and it generally begins by neuralgiform pains in the neck and head which shoot into the shoulders and arms, and by a painful girdle sensation in the upper part of the chest. The muscles of the neck are in a state of spasmodic rigidity, the patient often complains of formication and numbness of the upper extremities, and vesicular herpetic eruptions may make their appearance on the upper extremities.

The transition to the second stage is characterized by the gradual development of paralysis, which is usually of the atrophic variety in the upper extremities, and of the spasmodic in the lower. When the lesion is situated on a level with the eighth cervical and first dorsal nerves, both hands are maintained in the position of exaggerated extension, and the segments of the thumb are also extended, while the phalanges of the fingers are partially flexed on the metacarpal bones and upon one another, the fingers being thus held like claws (Fig. 175). In a case of *pachymeningitis* on the level of the eighth cervical and first and second dorsal nerves which was under my care, the hands were only hyperextended when the patient endeavored to grasp, while the

distribution of the anæsthesia is seen in Fig. 174. Oculo-pupillary phenomena, first of the irritative kind, and as the disease advances of the paralytic kind, are always present when the eighth cervical and first dorsal nerve roots are implicated, and they may also be present when the lesion is situated on a higher level if the cord undergoes considerable compression, or if there be transverse myelitis. When the lesion is situated above the level of the eighth cervical nerve the

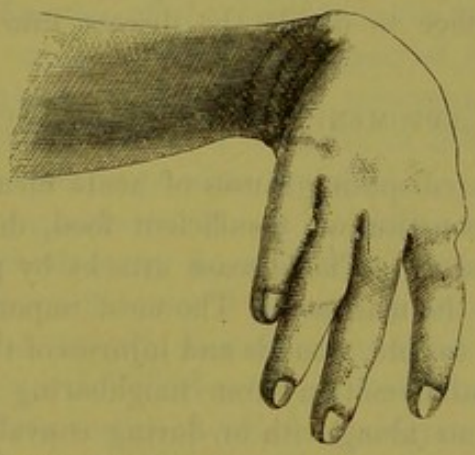
Fig. 174.



distended position of the hand, and the distribution of the anæsthesia differ from those just described. In a case under the care of my colleague, Dr. Leach, in which the lesion was situated on a level with the fifth, sixth, and probably the seventh cervical nerves, the arm was held close to the side, the forearm was extended on the arm and strongly pronated, the hand was strongly flexed on the arm, the fingers were on a line with or only slightly extended on the metacarpal bones, and the phalanges were extended upon one another, while the thumb was flexed into the palm. All the muscles of the forearm and hand were doubt-

less more or less paralyzed, but the muscles supplied by the musculo-spiral nerve were, on the whole, more affected than those supplied by the ulnar and median nerves, contrary to what takes place when the lesion is situated on a level with the junction of the cervical and dorsal regions of the cord. In another case under my care, in which the spinous process of the fourth cervical vertebra was tender to pressure and the skin of that region sensitive to the cathode of the galvanic current, the symptoms were slight spasmodic paralysis of the lower extremities; feebleness with diffused wasting of all the muscles of the

FIG. 175.



ATTITUDE OF THE HAND IN PACHYMENINGITIS CERVICALIS HYPERTROPHICA, WHEN THE LESION IS SITUATED ON A LEVEL WITH THE UPPER HALF OF THE CERVICAL ENLARGEMENT.

upper extremities, most marked in the deltoid, biceps, and supinator longus; arthropathies of the small joints of both hands; dilatation of the pupils, with widening of the palpebral apertures, most pronounced on the left side; and troublesome hiccough, which persisted continuously during waking hours for about ten days after the patient was laid on her back in bed, and which, after abating, could be readily excited again by any movement of the head. The sensory phenomena consisted of stiffness of the neck, and pains on a level with the fourth cervical vertebra, which radiated on both sides of the neck and down the shoulders, but there was no anæsthesia. The patient recovered.

b. Pachymeningitis Interna Hemorrhagica.

The symptoms of internal hemorrhagic pachymeningitis are very obscure, and are usually complicated with those of coexisting cerebral disease. They consist of pains in the loins and back, tearing pains in the extremities, stiffness of the vertebral column, increasing muscular

weakness which may gradually develop into complete paraplegia with contractures, various degrees of cutaneous hyperæsthesia or anæsthesia, and weakness of the bladder. If a patient with these symptoms is at the same time suffering from chronic alcoholism and cerebral paralysis, internal hemorrhagic pachymeningitis may be suspected.

III. INFLAMMATION OF THE SPINAL PIA MATER AND ARACHNOID—LEPTOMENINGITIS SPINALIS; PERIMYELITIS AND ARACHNITIS.

Spinal leptomeningitis presents many varieties, but for practical purposes it will suffice to divide the disease into the acute and the chronic forms.

1. Leptomeningitis Spinalis Acuta.

Etiology.—The predisposing causes of acute meningitis are a scrofulous or tubercular constitution, insufficient food, damp dwellings, and sexual or other excesses. The disease attacks by preference children, young persons, and the male sex. The most important of the exciting causes are exposure to cold, wounds and injuries of the vertebral column, and extension of inflammation from neighboring structures. Spinal meningitis may occur along with or during convalescence from pneumonia, acute articular rheumatism, and febrile and infectious diseases, and it is a very usual complication of tubercular basal meningitis. Epidemic cerebro-spinal meningitis is an infectious disease, and its consideration is beyond the scope of this work.

Symptoms.—Acute spinal meningitis is generally complicated by a simultaneous affection of the cerebral pia mater, and it is not always easy to separate the spinal from the cerebral symptoms. The outbreak of the characteristic symptoms of spinal meningitis may be preceded by premonitory symptoms consisting of general heaviness and depression, slight chilliness, gastric disturbances, transitory pains in the head and back, restlessness, and sleeplessness. The characteristic symptoms of the disease are ushered in by a rigor followed by fever of irregular type, and, if the pia mater of the brain be affected, vomiting and other severe cerebral symptoms are present. The patient now complains of an intense, deep-seated, boring pain in the loins, back, or nape of the neck, corresponding to the seat of the lesion. The pain radiates from the vertebral column round the trunk, and shoots in all directions through the extremities, while it is aggravated by all movements of the spine, and by pressure on the spinous processes. It may abate for a time, but a remission is generally followed by an exacerbation of great severity.

The muscles of the back are in a state of spasmodic rigidity, which may sometimes extend the whole length of the spine so as to resemble a tetanic seizure, but at other times is limited to a part of the vertebral column corresponding to the seat of the lesion. The muscles of the extremities are also tense and contracted, and the limbs are then rigid and immovable or the subjects of painful twitchings.

Cutaneous and muscular hyperæsthesia are often present in the extremities and trunk, the distribution corresponding to the areas supplied by fibres derived from the posterior roots of the part affected. The reflex actions are increased at first, but they are liable to be diminished or lost in the later stages of the lesion.

Disorders of the function of the bladder occur at an early period of the disease, caused, most probably, by spasm of the sphincters, and when the cervical region is affected the respiratory muscles become rigid and painful, giving rise to a difficulty of breathing, which may increase to such an extent as to cause asphyxia.

As the disease progresses cutaneous sensibility becomes diminished and complete anæsthesia may be established, while the extremities manifest various degrees of motor weakness up to complete paralysis. The pupils may be contracted, dilated, or unequal, and if the inflammation extends to the medulla oblongata or to the base of the cranium, the patient suffers from vertigo, headache, paralysis of ocular muscles, and delirium, which soon terminates in unconsciousness and fatal coma with hyperpyrexia. At other times deceitful signs of temporary improvement appear, but paralysis and bedsores supervene, and the patient dies from septic fever. In slight cases recovery may take place, but convalescence is slowly established, and the symptoms of sensory and motor irritation disappear only after a long period. In many cases incurable defects are left behind, consisting of anæsthesia, some degree of ataxia, atrophic paralysis of some groups of muscles, and spasmodic paralysis of other groups.

2. Leptomeningitis Spinalis Chronica.

Etiology.—The disease frequently develops from the acute form and is produced from the same causes.

Symptoms.—The disease begins more insidiously than the acute variety. The patient complains of a gradually increasing pain and stiffness in the back, various paræsthesiæ in the lower extremities, and a feeling of weakness and heaviness of the limbs. Eccentric pains are felt in the areas of distribution of the nerves whose roots are impli-

cated in the lesion, consisting of shooting pains in the lower or upper extremities or a girdle sensation round the body according to the situation of the lesion. Hyperæsthesia of the skin of the lower extremities is frequently present, but anæsthesia is rare, and when present it consists mostly of blunting of the sensibility of the feet and lower part of the legs. The motor symptoms consist of stiffness of the back and neck, and an involuntary drawing up or extension of the extremities, but the phenomena of motor irritation are never very prominent in this disease. After a time the limbs become heavy and feeble, but complete paraplegia is rare. When, however, the lower extremities are more or less completely paralyzed the muscles become tense, and contractures with flexion of the limbs or flexion of one limb and extension of the other are established, but the patellar tendon-reactions are often absent under such circumstances. If there be a considerable effusion of spinal fluid, the paralysis becomes augmented when the patient stands, but at other times it is most marked when the patient is lying on his back, probably because the cord becomes passively congested. In severe cases the paralyzed muscles may undergo atrophy; anæsthesia is developed; the cutaneous reflexes and the tendon-reactions are lost; bedsores and cystitis appear and the patient dies from pyæmia.

Morbid Physiology.—The morbid anatomy of hemorrhage and inflammation of the spinal membranes has already been sufficiently considered. In connecting the morbid process with the symptoms the chief points to notice are that the most prominent phenomena of the various affections are caused by irritation or paralysis of the posterior and anterior roots on a level with the lesion, and irritation or paralysis of the sensory and motor conducting paths below the level of the lesion. In disease of the membranes there are two causes of sensory disorder—disease of the posterior roots and of the conducting paths in the posterior columns and posterior gray horns; and two causes of spasm or paralysis—disease of the anterior roots and of the motor conducting paths in the lateral columns. It is important to remember that disease of the anterior root gives rise to an atrophic, and of the lateral column to a spasmodic paralysis. In cases recovering from an attack of meningitis ataxic symptoms may be present from the persistence of the disease in the posterior columns, and when the posterior columns are affected on a level with the upper part of the lumbar enlargement the patellar tendon-reactions will be absent from interference with its reflex loop, whether paralysis be present or not. When the lesion is on a level with the cilio-spinal region, oculo-pupillary symptoms, and when on a level with the upper part of the lumbar enlargement, disorders of the bladder and rectum will be present.

Treatment.—In *meningeal hemorrhage* the patient should be maintained at rest on his side or face and an ice bag applied to the vertebral column, and ergot may be given internally. Leyden recommends the use of mercurial inunction and repeated small doses of calomel during the period of reaction, but when this stage has passed absorption may be promoted by the administration of iodide of potassium. The vertebral column may be painted with iodine, and the galvanic current will also be found useful in promoting recovery.

In *external pachymeningitis* the treatment must be directed against the primary lesion, and when vertebral caries is present the patient ought to be placed on his back on a water bed until the acute symptoms have subsided, and the subsequent treatment must be conducted with a Sayre's jacket or other form of spinal support. The actual cautery applied in the vicinity of the diseased vertebræ is often a very useful remedy, and iodide of potassium, iodide of iron, or friction with mercurial ointment may each be advantageously employed according to the nature of the case. When the primary disease has been arrested and the symptoms have become chronic, galvanic treatment will be found to promote recovery from the sensory and motor disorders.

In internal pachymeningitis and leptomeningitis the same general principles are applied to the treatment of both diseases. In the early stage of acute meningitis cold must be applied to the spine by means of Chapman's ice bag, while leeches may be applied to the vertebral column and a smart saline purgative administered. When the temperature has fallen blisters or some other form of counter-irritation should be applied along the spine, and when the acute symptoms have subsided a mercurial treatment will prove useful, inunction being probably the most efficient method of administration. The patient should lie on his face or side, and when hyperæsthesia, pain, or sleeplessness are urgent symptoms sedatives must be given, the best form of administration being a subcutaneous injection of morphia. Warm baths afford great relief, and moist packing of the whole body often soothes and induces sleep.

In chronic spinal meningitis repeated blisters may be applied along the vertebral column, while moderate doses of iodide of potassium are given internally. Mercury should not be administered unless the presence of syphilis is suspected, while ergot and belladonna have not been found so useful as in myelitis. Warm baths and douches are very soothing to the patient, and a galvanic current of moderate strength may be employed, either applied to the spine or in the course of the nerves which are the subjects of eccentric pains.

CHAPTER XIII.

CERTAIN FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

I. SPINAL IRRITATION.

Etiology.—The female sex predispose to this disease, although men are occasionally affected. The members of neuropathic families are generally attacked, and most of the cases occur between fifteen and thirty years of age.

The exciting causes are emotional disturbances, sexual excesses, exhausting diseases, and everything which weakens the nervous system.

Symptoms.—The symptoms begin with headache, sleeplessness, increased nervous irritability, ill-defined pains in the back, neuralgiform pains in the face or extremities, and general feebleness, these symptoms gradually increasing in intensity until the disease is fully developed.

The patient now complains of pain in the back, which is aggravated by exertion, and is situated most frequently between the shoulder-blades, or in the back of the neck, and less frequently in the loins. The spinous processes of some of the vertebræ are excessively tender to pressure, and over these processes the surface is found to be very sensitive when a hot sponge or the cathode of a galvanic current is applied. Tenderness of the vertebræ to pressure is, indeed, the most constant and important symptom of spinal irritation, and this sign is rendered all the more valuable from the fact that spinal tenderness is never a prominent symptom of myelitis and other organic diseases of the cord.

The patient complains of various paræsthesiæ and neuralgiform pains in the upper and lower extremities, occiput, face, pelvic region, bladder, genitals, or viscera; the slightest exertion occasions great fatigue and exhaustion; and walking soon becomes impossible owing to the excessive pain caused by it.

The motor symptoms consist of fibrillary twitchings, spasms of some muscles, choreic movements, hiccough, and even permanent contractures in rare cases. Epileptic attacks are said to have been occasionally observed, but it is more likely that these general convulsions were hysterical seizures. A certain degree of muscular weakness may be present in some cases, but real paralysis has never been observed.

The vaso-motor disorders consist of coldness of the hands and feet, and the patients are apt to turn pale or red on the slightest provocation.

The most common visceral disorders met with are eructations, nausea, vomiting, palpitations, asthmatic breathing, cough, vesical spasm, and polyuria. The patient also complains of noises in the ears, dizziness, muscæ volitantes and other disorders of vision, sleeplessness, and great mental depression or irritability. The brothers Griffin divided the disease into several varieties, and endeavored to connect the symptoms present in each case with the localization of the spinal tenderness in the cervical, dorsal, or lumbar regions, but the symptoms first enumerated are frequently met with in cases of hysteria in the entire absence of spinal tenderness. I have never been able to assure myself that the divisions of the brothers Griffin are borne out by clinical facts. Spinal irritation is never a fatal disease, althongh it may last for months or years and cause great suffering to the patient.

II. NEURASTHENIA.

Etiology.—Neurasthenia consists of exhaustion of the cerebro-spinal centres, and generally occurs in neuropathic families, and the male is more liable to the disease than the female sex. Neurasthenia may be a sequel of any exhausting disease, but it is generally caused by overwork and worry, or by sexual and other excesses.

Symptoms.—The symptoms vary according as the weakness declares itself in the spinal or cerebral centres, although in most cases both groups of symptoms are variously combined.

In *spinal neurasthenia* the patient complains of weakness of the lower extremities, which may sometimes be so great as to amount to a slight degree of paralysis. The arms also are readily fatigued, but the weakness in them never reaches the same degree of intensity as in the legs. The sensory disturbances consist of pains in the back and neuralgiform pains in the extremities, which may sometimes be so intense as to simulate the pains of locomotor ataxia, although in the former disease they are never so acute and darting as in the latter. The patient also complains of numbness and formication in the lower extremities, the hands and feet are cold, and the body is apt to be bathed in sweat at night, and during the day on slight exertion. The sexual power is generally diminished, and the act of coition is followed by great prostration, disturbed sleep, and a fidgetty, restless feeling in the lower extremities. The functions of the bladder are usually normal, although a little dribbling of urine may occasionally be present.

In *cerebral neurasthenia* one of the most constant symptoms is a diffused tenderness of the scalp, which is exquisitely sensitive to a slight touch, such as that caused by a comb or by a slight ruffling of the hair over the part, but the pain is relieved by deep and steady pressure. The patient also complains of a sense of pressure over the vertex, which is sometimes compared to the feeling that might be caused by a mailed hand being laid heavily over the head. This pain, which is distinct from the sense of tenderness, is most probably caused by the anæmia which so frequently accompanies the affection. Neurasthenic patients are also liable to suffer from migraine headaches, and facial neuralgia, but the only connection between it and these affections appears to be that all of them are liable to occur in neuropathic families.

The motor disorders of cerebral neurasthenia are jerkings of the limbs, which are particularly troublesome when the patient is about to fall asleep; tremors of the hand; and slight clonic spasms of the facial muscles, which may be limited to the orbicular muscle of the eye, or to the angle of the mouth, or implicate a large number of the muscles, so as to constitute a decided facial tic. The patient complains of heaviness of the limbs and is soon fatigued, and decided paralysis is common in females but not in males. The sensory disorders are very numerous in cerebral neurasthenia. In addition to the sensory disorders already described as belonging to the spinal disease, patients are liable to suffer from numbness and tingling in the extremities from slight pressure on the nerves.

Pressure by the edge of a chair over the sciatic nerve causes numbness of the foot, while tingling of the hand is readily caused if the arm rests for a short time over a chair. Sleep is frequently disturbed by feelings of "pins and needles" in one hand and arm, caused doubtless by a slight pressure exerted on the brachial plexus or some of its branches. Hemianæsthesia is a common symptom of neurasthenia in women, and men sometimes complain of abnormal sensations in one-half the body but in them they usually assume the form of a hyperalgesia rather than loss of feeling. The nerves of special sense are almost always affected. The patient complains of a sour or bitter taste, and may have subjective sensations of the smell of phosphorus or other odors, while his perception of flavors and relishes is much diminished.

The patient is also troubled with various noises in the ears, and on lying down at night he is so much disturbed with rushing sounds and hearing the beating of his heart with unusual distinctness that he is obliged to lie on his back to avoid his ear coming in contact with the pillows, while in the morning there is decided deafness which is accompanied by distressing attacks of yawning. Sight is affected in various

ways; the patient suffers from muscæ volitantes, colored vision, and spectral illusions, the latter being specially liable to appear when the eyes are closed for sleep at night. Vision may be impaired either from defective nutrition of the sensory nervous mechanism, or from feebleness of accommodation and of the internal recti muscles.

The *vaso-motor* disorders of neurasthenia are numerous. One of the most common is blushing, which may spread not only over the face, but also over the neck and other parts of the body, or pallor may take the place of a blush owing to vaso-motor spasm. Neurasthenic patients are very liable to suffer from cold hands and feet, which are sometimes bathed in a clammy sweat and at other times abnormally dry; they also complain of coldness of the knees and other joints, and chills passing along the spine, all of them being symptoms which may be attributed to disorder of the vaso-motor system.

The organic functions are profoundly affected in cerebral neurasthenia. The heart is irritable, its action is often tumultuous, and the patient is painfully sensible of its beatings. The pulse is compressible, irregular, and increased in frequency, and may on slight exertion beat one hundred times or more in the minute. Some of the most distressing symptoms are caused by nervous dyspepsia (apepsia nervosa). The patient may take a fair quantitity of food at a meal although he is never really hungry and scarcely ever feels as if the stomach were quite empty. It seems as if a portion of the food were always retained owing to the inability of the stomach to empty itself from simple inertia, and a certain degree of dilatation of the organ may be discovered on physical examination. The introduction of fresh food into the stomach often brings a sense of relief and comfort, but it is only of short duration, and soon after a meal the patient experiences a painful feeling of distention in the epigastrium, a sensation of fulness in the head, confusion of thought, an indisposition to undertake any work, and a sense of profound and indescribable misery. The bowels are constipated, or attacks of diarrhœa alternate with constipation, and the patient is much distressed with flatulent distention of the abdomen which often becomes blown up suddenly in the most unaccountable manner. The urine is sometimes abundant and of low specific gravity, at other times it is unusually acid and contains an excess of urates, while oxalic acid crystals are often found in it.

The *psychical disorders* are numerous and variable. One of the most constant is sleeplessness. Some patients sleep soon after going to bed, but awake with a start in the early morning, and are unable to fall asleep again. This start is often very painful, the patient sometimes experiencing a feeling as if he had received a blow with a heavy mallet

on the top of the head. In other cases the subject lies awake for hours after going to bed and sleeps best in the morning. In almost all cases the sleep is disturbed by dreams which are always of a disagreeable character, and in the morning the sufferer rises unrefreshed and with a dull aching head.

The patient is liable to suffer from morbid fears, which may assume various forms in different cases. The chronic hypochondriacal patient, who goes from physician to physician and from hospital to hospital with the fixed idea that he is the subject of every imaginable disease, is well known, but many other varieties of morbid fear are met with in neurasthenia. The following morbid fears have received separate names: *Astrophobia*, fear of lightning; *topophobia*, fear of places, including *agoraphobia*, fear of open, and *claustrophobia*, fear of narrow places; *anthrophobia*, fear of men and of society generally, including *gynephobia*, a special fear of women; *monophobia*, fear of being alone; *pathophobia*, fear of disease; *photophobia*, fear of light; and *mysophobia*, fear of contamination. In addition to these many other unreasoning fears might be mentioned, such as a dread of poverty, which, even in cases in which the fear is not altogether groundless, becomes morbidly exaggerated. Another fear which haunts the neurasthenic patient is an intense dread of not falling asleep on going to bed, a dread which in many cases becomes the cause of the sleeplessness which is so much feared. In some cases this fear is so overpowering that the patient sits in an armchair the whole night rather than risk going to bed, while at other times he will not go to bed without having a glass of whiskey or some narcotic. Such patients are, indeed, liable to develop a morbid craving for stimulants or narcotics. The power of self-control is diminished, and if once a neurasthenic patient give way to an illegitimate indulgence or to excess in a legitimate one the vice is apt to acquire an overpowering mastery over him. In neurasthenia, therefore, the patient is apt to give way to alcoholic and sexual excesses, and many are probably tempted to overstep the line which divides vice from crime.

The intellectual condition of those suffering from neurasthenia is very interesting. The first indication of commencing nervous exhaustion may be an unwonted difficulty in recalling the names of individuals. In a further stage of this amnesia the patient forgets the names of familiar objects and he has frequently to indicate his meaning by a paraphrase; for example, he may have to call a "chair," "the thing yon are sitting upon." I have known a person suffering from neurasthenia who had the greatest possible difficulty in completing a simple sentence, owing to his forgetfulness of the names of objects, and to maintain a conversation for a brief period required an intense effort

which was followed by profound exhaustion. In other cases the patient is able to converse freely, but is unable to write a letter without danger of misspelling simple words and committing mistakes in grammar and diction. But a man who is apt to forget the name of his most intimate friend, who is exhausted by having to carry on a brief conversation, who is unable to write a simple business letter, and who cannot concentrate his attention for more than a few minutes at a time upon any ordinary subject, may be quite capable of grasping as well as ever the profoundest speculations of philosophy. Educated men, indeed, have a tendency, when their nervous systems become exhausted, to brood over such questions as the relation between mind and matter, the doctrines of free will and necessity, the existence of evil, the nature of the first cause, and the other great insoluble problems of the universe. And it may be taken as one of the first indications of recovery when the patient is able to set his mind free from these all-absorbing questions and becomes again interested in the practical details of life.

In disposition the patient is shy, and to strangers he is reserved, but he is apt to exhaust the patience of his friends by interminable complaints of his morbid feelings and sufferings. The degree of shyness which the patient manifests may vary from simple coyness to a condition in which he is utterly unable to enter any kind of society, and avoids converse with anyone except a few intimate friends. We have already seen that the patient blushes or becomes pale on the slightest provocation, and when conversing with a stranger he averts his eyes, or he may be unduly watchful and have a slight staring expression as if he were trying to read what is being thought of him. In manner he is fidgetty and frequently changes his position; his hands are in constant movement and he either nervously pulls his beard or seizes and displaces small objects which may be near him; his expression is changeable but always self-conscious; his voice is feeble, in aggravated cases the patient experiences difficulty in articulating polysyllabic words; and even the manner in which he shakes hands is lacking in energy.

The symptoms of neurasthenia usually make their appearance between the sixteenth and fiftieth year of age, and the attack generally supervenes when the mind is subjected to some unusual strain or worry. The symptoms are exceedingly variable and it is impossible to draw the line which separates neurasthenia from hysteria on the one hand and melancholia on the other. The duration of the affection depends greatly upon the circumstances and surroundings of the patient. If the symptoms have been induced by overwork, and it be possible for the patient to suspend work and take a long holiday, the improvement usually appears early and proceeds to complete recovery. But even in such

circumstances it is not always easy to shield the patient from family troubles and anxieties, and it may be still less possible to protect him from the consequences of previous imprudence and immorality. But, although the affection is very variable in its duration, it nearly always terminates in recovery, and neurasthenic patients are often long-lived, being less liable than others to the degenerative changes of the blood-vessels and other tissues which indicate age and shorten life.

III. HEADACHE (CEPHALALGIA).

Headache is so important a sign of nervous disease and arises from so many different causes that it demands separate consideration. The following varieties may be distinguished:

(1) *Anæmic headache* usually affects the temples and brow, or extends along the sagittal suture. It is aggravated by long maintenance of the erect posture, and by all causes which exhaust the nervous system, such as anxiety, night watching, and sexual excess; while it is relieved by rest in the recumbent posture.

(2) *Hyperæmic or congestive headache* usually affects the whole head; the eyes are suffused; the carotids pulsate strongly; and the headache is accompanied by throbbing, agitation, hyperæsthesia with illusions of the special senses, and sometimes by redness and heat of the brow and vertex.

(3) *Hysterical headache* occurs in females and is generally accompanied by other symptoms of hysteria. The pain is sometimes diffused and deep-seated, but it is more frequently localized in one spot, and feels as if a nail were being driven through the skull; hence it is called *clavus*. Hysterical headache is increased in severity during the menstrual period and by mental worry, whilst it is removed by amusement and anything which engages the attention.

(4) *Toxic headaches* are caused by poisons in the circulation. One of the best known examples of this form of headache is that which follows alcoholic intoxication. In the morning after a carouse a severe headache is experienced, which is chiefly localized in the deeper parts of the eyes and at the base of the brain, and is accompanied by a feeling of pressure and weight. Severe headaches also follow the use of narcotics and anæsthetics, and the inhalation of various gases, such as carbonic oxide, sulphuretted hydrogen, the impure atmosphere of crowded rooms, and sewer gas. Obstinate cephalalgia is likewise one of the most common symptoms of uræmia, and in many cases of granular con-

tracted kidney, headache is the chief symptom complained of when the patient seeks medical advice for the first time.

(5) *Pyrexial headache* is probably a variety of the congestive or hyperæmic headache, but it is accompanied by elevation of temperature and the other symptoms which indicate the onset of an acute disease, such as the specific fevers and acute inflammatory diseases like pneumonia. The headache is generally moderate in intensity, dull and deep-seated, and is accompanied by a feeling of lightness in the head, and not infrequently by delirium. This form of headache ceases, according to Jenner, when delirium begins.

(6) *Neurasthenic headache* occurs when the nervous system is exhausted by mental anxiety and worry, night watching or other depressing circumstances. The pain varies considerably in its character, but it is generally deep-seated, heavy, dull, and oppressive. It is often attended by a feeling of pressure and tension above the occiput, and often by great sensitiveness to touch in that region.

(7) *Rheumatic headache* consists of a violent and tearing pain localized in the muscles of the head, or in the fascia of the occipito-frontalis muscle. It is often attended with marked tenderness of the scalp, and is usually brought on by exposure to cold.

(8) *Gouty headache* takes the form of a dull, heavy pain in the forehead, and is generally attended by great depression of spirits, giddiness, pain and fulness in the right hypochondrium, flatulence, and a high-colored urine loaded with lithates. Intense neuralgiform headaches, which are often termed gouty, are probably in most cases an expression of commencing granular changes in the kidneys.

(9) *Sympathetic headache* may supervene on disease of almost all the peripheral organs, although it is commonly associated with diseases of the digestive and sexual organs. The most common form of this variety is the brow-ache of gastric catarrh, but headache may accompany irritation of the intestinal canal or of the uterus and ovaries. This is probably the place in which to mention the headache caused by excessive straining of the eyes. This form of headache is often accompanied by vertigo, insomina, and sickness, and occasionally by vomiting. Headache may be caused by excessive use of the normal eye, but it is much more liable to be induced when the eye is deficient as an optical instrument, and is especially liable to occur in disorders of accommodation. In cases of headache of obscure origin, and more especially when it is found to supervene or to become intensified when the patient begins to read or write, the state of vision should be carefully investigated by an ophthalmologist.

(10) *Syphilitic headache* is characterized by a deep-seated and in-

tense pain, which is either attended by a feeling of weight on the vertex or a sense of constriction as if the head were held fast in a vice. The patient compares it to that which might be caused by successive blows on the head with a heavy mallet. The pain is at times distinctly circumscribed, at other times it is diffused, and may then occupy either the frontal, temporal, or occipital regions. In some cases it is diffused and invades the whole head. The headache never completely intermits, but it is liable to paroxysmal exacerbations of great severity, which are particularly intense during the night. There is, indeed, no other form of headache in which a nocturnal exacerbation is so characteristic as the syphilitic variety, and it is always accompanied by a degree of sleeplessness which is often out of proportion even to the intensity of the headache.

(11) *Organic headache* occurs as one of the symptoms of structural disease within the cranium, and is especially characteristic of intracranial growths. The pain is persistent and continuous, but liable to paroxysmal exacerbations of great severity. It may be frontal or occipital, and it is generally deeply seated in one spot, although it may extend over the whole head. Tenderness of the scalp often attends the headache, and pain may be elicited on percussing the skull over the seat of the tumor. An ophthalmoscopic examination often reveals the presence of optic neuritis.

(12) Hemicrania.

Etiology.—The predisposing causes of hemicrania are inheritance, generally from mother to daughter; a neuropathic disposition; the female sex, and the ages of fifteen to thirty years, while the exciting causes are digestive disorders, constipation, exhaustion from want of food, fatigue, sleeplessness, and excessive mental exertion.

Symptoms.—Hemicrania consists of paroxysms of headache which are separated by free intervals of shorter or longer duration. The attack is frequently preceded by premonitory symptoms, the patient has a sense of pressure in the head, and feels chilly, nauseated, depressed, weary, and indisposed to work. Other interesting symptoms often precede the attack. The most usual of these are transitory impairment of cutaneous sensibility, which is accompanied by tingling, numbness, and formication. At other times there may be, separately or in various combinations, deafness, tinnitus aurium, a bitter taste, loss of taste, embarrassment of speech, momentary incoherence, drowsi-

ness, transitory paresis of one of the limbs, vertigo, indistinct vision, muscæ volitantes, scintillating scotoma, or hemiopia.

The characteristic pain comes on by degrees in the course of the day, but almost never with the lightning rapidity of the pain of neuralgia. The patient often feels great drowsiness and weariness in the evening, and awakens with headache in the morning. It often begins with a dull pain over the forehead, and as it increases in severity it passes down to one eye and remains fixed over the temple. Occasionally it is localized at the top or back of the head. The left is more frequently attacked than the right side, but occasionally successive attacks occur on alternate sides, these cases being named *hemicrania alternans* by Eulenburg. Patients describe the pain as being dull, burning, or bursting, and it is frequently associated with an intense feeling of sickness. Every beat of the heart is felt as a throb of pain, and the slightest movement, even raising the head from the pillow, or the exertion of talking, augments the throbbing pain to an almost unendurable pitch by exciting the circulation. The patient suffers from continuous nausea, the appetite is lost, there may be more or less jaundice, and the attack often terminates by free vomiting, first of the usual contents of the stomach and afterwards of bile. The skin over the forehead, temples, and parietal region is often sensitive to a light touch during an attack of migraine, but deep diffused pressure affords relief. Berger has shown that the sense of touch may become morbidly acute (*hyperpselaphesia*), the circle of perception being sometimes only one line in diameter on the affected, and four lines on the sound side of the forehead. He also found that variations of temperature of 0.4° C. were perceived on the affected, and only of 0.8° C. on the sound side. The electro-cutaneous sensibility was likewise increased on the affected side. Deep pressure over the superior or middle cervical ganglion of the sympathetic, and over the spinous process of the cilio-spinal region of the cord is painful, and a small spot is sometimes found over the parietal protuberance which is painful on pressure, but the *painful points* of genuine neuralgia are never present.

The *vaso-motor phenomena* of migraine are exceedingly important, and the disease has been divided into (1) *hemicrania spastica* or *sympathico-tonica*, (2) *hemicrania angioparalytica* or *neuroparalytica*, and (3) *hemicrania mixta*, according as the phenomena of vascular contraction, dilatation, or first contraction and then dilatation, are present respectively.

1. *Hemicrania Spastica or Sympathico-tonica.*—On the affected side, at the height of the attack, the eye is prominent; the pupil is dilated; the temporal artery feels like a hard cord; the side of the face and the ear are pale; and the temperature in the external meatus is lower

by 0.4° C. or 0.6° C. than that of the meatus of the opposite side. The pain is aggravated by every circumstance which excites the circulation and increases the arterial tension, and compression of the carotid on the affected side augments, and of that on the unaffected side diminishes the pain. The salivary secretion is much increased in quantity and becomes very viscid. Towards the end of the attack the affected side of the face and ear become red and hot, the conjunctiva is injected, there is a copious flow of tears, the pupil becomes contracted, a diffused feeling of warmth spreads over the body, and the paroxysm terminates by palpitations, vomiting, a free discharge of limpid urine, and sometimes a watery evacuation from the bowels.

2. *Hemicrania Angioparalytica or Neuroparalytica.*—On the affected side, at the height of the attack, the eye is retracted; the palpebral fissure is narrowed; the pupil is contracted; the conjunctiva is injected; there is an increased flow of tears; the side of the face and ear are red, hot, and turgid; the temporal artery is large, tortuous, and beats with unusual force; and the temperature in the external meatus is higher by from 0.2° C. to 0.4° C. than on the opposite side; and compression of the carotid on the affected side and of that on the opposite side aggravates the pain. The radial artery is sometimes small and contracted, and the pulse is slow, beating only from forty-eight to fifty-six times a minute. Towards the end of the attack the face becomes paler and the other phenomena pass off. Vomiting is not so urgent a symptom in the paralytic as in the spastic variety, and the course of the former is, on the whole, milder than that of the latter.

3. *Hemicrania Mixta.*—In this form the phenomena of the spastic variety are present at first, but these are soon followed by those of the paralytic variety.

An attack of hemicrania usually lasts from a few hours to half a day, but may, at times, continue a whole day or even several days. If the pain be present on waking it often wears off gradually towards evening, and the patent falls into a sleep from which he usually awakes free from pain. But if it should come on during the day, it gradually increases in severity, and the patient is unable to sleep. The headache is generally most tolerable when the patient is laid down in a darkened room, but at other times it is aggravated by the recumbent position, and the patient can best endure his sufferings in the sitting or upright posture. The paroxysms often recur with great regularity at intervals of three or four weeks, and females are frequently attacked during the catamenial period, although this rule is not without numerous exceptions. Those who are liable to migraine often enjoy excellent health during the intervals between the attacks. Migraine headaches fre-

quently cease to recur after the climacteric period in women, and after the fiftieth year of age in men, and in persons who do not inherit a strong tendency to the disease they may cease at an early age, either spontaneously or under the influence of remedies.

Morbid Physiology.—In *spinal irritation* there is an excessive irritability of the spinal cord, but the brain also must participate in this disorder, inasmuch as the spinal symptoms are usually associated with an emotional condition, such as is met with in hysteria. It must be remembered that the increased irritability of the nervous tissues in spinal irritation is a paralytic irritability and therefore the affection is allied to exhaustion of the nervous system or to neurasthenia.

Neurasthenia is, as its name implies, caused by exhaustion of the nervous system, those portions of it which are the last to be developed being more profoundly affected than the earlier evolved parts. Exhaustion of the latest evolved tissues implies diminution or temporary abolition of the latest evolved functions, and consequently the control of the conscience is diminished, and the animal instincts and propensities become more urgent and active. But although the stock of energy of the nervous system is diminished the irritability is increased, just as occurs in the nervous tissues of animals after prolonged starvation. The consequence is that stimuli which produce little or no effect in health have now widespread results. This excessive irritability of the nervous tissues explains the great mental excitability of neurasthenic patients, as well as their sensitiveness to changes of weather and other external stimuli.

Hemicrania is the only form of headache, the morbid physiology of which we shall discuss in this place. The chief symptoms which require explanation are (1) the vaso-motor and oculo-pupillary symptoms, (2) the disorders of the general circulation, and (3) the pain.

(1) The *vaso-motor* and *oculo-pupillary* disorders which are observed in hemicrania spastica are explained by supposing that the cilio-spinal region of the spinal cord and the cervical sympathetic are in a state of irritation, those of hemicrania paralytica by supposing that the same nervous mechanism is paralyzed, and those of mixed hemicrania by supposing that this mechanism is first irritated and then paralyzed.

(2) The retardation of the pulse which is often observed is supposed by Landois to be due to direct irritation of the vaso-motor centre in the medulla. Other symptoms which show that this centre is in a state of irritation are coldness of the hands, chilliness, suppression of perspiration, contracted state of the radial artery, and increased arterial tension. Towards the end of the attack symptoms of vascular dilatation, such as increased secretion of saliva and urine, and watery stools

indicate that the irritation of the vaso-motor centre has been followed by exhaustion and temporary paralysis.

(3) The pain in migraine has been variously explained. Romberg thought it due to a hyperæsthesia of the brain, and he called it "neuralgia cerebralis" in order to distinguish it from peripheral neuralgia. Others suppose that the pain is caused by a neuralgia of the intracranial and meningeal branches of the trigeminus and the other nerves which accompany the bloodvessels, and not of the cerebrum itself as Romberg supposed. But when once the vaso-motor and oculo-pupillary symptoms of hemicrania attracted attention the supposition that the pain was secondary to vascular disturbances in the brain lay near at hand. Du Bois-Reymond suggested that the pain was caused by a tonic spasm of the muscular coat of the vessels and was thus similar to the pain felt in cramp of the muscles, or to that felt in the uterus during labor. Others have suggested that the pain is caused by the anæmia which results from spasm or the hyperæmia which results from dilatation of the vessels of the brain; this view being supported by the well-known fact that anæmia or hyperæmia of the peripheral nerves is one of the most frequent causes of various neuralgias. But some pathologists believe that hemicrania is allied to genuine epilepsy. The facts in favor of this opinion are the paroxysmal character of the disease, its occurrence in members of neuropathic families, and the attack being often preceded by symptoms, like scintillating scotoma, resembling an epileptic aura and occasionally accompanied by embarrassment of speech or a slight degree of aphasia. Observing the resemblances between certain forms of headache and epilepsy Dr. Sieveking proposed to name the periodical headaches of nervous people *cephalalgia epileptica*, while Dr. Liveing for the same reason came to the conclusion that hemicrania was caused by "nerve storms." But Dr. Hughlings-Jackson has given a more scientific expression to this theory by attributing the headache of migraine, especially the form which is associated with ocular phenomena, to a discharging lesion from the cortex of the posterior lobes or the sensory area of the brain, or in that part of the sensory area which is the anatomical correlative of the sensation of pain in the head. But during the attack the nervous discharge does not remain limited to the sensory area, since some is directed outwards to the medulla oblongata and cilio-spinal region of the spinal cord, causing irritation or inhibition of some of these centres, and giving rise to the vaso-motor and oculo-pupillary phenomena of the disease.

Treatment.—The treatment of spinal irritation must first be directed to remove the cause, while an endeavor is made to improve the general nutrition by a full diet including the moderate use of wine, and much

exposure to the open air and to sunlight. The patient should rest frequently in the recumbent posture, and all causes of fatigue should be avoided. Change of air to a dry mountainous district or to the seaside, or a moderate hydropathic treatment may be found useful.

The internal remedies which are found most generally useful are quinine, iron, zinc, and strychnine. The galvanic current should be passed through the vertebral column, and the painful portions should be included between the poles. Each sitting should be short and the strength of the current moderate. The negative pole placed directly over the painful vertebra appears to be a good method of applying galvanism, and general faradization or central galvanization is said to be useful. Counter-irritation applied directly over the painful portion of the spine is a most effectual method of treatment and Corrigan's button gives very favorable results.

Neurasthenia must be treated on the same general principles as spinal irritation. The means to be adopted are complete mental rest, generous diet, and much exposure to fresh air and sunlight, but with only a moderate amount of active exercise. It is almost incredible what a small amount of exercise suffices to induce great fatigue followed by profound mental depression in such patients, even when the subject of the disease appears to be possessed of great muscular strength. In aggravated cases the treatment introduced by S. Weir Mitchell of rest, seclusion, over-feeding, electricity, and massage, should be adopted.

The treatment of *headache* may be directed against the cause, or against the symptom of pain. In order to fulfil the first indication the remedies for anæmia, hyperæmia, hysteria, syphilis, nervous exhaustion, Bright's disease, or for allaying local irritations must be employed according to the nature of case.

It is scarcely possible to adopt a casual treatment against migraine, inasmuch as so little is known of the circumstances which concur to induce an attack. An emetic may sometimes arrest an impending attack, but it appears to act less by removing indigestible substances than by the impression it produces upon the nervous system.

The direct treatment of migraine may be divided into that which is appropriate during the intervals and for removing or palliating the paroxysm. In the intervals between the attacks the preparations of iron, especially the carbonate, have been found useful in feeble and anæmic subjects. Arsenic is another very useful drug during the intervals, and other periodic remedies, such as quinine, quinoidin, and salicin, have been tried, but they appear to act better in arresting an impending attack than during the interval. Strychnine, nitrate of silver, sulphate of nickel, bromide of potassium, chloride of ammonium, oil of

turpentine, and lupulin have been tried during the interval, but with doubtful success. Chalybeate springs, mud and sea baths, hydropathy, and residence in lofty mountainous regions, have all been found beneficial, but they appear to influence the disease favorably by improving the general health. Dr. Ringer believes that cannabis indica in doses of from one-quarter to one-half a grain, or ten minims of the tincture three times a day, is one of the best remedies we possess for preventing the attack. In the treatment of the attack every source of external irritation should be removed, the room should be moderately darkened, and all noises should be prevented. In spastic hemicrania the patient should lie flat on the back with the head a little raised, but in the paralytic variety the patient usually prefers to maintain a sitting posture, and to rest the head against some hard substance. Firm compression of the head by a handkerchief bound round it is an old remedy and appears to give temporary relief. The application of cold to the head by means of an evaporating lotion or ice bag is often grateful, while a warm fomentation to the forehead and temple is sometimes more soothing than the cold. Quinine given in doses of from five to fifteen grains two or three times in succession often arrests an attack, and half drachm doses of the liquid extract of ergot are often useful in the angioparalytic variety.

Coffee is another remedy which appears to act by stimulating the vaso-motor nerves, and may be given in the form of a strong infusion, but its alkaloid, caffeine, is more effectual. The citrate of caffeine may be ordered in doses of one grain every four hours for some time before the expected attack. Guarma powder in half drachm doses every four hours till relief is obtained is sometimes very effectual. The inhalation of the nitrite of amyl has been found useful in the spastic form of hemicrania, but it acts only as a palliative, its effect being transitory.

Croton chloral hydrate is a very useful remedy for arresting the attack. It may be administered in five grain doses every four hours or in one dose of from fifteen to twenty grains. The late Dr. Austin thought the best means of arresting a sick headache was to give twenty grains of chloral and make the patient plunge his feet into very hot water and mustard while breathing the steam. A full dose of bromide of potassium either alone or in combination with chloral or with tincture of opium, or better still nepenthe, is also very efficacious. Chloride of ammonium in half drachm doses is another useful remedy. Morphia and other narcotics may be employed in aggravated cases, but they are not so useful in migraine as in neuralgia.

The galvanic current when properly applied is one of the most powerful remedies we possess for hemicrania. Holst recommends that one

electrode be placed at the inner edge of the sterno-cleido-mastoid muscle, and the other on the palm of the hand. The pole on the neck is made positive in hemicrania spastica, and from ten to fifteen elements are used for two or three minutes, while the negative pole is used at the neck in hemicrania angioparalytica and powerful excitations are produced by repeated closures and openings, or by reversals. Other authors recommend that a weak current should be passed continuously through the head. The induced current has been recommended by Frommhold; he applies one of the poles high up the back of the neck in the median line, and the other upon the forehead or over the superciliary arch. Fieber employs the "electric hand," and the application is certainly grateful to the patient, but I have not found that it produces a marked effect upon the duration of the attack.

In dealing with the various forms of headache, other than hemicrania, the treatment must be directed against the cause of the pain. In the anæmic form, iron and sustaining diet; in the hyperæmic, saline purgatives and low diet; in the febrile, ice to the head; in the hysterical, iron and moral management of the patient, are the means of treatment which must be chiefly relied upon. In symptomatic headache the stomach or other organ which is the source of irritation must be subjected to treatment; in gouty headache with hepatic derangement saline purgatives bring relief; and in the neuralgiform variety saline diuretics and agents which lower the arterial tension are indicated. In rheumatic headache salicylate of soda may be given, but iodide of potassium is, as a rule, the most effectual remedy for such cases. In syphilitic headache an energetic antisyphilitic treatment must be adopted.

CHAPTER XIV.

SHOCK, CONCUSSION, AND ALLIED CONDITIONS.

I. SHOCK.

Etiology.—Shock is more easily produced in persons inheriting an irritable and unstable nervous system than in robust persons, in women than in men, and in young than in old people. The exciting causes of shock are sudden and severe or extensive injuries of any part of the body, and strong emotional excitement, especially the depressing passions.

Symptoms.—Cases of shock may be divided clinically into two, (1) *torpid* shock or those in which the symptoms of depression predominate, and (2) *erethismic* shock or those in which the symptoms of prostration are associated with those of excitement.

(1) *Torpid Shock.*—In the torpid form of shock the patient lies utterly prostrate, the surface of the body is pale, cold, and covered by a clammy sweat, which collects in drops on the forehead and eyebrows; the lips are bloodless, the nostrils dilated, and the countenance of a dull aspect and shrunken; while the eyes have lost their lustre, are sunk in their sockets, and partially concealed by the drooping lids. There is complete muscular relaxation, which may even extend to the sphincters. If the patient be conscious he may complain of feeling cold and faint, and the whole body may tremble. The pulse is frequent, unequal, and feeble or imperceptible at the wrist, but the fluttering action of the heart may be heard on auscultation. The respiratory movements are irregular and gasping, or short and feeble, the respirations being sometimes so feeble that they are scarcely visible, although a slight movement of the diaphragm may generally be discovered by careful observation. The temperature of the body is depressed. The patient suffers from vertigo and dimness of vision, while in the less severe cases there are nausea, vomiting, and hiccough. The psychical symptoms consist of mental depression, restlessness, confusion of thought, incoherence, or drowsiness, but the patient generally gives rational replies to definite questions. At other times the patient appears singularly calm and rational, while the various senses remain unaffected, hearing being sometimes unusually acute.

(2) *Erethismic Shock.*—This form of shock is rare, the majority of cases in which symptoms of prostration are mixed with those of excitement being preceded by a distinct, though it may be transient stage of collapse. The skin is at first hot and dry; the pulse is quick and bounding, but always compressible; the respirations are hurried, imperfect, and interrupted by sighs; the tongue is tremulous; the patient complains of thirst; rigors are occasionally present; and vomiting is a frequent and sometimes obstinate symptom. The mental and bodily prostration of collapse is succeeded by tremor and twitchings of the muscles, and the patient suffers from restlessness, jactitation, præcordial anxiety, and delirium. At times the patient merely presents a peculiar irritability of manner with an increased disposition to talk, sometimes rationally, occasionally incoherently. At other times the patient has strange illusions attended by a peculiar dread of impending evil.

In some cases, however, there is fierce maniacal raving, which is most pronounced during the night, or the delirium may assume all the characteristics of that observed in *delirium tremens*. The patient obtains no sleep, or it is partial, interrupted, and unrefreshing. As the exhaustion increases the skin is covered by a cold, clammy, and often profuse sweat; the face becomes pale; the expression haggard; and the pulse is frequent, irregular, fluttering, and uncontrollable. Towards the end subsultus and slight convulsions supervene and the patient dies comatose. In the severest form of shock the functions of the nervous system are suddenly abolished and the heart ceases to beat; in the mild forms of the affection the symptoms are more or less similar to those of an ordinary fainting fit, while between these two kinds innumerable transitional forms are observed. The medium degrees of shock are named *collapse*. In the intermediate forms of shock the symptoms of collapse give place to those of excitement, this stage being called the *period of reaction*. The period of reaction is characterized by improved pulse and respiration, restoration of muscular power, and increase of temperature. These symptoms often merge quickly into health, but in some cases relapses occur, and convalescence is then protracted. In other cases the torpid may be replaced by the erethismic form of the disease. Recovery after shock is often partial only, the irritability of the nervous system remains permanently increased, so that slight exciting caases suffice to give rise again to the phenomena of shock.

II. CONCUSSION OF THE SPINAL CORD (COMMOTIO SPINALIS).

Etiology.—The most usual causes of spinal concussion are falls upon the feet or buttocks, blows over the back, and railway collisions, those who sit with their back in the direction from which the shock comes being most liable to suffer. Lightning passing through the body causes a general shock in which the spinal cord participates, and violent mental excitement appears to be able to produce the symptoms of spinal concussion occasionally.

Symptoms.—The most usual symptoms of spinal concussion are feebleness or, on rare occasions, paralysis of the extremities; paræsthesiæ of various kinds; pain in the neck, loins, or along the spinal column; tenderness on pressure of some of the spinous processes; some degree of cutaneous hyperæsthesia or, more often, anæsthesia, weakness of the bladder, and considerable emotional disturbance.

Varieties of Spinal Concussion.

(1) *Severe Forms of Shock causing Rapid Death.*—The patient, after the injury, is found more or less paralyzed in all his extremities; the surface of the body is distinctly anæsthetic; the pulse is small, weak, and slow; the skin is cold and pale or slightly cyanotic; the respiration is disordered and sometimes the difficulty of breathing may amount to dyspnœa; the mind is confused or there may be complete loss of consciousness; the stools and urine are passed involuntarily; and the patient dies in a few hours or days from general prostration and paralysis of respiration.

(2) *Slight Shock causing Severe Symptoms at first, but soon ending in Recovery.*—Immediately after the accident the patient is found more or less paralyzed and anæsthetic in the lower, and occasionally in the upper extremities, but the bladder is not usually affected, while improvement begins in a few days, and recovery is complete in a few weeks or months.

(3) *Severe Symptoms at first, followed by a Protracted Illness from which the Patient usually Recovers.*—Soon after the accident the patient complains of a motor weakness, which gradually increases until the extremities are paralyzed. He also complains of pains in the back of the neck, loins, or along the vertebral column, which is often excessively sensitive and tender to pressure, and various paræsthesiæ, but anæsthesia is not well marked. Vomiting and loss of consciousness may be present at first, the extremities are cold and livid, and occasion-

ally there is retention of urine, while patients often manifest a high degree of mental irritability for a long time. Gradual improvement now occurs, but the patient complains of great weakness, and even slight atrophy of some of the muscles may occur, and complete recovery from the paralysis may not take place for several years, while patients may remain irritable and sensitive long after all the paralytic symptoms have disappeared.

(4) *Very Slight Symptoms at first, but subsequently a Severe Progressive Spinal Disease develops, and the result is doubtful.*—This form of concussion is usually caused by railway collisions, and immediately after the injury the symptoms are insignificant. At first, indeed, the patient is unusually calm and self-possessed, and he may busy himself for some hours in assisting his less-fortunate fellow sufferers, but on reaching home he becomes talkative and excited, and is now apt to burst into tears. On the following day he complains of feeling shaken or bruised all over, but it is not until the lapse of from a few days to a week or more that he finds himself unfit for exertion and unable to attend to business. When the patient is examined some weeks after the injury, the back is found painful on movement; some of the spinous processes are found tender on presure; the patient holds himself stiff and erect in walking, as if the various vertebræ were soldered into one piece, and he is unable to bend forwards to pick anything off the ground, to move the spine backwards or laterally, or to raise himself from the horizontal position without the aid of his hands. Girdle sensations, paræsthesiæ of all sorts, and anæsthesia or hyperæsthesia in varying degrees and in different situations may be present, while weakness of the bladder and diminution of the sexual power are frequently observed. After a time marked atrophy may take place in individual muscles and groups of muscles, and at times it may be extensively distributed. The extremities are cold and bluish, the general nutrition is impaired, and the expression of the countenance changes to one indicating great anxiety and nervousness. Patients often suffer from muscæ volitantes and spectral images, intolerance of light, or diplopia and strabismus, while they are often annoyed by deafness, noises in the ears, or great intolerance of sound. The patient is irritable and timid, suffers from a feeling of constriction in the head, sleeplessness, confusion of thoughts, weakness of memory and intelligence, impaired power of work, and his friends observe that his whole character has undergone a serious change. In cases of this kind the patient may die from bedsores and pyæmia, become permanently paralyzed in the lower extremities, or gradually recover.

III. CONCUSSION OF THE BRAIN.

Etiology.—The exciting causes of cerebral concussion are severe injuries, such as falls from a height or blows on the head, which cause the whole mass of the brain to be jolted or shaken. Concussion may be complicated by fracture of the skull, and in such cases the effects of the concussion are often less severe than in uncomplicated cases, apparently because a certain amount of the applied force is expended in producing the fracture.

Symptoms.—Concussion is a special form of shock and its symptoms may be described under four stages: (1) the stage of *collapse;* (2) the stage of *rallying or vomiting;* (3) the stage of *reaction;* and (4) the stage of *gradual convalescence.*

(1) *The Stage of Collapse.* The symptoms of this stage are somewhat variable. In the slighter forms the patient suffers from transient confusion of ideas, and slight giddiness. He may feel weak and faint and be unable to maintain the erect posture. In the more severe forms the symptoms are those of collapse, with loss of consciousness; but paralysis, such as occurs in compression of the brain, is never present. The patient is semi-conscious or insensible, most reflex actions are abolished, the skin is cold and pallid, the respirations superficial and shallow, the pulse feeble or imperceptible at the wrist, whilst the pupils may either be contracted, dilated, or unequal.

(2) *The Stage of Rallying or of Vomiting.* After a period varying from a few minutes even up to days, according to the severity of the attack, the patient usually begins to show signs of rallying. This stage is often ushered in by vomiting, or very occasionally by an epileptiform attack; the pulse improves in strength, the respirations become less shallow and more perceptible, the body becomes warmer, reflex actions can be excited, and the patient gives evidence of returning sensibility, while he may exhibit signs of mental distress.

(3) *The Stage of Reaction.* The symptoms of the stage of rallying are succeeded by those of reaction. In this stage the phenomena of febrile reaction manifest themselves by the usual symptoms—hot and dry skin, quick and hard pulse, and scanty urine; while the patient is drowsy, yet quite conscious when roused by a question addressed to him.

In some cases these symptoms gradually develop into those of compression and the patient dies comatose, while in other cases the symptoms of reaction give place to those of inflammation of the brain. This stage may continue from three to twelve days in cases which recover.

(4) *The Stage of Convalescence.* Reaction is followed by a pro-

gressive subsidence of the symptoms, and either by a gradual restoration of the patient to health, or the establishment of one or other of several chronic affections of the nervous system.

Cerebral Irritation.—In another form of nervous disturbance following injuries of the head, and described by Erichsen under the name of cerebral irritation, the phenomena of cerebral excitement are associated with those of loss of function. The patient assumes a peculiar attitude: he lies with the body bent forwards, the knees drawn up on the abdomen, the legs bent on the thighs, the forearms flexed on the arms, and the hands drawn. The patient is restless, and frequently changes his position, but never stretches himself out or assumes the supine posture. The eyelids are firmly closed; the pupils are contracted; the surface of the body is pale and cold; and the pulse is small, feeble, and slow, being seldom more than seventy beats per minute. The sphincters remain, as a rule, unaffected.

The patient is indifferent to everything around him, and is only partially conscious. He may, however, be roused when addressed in a loud voice, and then looks up, mutters indistinctly, or frowns and turns hastily away. His sleep is not stertorous.

After a period of from one to three weeks the pulse improves, the body becomes warmer, the flexed attitude is abandoned, and the mental irritability gives place to mental feebleness and torpidity.

The course of concussion is variable. Some patients die during the stage of collapse, and others from compression or encephalitis following an excessive reaction. Even after apparent recovery the patient often suffers from persistent headache; his mental powers are impaired; his speech may be indistinct and stuttering, his special senses may be permanently impaired; and he remains excitable and is apt to give way to uncontrollable bursts of passion.

IV. CONTUSION OF THE BRAIN.

Whenever the skull undergoes a change of form as the result of injury the substance of the brain may be contused or lacerated. The contusion may be situated immediately beneath the portion of the skull where the injury was inflicted, or on the opposite side of the brain as the result of *contrecoup*, or both these places may be simultaneously affected.

The symptoms of contusion are always complicated by those of concussion and of compression. The diagnosis of contusion must be made by the presence, in addition to the symptoms caused by a general injury to the brain, of phenomena like monospasms or monoplegia which indicate a local lesion in the absence of any signs of fracture of the skull.

V. COMPRESSION OF THE BRAIN.

Compression of the brain may occur after injuries from the pressure of a fractured portion of the bones of the skull, the presence of extravasated blood, pus formed within the skull, or of a foreign body lodged there.

The patient becomes unconscious, the breathing is slow, deep, and stertorous, while the cheeks are puffed out during respiration. The surface of the body is cool at first, but soon becomes hot and bathed in perspiration. The pupils are dilated or unequal, the pulse is slow and full, the feces pass involuntarily, and there is retention of urine. This condition of stupor sometimes alternates with paroxysms of delirium, while local spasms or paralyses are sometimes observed, but it is probable that in these cases the motor area of the cortex is not often lacerated or contused.

For further information with regard to contusion and compression of the brain the reader is referred to surgical works.

Morbid Anatomy.—In *shock* all the cavities of the heart are usually distended with blood and the veins of the body, especially the abdominal veins, are engorged. No changes have been discovered in the nervous system. In cases of concussion which have terminated fatally at an early period, small extravasations of blood have been found, in the spinal variety, in the cord and its membranes; and in the cerebral variety, in the brain and its membranes. In rapidly fatal cases of spinal concussion, fractures of the vertebræ, laceration of the membranes, or meningeal or spinal hemorrhage have been observed, while in the cerebral variety the morbid changes discovered consist of superficial lacerations, minute hemorrhagic extravasations either studded on the surface of the brain or in its substance, and occasionally of a diffused ecchymosis of the pia mater. In cases of spinal concussion which give rise to a protracted illness or to progressive spinal disease the symptoms are caused by secondary meningo-myelitis. It is probable that death is caused suddenly in some cases of cerebral concussion from injury of the roots of the pneumogastric nerves.

In contusion of the brain capillary hemorrhagic extravasations have been found either limited to the cortex or diffused through the substance of the brain. When the injury is localized the extravasations may be so closely aggregated that the part affected presents the appearance of a hemorrhagic infarction. The extravasations are more diffused when the substance of the brain is contused. Compression of the brain is

caused by depressed fractures, extravasated blood, the formation of pus, or the lodgement of a foreign body within the cavity of the skull.

Morbid Physiology.—The most striking phenomena of shock are those which cluster around the organs of circulation. The experiments of Goltz and Brunton show that shock results probably from cardiac paralysis combined with vaso-motor paralysis of the large vascular trunks of the abdomen. Brunton states that moderate blows on the abdomen of frogs produce in some stoppage of the heart without dilatation of the abdominal vessels, and in others vascular dilatation without arrest of the cardiac pulsations, while severe blows produce both effects simultaneously. The vessels of the abdomen are so large that when fully relaxed they are capable of containing almost all the blood in the body, and consequently the condition resulting from this rapid dilatation is equivalent to a sudden hemorrhage. This double condition of cardiac failure and vascular dilatation produces anæmia of the nerve centres, and this accounts for the pallor and coldness of the surface of the body, and the weak, compressible, and fluttering pulse. It must not, however, be forgotten that the injury which has disordered the functions of the cardiac and vaso-motor centres in the medulla oblongata must also have produced a directly deleterious effect upon the nerve centres. The disorders of respiration, the cries of pain, and the various bodily contortions which are caused by bodily injuries or severe mental excitement show that excessive stimuli occasion powerful outgoing discharges from the higher nerve centres. But a powerful discharge from a nerve centre is followed by temporary impairment or abolition of its functions, and it is probable that the arrest of the functions of the higher nerve centres, caused by the application of a sudden and powerful stimulus, is the most important factor in the production of the phenomena of shock. The symptoms of erethismic shock may be explained partly on the supposition that the nervous tissues are in the irritable condition frequently observed when they are imperfectly nourished, and partly on the supposition that the phenomena of excitement are in great part due to the abolition of the functions of the higher nerve centres, thus permitting a greater activity of the lower nerve centres to take place.

The symptoms of spinal and cerebral concussion have both been attributed by one set of authorities to vascular spasm, and by others to vascular paralysis, but they are most probably caused by molecular disturbance of the gray matter of the spinal cord and brain respectively, which is of such a character that it is attended by impairment or loss of the functions of the nerve cells and fibres. In contusion of the brain the part that is the chief centre of the injury is more or less permanently

damaged and its functions destroyed, while a widely diffused molecular disturbance occurs in the remaining parts of the brain. In compression of the brain the functions of the organ are abolished partly because the whole brain is rendered anæmic by the pressure to which the organ is subjected owing to the diminution of the cranial cavity, and partly by the molecular disturbance produced by the suddenness of the injury.

Treatment.—The great aim of treatment is to excite reaction, but it must be borne in mind that when once reaction appears it is apt to become excessive. If the threatened arrest of the heart's action is of purely nervous origin, Savery recommends that blood should be immediately drawn from the external jugular vein, but if it be caused by hemorrhage then transfusion of blood appears to afford the best chance of success. In every case of shock the patient should be well wrapped up in warm blankets and surrounded by hot bottles. Stimulants must now be given internally, brandy being generally the readiest and best. If the patient be unable to swallow, ammonia or ether may be injected subcutaneously or into a vein, or a stimulating enema may be given. Tincture of digitalis may be administered in half drachm doses, but its action is too slow to be of much use in urgent cases.

The treatment of *concussion* consists of absolute and prolonged rest. One of the most important rules of treatment is to abstain from giving stimulants during the stage of collapse except in unusually severe cases. In this stage the patient should be surrounded by warm blankets and hot bottles, and absolute rest in a darkened room should be enjoined until the stage of reaction is passed. During the stage of reaction a moderate purgative may be given, and ice may be applied to the head if agreeable to the patient, while the diet should be plain and unstimulating consisting chiefly of milk. Spinal concussion must be treated on the same general principles as the cerebral variety. The patient should be made to lie on his face or side, or on his back, as recommended by Erichsen, on a couch which is tilted a little at its foot. When the case has become chronic the usual remedies for meningomyelitis must be employed. Contusion and compression of the brain from external injury come under the care of the surgeon.

CHAPTER XV.

VASCULAR DISEASES OF THE BRAIN.

I. ANÆMIA AND OCCLUSION OF THE INTRACRANIAL VESSELS.

Etiology—Cerebral anæmia may be divided into 1, *universal* anæmia, which results from the same causes as anæmia of the body generally; and 2, *partial* anæmia, which is caused by obstruction of one of the cerebral vessels.

1. Universal Cerebral Anæmia.

Symptoms.—The universal form of the disease may be divided into *a*, acute; and *b*, chronic universal cerebral anæmia.

a. Acute Universal Cerebral Anæmia.—The initial symptoms of the anæmia which is caused by severe hemorrhage, are obscuration of the senses; buzzing in the ears; dizziness; contraction, followed by dilatation of the pupils; imperfect reaction to external stimuli; and loss of consciousness. The surface becomes cold and pale, the respiratory movements, accelerated at first, become slow, and this condition is frequently followed by general convulsions and coma. In an ordinary *fainting fit* or syncope there is at first some degree of mental incoherence manifested by the inability of the patient to direct his attention to a particular object, a feeling of oppression in the chest, along with a tendency to yawn. The face becomes pale, a cold perspiration breaks out on the forehead and sometimes over the entire body, and there are general muscular relaxation, ringing in the ears, dimness of sight, nausea, and sometimes vomiting. The pulse is small, compressible, but regular. The patient may now recover or fall insensible to the ground, and after a few moments in the recumbent position he begins to rally.

b. Chronic Universal Cerebral Anæmia.—In these cases the patient is fretful and restless, his sleep is disturbed by dreams, while he suffers from intolerance of light and sound and great mental irritability. These symptoms are frequently succeeded by the phenomena of depression, and sometimes the latter predominate from the first. The patient suffers from almost constant headache, vertigo, nausea, and faintness.

The pulse is small and compressible, the cardiac impulse feeble, and there is great disinclination for either mental or physical exertion.

In the severe forms of chronic or subacute cerebral anæmia, such as that produced by starvation, or that which arises during the course of exhausting fevers, delirium becomes a prominent symptom. During the delirium of cerebral anæmia the patients are excited and sometimes maniacal; there are illusions of sight and hearing, and delusions of persecution. The duration of this condition is variable; it may last a few hours or days only, but it sometimes continues for weeks, and occasionally passes into permanent insanity.

Cerebral anæmia is seen in infants after severe diarrhœa, or other exhausting disease; and as this is the form which was called by Marshall Hall *hydrocephaloid* or *hydrencephaloid* disease, it demands special notice. The affection may be divided into two stages—that of irritability, and of torpor. In the first stage, the infant is irritable and restless, with flushed face, warm skin, and frequent pulse; the patient starts on being touched or on hearing any sudden noise, and sleep is disturbed and interrupted by sighs, moans, or screams. During the second stage the countenance becomes pale, the cheeks and extremities cold, the eyelids are half closed, the eyes sunk in their sockets, there is frequently slight strabismus, and the pupils are dilated and do not contract to light. The breathing is irregular and sighing, the voice husky, and there is sometimes a teasing cough with rattling in the throat. A most important symptom which distinguishes this disease from hydrocephalus is that the fontanelle, instead of being tense as in the latter disease, is depressed. The child inclines almost constantly to fall into a sleep, which may pass into coma and death, but under appropriate treatment gradual recovery usually takes place.

2. Partial Cerebral Anæmia (Occlusion of the Intracranial Vessels).

Occlusion of the cerebral vessels may take place in *a*, the arteries; *b*, the veins and sinuses; and *c*, the capillaries of the brain.

a. Occlusion of the Cerebral Arteries.

Etiology.—The arteries of the brain may be occluded by a substance carried from a distant part of the circulation, constituting *embolism*; or by a clot formed on the spot, constituting *thrombosis*.

An *embolus* consists of a fibrinous mass washed away from the left cavities of the heart, the mitral or aortic valves, or the arch of the

aorta. In cases of cancer of the lungs, a nodule of the growth may possibly be carried from the pulmonary veins, and pass into the cerebral vessels.

Thrombosis of an artery occurs most frequently when the internal surface of the vessel is rendered rough and uneven by arterial degeneration. The predisposing causes of thrombosis are all those conditions which tend to diminish the force of the heart's action and to alter the quality of the blood, and thrombosis is particularly liable to occur when these general causes and local arterial degeneration are conjoined. Embolism occurs most frequently in young, and thrombosis in old people.

Symptoms.—The symptoms of embolism of a cerebral artery are almost identical with those of cerebral hemorrhage in their mode of onset and general characters, but the unconsciousness caused by embolus is, as a rule, more transient than that caused by hemorrhage. In some cases the patient is suddenly attacked with dizziness and utters an involuntary cry, or complains momentarily of headache and immediately loses consciousness, but in other cases the patient only complains of dizziness and slight confusion of mind for a minute or two and becomes suddenly paralyzed and often aphasic, but without loss of consciousness. In still other cases the attack is ushered in by an attack of convulsions, which may be general like an ordinary epileptic attack, or limited to one-half or to a part of one-half of the body. When general convulsions are present they occur simultaneously with the loss of consciousness and are immediately followed by paralysis, while unilateral and partial convulsions may recur repeatedly before paralysis is fully established. In many cases sudden speechlessness constitutes the only symptom of embolus of a cerebral artery and in these cases this symptom may disappear when collateral circulation is established, but, as a rule, the aphasia is associated with right-sided hemiplegia. The state of the pupils varies during the attack.

The *symptoms of thrombosis* are, as a rule, more gradual in their development than those of embolus. The more usual premonitory symptoms of thrombosis of a cerebral artery are headache, dizziness, a sense of general confusion, numbness, coldness, or formication of one extremity or of one-half of the body, and considerable mental disturbance, especially, marked loss of memory.

Convulsive movements occasionally precede the appearance of paralysis, but, as a rule, the loss of motor power begins gradually, and its progress is marked by successive remissions and exacerbations until ultimately a more or less extensive paresis is established. The duration of the prodromal stage may vary from a few days to a few months, and occasionally apoplectic symptoms may come on suddenly as in embolism.

When once the vessel has become completely occluded the further progress of thrombosis is like that of embolus of a corresponding vessel.

When softening occurs either from embolus or thrombosis, the temperature, according to Bourneville, begins to rise on the second or third day of the attack and in two or three days it may reach 40° C. (104° F.). After a few days longer the temperature sinks rapidly, its decline being more rapid than after the period of inflammatory reaction in cases of hemorrhage. When once softening has become thoroughly established the symptoms are generally the same as those of other localized cerebral diseases, while the sensory, trophic, and vaso-motor disorders, and the affections of the special senses are the same as those occurring in cerebral hemorrhage. In some cases of embolism, however, the ophthalmic artery is occluded, and the patient becomes suddenly blind on that side, while an ophthalmoscopic examination reveals the usual signs which indicate occlusion of the central artery of the retina.

In cases of embolism the signs of valvular disease of the heart or of disease of the large arteries are detected on physical examination, and in cases of thrombosis the signs of degeneration of the arteries are present.

In some cases of occlusion of cerebral arteries the symptoms begin to improve at an early period and the patient may ultimately recover completely. In these cases it is evident that collateral circulation has become established before softening has taken place. In cases in which recovery has taken place the patient is liable to be suddenly attacked by occlusion of other vessels.

b. Thrombosis of the Cerebral Sinuses.

Etiology.—Thrombosis is prone to occur in the cerebral sinuses because they are traversed by bands of connective tissue, are destitute of muscular walls, and are rigid tubes incapable of collapsing. Thrombosis of the sinuses is particularly liable to occur in infants during the collapse induced by severe diarrhœa, while in adults it occurs in cases of profuse suppuration, cancer, senile marasmus, and other diseases associated with great debility. In other cases inflammation of the sinuses is produced by disease or injury of the cranial bones, which is followed by the formation of purulent thrombi. Erysipelas of the head and face, furunculus of the face, and purulent inflammation of the deep muscles of the neck, are other causes of thrombosis of the sinuses.

Symptoms.—The symptoms of thrombosis of the sinuses in children consist of convulsions or paralysis in addition to the collapse, and somnolence caused by the cerebral anæmia which results from an exhaust-

ing diarrhœa. The motor symptoms most commonly observed are rigidity of the muscles of the neck and sometimes also of those of the limbs, nystagmus, strabismus, ptosis, and paresis of the facial muscles. The fontanelles are at first depressed as in the hydrencephaloid disease described by Marshall Hall, but as the disease advances either effusion of serum or extensive meningeal or intra-cerebral hemorrhages may take place and the fontanelles become less tense and the cranial bones pushed apart.

Thrombosis of the sinuses from marasmus in adults give rise to very indefinite symptoms, and at times a slight degree of apathy and general depression are the only symptoms present. At the outset the patient may complain of headache, nausea, and vomiting, but these soon give place to coma, and in a few cases loss of consciousness is preceded by delirium, which occasionally assumes a maniacal character. The most usual motor disturbances are strabismus, trismus, tremors and contractures, epileptiform convulsions, or paralysis, which may involve one-half or both sides of the body. At other times paralysis and contractures may be associated, one extremity being the seat of contracture and the other of paralysis. Swelling of the veins outside the skull affords a valuable sign of thrombosis of the sinuses. The superior longitudinal sinus communicates directly with the veins of the nasal cavities and with those on the upper surface of the skull, and consequently thrombosis of it may give rise to epistaxis, or to distention of the veins which run from the temples on both sides to the anterior fontanelle, and there may also be cyanosis of that part of the face which is supplied by the anterior facial veins.

The lateral sinus communicates with a small vein which traverses the mastoid process, and in thrombosis of the sinus localized œdema behind the ear occasionally may make its appearance. Simultaneous thrombosis of both lateral sinuses gives rise to the same symptoms as occlusion of the superior longitudinal sinus.

The cavernous sinus communicates with the ophthalmic vein, and in thrombosis of the sinus, venous hyperæmia of the fundus oculi has been observed, along with œdema of the eyelids and conjunctiva, and prominence of the eyeball caused by congestion of the retrobulbar frontal veins. Interference with the nutrition of the nerves which pass along the floor of the sinus may also give rise to paralysis of the oculo-motor nerves, trigeminal neuralgia, and neuroparalytic ophthalmia.

The *phlebitic* variety is generally complicated with meningitis and cerebral abscess, and it is not, therefore, always possible to distinguish between the symptoms caused by each of these morbid conditions. In the few cases in which suppurative thrombosis was alone present, the

symptoms were the same as those of septicæmia with pronounced cerebral symptoms. The attack generally begins with chilliness, which recurs repeatedly during the course of the disease, and the patient has a characteristic typhoid look, with dry tongue, loss of appetite, and mental confusion. After a time the patient falls into a somnolent condition which gives place to coma, which soon terminates fatally. When suppurative thrombosis is complicated with meningitis, more pronounced sensory and motor symptoms are present, consisting of headache, hyperalgesia, paresis, paralysis, or convulsions. The disease sometimes pursues a latent course, and is only discovered after death.

c. Occlusion of the Cerebral Capillaries.

Etiology.—In severe cases of intermittent fever the cerebral capillaries are liable to be obstructed by dark colored masses which have been called *pigment embolism*. The cerebral capillaries may also be obstructed by oil globules, constituting *fat embolism*. The cerebral capillaries are said to be sometimes occluded by lime becoming deposited on their walls, a process named by Virchow *lime metastasis*. Capillary embolism is supposed to give rise to chorea, but this subject will be discussed hereafter.

Symptoms.—If the embolic masses are few in number they do not give rise to symptoms which can be recognized during life, but if a considerable part of the brain is deprived of its nourishment the patient suffers from diffused cerebral symptoms such as dizziness, headache, nausea, trembling, weakness of the extremities, and loss of memory with other signs of mental decay. If the circulation is suddenly arrested in a large portion of the brain the symptoms of a localized destructive disease of the cerebrum may be produced.

Morbid Anatomy.—Capillary occlusion can only be discovered with the aid of the microscope. The first effect of these emboli is to cause anæmia, and subsequently the various stages of necrobiosis up to complete softening are produced.

Treatment.—The treatment will depend upon the cause of the emboli and must be conducted on general principles.

II. CONGESTION OF THE BRAIN.

Etiology.—Congestion of the brain may be either active or passive, and is produced by the same general causes as congestion of other organs. It is, indeed, asserted by Moxon that congestion of the brain does not give rise to symptoms which can be set up as a distinct disease,

but apoplectiform attacks occur in the course of such diseases as locomotor ataxia, general paralysis, and sclerosis in patches, for which it is difficult to assign any other cause than congestion, and although it is probable that congestion has been often assumed to exist without due warrant, we do not feel ourselves justified in rejecting the condition as a cause of a distinct and separate disease.

Symptoms.—Congestion of the brain gives rise to symptoms which vary widely in different cases, but for clinical purposes three varieties may be described: (*a*) the *slight*, (*b*) the *severe*, and (*c*) the *apoplectic* form.

(*a*) In the *slight* form of congestion the prominent symptoms are sensory disorders. The patient complains of severe headache, either deep-seated or lancinating, aggravated by movement, light, sound, or heat, while all intellectual efforts become impossible. Patients at the same time complain of dizziness, tinnitus aurium, and optical illusions. Sleeplessness is an early and important symptom. It is accompanied by restlessness and agitation, and if sleep do supervene, it is disturbed by horrible dreams, and the patient awakes unrefreshed without relief to the headache.

In venous hyperæmia the phenomena of mental depression are usually more marked than those of excitement. There is a dull sense of oppression in the head, the face is livid, there is mental torpor with a tendency to sleep, and a certain amount of confusion of ideas, especially on awaking after a short sleep.

(*b*) In the *severe* forms other symptoms are added. The patient suffers so much from vertigo that he is unable to maintain the erect posture, and sudden vomiting may occur in the absence of any gastric irritation to account for it. The pulse is slow, full, and hard; the arteries of the head and neck beat forcibly; the face is frequently, although not always flushed, and may at times be livid, while a sensation of flying heat shoots over the head and neck. The pupils are generally contracted, and there is some degree of intolerance of light and sound. The patient complains of intense headache, and is the subject of hallucinations and illusions which pervert the judgment, and may lead to strange and disorderly acts. He sometimes endeavors to quit his bed, and to pursue or run away from imaginary objects; he is loquacious or bursts into a flood of tears, and struggles with and tries to escape from his attendants. After some hours of excitement and struggling the skin becomes covered with sweat, the pulse is accelerated, the face of a deep red color, and the patient presents the leading symptoms of encephalitis; the thermometer shows that the temperature, if at all altered, is only slightly above the normal. If these symptoms

persist for some time, the phenomena of excitement are succeeded by those of depression, the delirium gradually gives place to mental torpor, the muscular agitation is replaced by muscular relaxation, the respiration becomes stertorous, there are involuntary evacuations, and the patient falls into a state of coma. An abundant secretion from the conjunctivæ and mucous membrane of the mouth is said to be a frequent symptom of congestion of the brain in old people. It is very probable that many of the cases which have been described as examples of cerebral congestion ought to be regarded as the delirium or mania which so frequently follows a slight epileptic attack.

The symptoms of cerebral congestion in infancy are somewhat similar to those of meningitis. Both are attended with partial or general convulsions, headache, contraction of the pupils, vomiting, and constipation. The course of the two affections, however, enables them to be readily distinguished. In congestion the child has been in good health up to the beginning of the attack, there is little or no elevation of temperature, and the disease terminates in recovery in two or three days at most.

(*c*) The *apoplectic* form is characterized by sudden and total loss of consciousness and complete resolution of the limbs, but reflex excitability is preserved. The patient recovers consciousness in a few hours, and after a short time—two or three days at most, all the symptoms disappear without leaving a trace behind. Sometimes, however, after complete restoration to consciousness, a certain amount of muscular paralysis remains in one limb, or it assumes the hemiplegic form and persists for some time. Apoplectiform attacks of this kind are liable to occur in the course of locomotor ataxia, sclerosis in patches, and general paralysis of the insane. Patients suffering from these diseases are sometimes seized with general convulsions and die comatose in a few hours, and at the autopsy no coarse lesion of any kind is discovered in the brain.

III. INTRACRANIAL HEMORRHAGE.

Intracranial hemorrhage may be divided into 1, *cerebral;* and 2, *meningeal* hemorrhage.

1. Cerebral Hemorrhage (Apoplexy).

Etiology.—Some families exhibit a predisposition to cerebral hemorrhage, but this tendency is only an indirect result of an inherited tendency to a general arterial degeneration. Cerebral hemorrhage is rare

before the fortieth year of age, and it occurs more frequently in men than in women, but age, sex, as well as occupation and climate, seem to predispose to it only in so far as they tend to induce degeneration of vessels. The chief circumstances which predispose to cerebral hemorrhage are (1) diseases of the bloodvessels, (2) increase of the arterial tension, (3) disease of the tissues surrounding the vessels, and (4) certain diseases of the blood.

(1) *Diseases of the Vessels.*—The great majority of massive hemorrhages into the substance of the brain are due to degeneration of those branches of the Sylvian artery which pass through the anterior perforated space to reach the corpus striatum. By far the most frequent cause of intracerebral hemorrhage is that condition of the arterioles which has been described by Charcot and Bouchard as *miliary aneurisms*. These aneurisms are of a reddish color, and vary from the size of a millet seed to that of a pin's head. Sometimes a few only are found in the neighborhood of the ruptured vessel, while at other times they are scattered in large numbers throughout the whole brain. The parts of the brain in which they are situated, in the order of decreasing frequency, are the lenticular nucleus, the optic thalami, the pons, the convolutions, the caudate nucleus, the cerebellum, the medulla oblongata, the middle peduncles of the cerebellum, and the centrum ovale. Miliary aneurisms result, according to Charcot and Bouchard, from a kind of arterial sclerosis of the nature of a chronic periarteritis. Atheroma of the vessels may also lead to aneurisms of the larger and medium sized arteries of the brain, and although rupture of these are apt to give rise to hemorrhages on the surface of the brain, yet the hemorrhage passes into the substance of the brain more frequently than is generally supposed. Atheromatous degeneration may also cause hemorrhage indirectly by rendering the walls of the larger vessels rigid, so that the pulse-wave reaches the arterioles without being modified by the normal elasticity of the arteries.

Primary fatty degeneration of the cerebral bloodvessels occurs at all ages, and Paget believes that it sometimes predisposes to cerebral hemorrhage in young people.

(2) *Vascular Tension.*—It is doubtful whether increase of the arterial tension ever gives rise to cerebral hemorrhage in the absence of disease of the vascular walls, but when arterial degeneration is present any increase of tension becomes a powerful predisposing cause of hemorrhage. Sudden exposure to cold may increase the arterial tension by inducing extensive contraction of the cutaneous arteries. During the winter months it is very common for individuals to be found in a comatose condition on the streets and taken up by the police on the

supposition that they are drunk. Such accidents usually occur in persons of middle age; the breath may smell of alcohol, and the person attacked may be known to have been drinking during the evening. The evening has been spent in a heated apartment, when, under the conjoined influence of a high temperature, alcohol, and emotional excitement, the cutaneous vessels have become dilated, the skin bathed in perspiration, and the cardiac action accelerated. On going out into the cold air the surface becomes suddenly chilled, and the arterial tension is rapidly augmented; the internal organs become gorged with blood; and if, as is frequently the case, the walls of the cerebral vessels are weakened by disease, rupture of one of them is apt to occur.

The hypertrophy of the left ventricle, which takes place in contracted kidney, taken along with arterio-capillary fibrosis, which invariably accompanies it, is a frequent cause of cerebral hemorrhage.

(3) *Condition of the Tissues.*—The theory that cerebral hemorrhage is generally preceded by softening of the cerebral tissue, was advanced by Rochoux, and he gave to this supposed process the name of *ramollissement hémorrhagipare*, but it is now generally admitted that when the softening is not caused by occlusion of vessels, it is secondary to the hemorrhage.

(4) *The State of the Blood.*—Cerebral hemorrhage has been occasionally observed in the course of diseases of the blood, like scorbutus, purpura, chlorosis, leucocythemia, pernicious anæmia, icterus, typhoid and other specific fevers.

Symptoms.—An attack of cerebral hemorrhage may begin suddenly without any warning, but in many cases premonitory symptoms are present some days or even weeks before the actual onset of the attack. The usual forerunners of an apoplectic attack are dizziness, headache, ringing in the ears, muscæ volitantes, numbness in the hand or foot, muscular twitchings of the face or some portion of the extremities, mistakes in talking or writing, vomiting, mental irritability, and drowsiness. These symptoms, either separately or in various combinations, are to be regarded as warnings of apoplexy only in old people, or in middle-aged people with evidences of arterial degeneration. The mode of onset of an attack of cerebral hemorrhage may be divided into (1) the apoplectiform, (2) the epileptiform, and (3) the simple mode of onset.

(1) *Apoplectiform Onset.*—This mode of onset is characterized by sudden loss of consciousness and resolution of the limbs, either with or without warning, constituting what is popularly called apoplexy.

When the apoplectic attack is well marked the patient lies in a state of profound coma, and is insensible to all kinds of stimuli. The face is usually flushed and swollen, although it may occasionally be pale

and clammy; the lips are livid; the head and neck feel warm and bathed in perspiration; the carotids and other arteries throb violently; the eyelids are closed; the conjunctivæ are ingested; the eyeballs are fixed; the pupils are sluggish to light; the respiration is usually deep, with or without stertor and protrusion of the cheeks during expiration; the pulse is generally full and slow; and there is either complete muscular resolution, so that the limbs when raised drop like inert bodies, or the resolution is more marked on one side of the body than on the other. In the severer cases there is not only complete absence of voluntary motion, but all reflex movements are lost, with the exception of the cardiac and respiratory movements and those concerned in the second act of deglutition. In such cases the patient may die after a few minutes, a few hours, or a few days. In cases which are less severe from the first or in which some degree of recovery has taken place, powerful irritation causes reflex movements, and in still less profound cases the patient, when loudly spoken to, raises his eyelids for a moment or two, and may even reply in a monosyllable when loudly pressed with any question. In such cases a difference can be detected between the two halves of the body; the extremities of one side offer some degree of resistance to passive movements, while those of the other side sink, when unsupported, like inert masses, and the corner of the mouth is lower on one side than on the other.

(2) *The Epileptiform Onset.*—In this mode of onset the patient, either with or without prodromata, drops down insensible in a kind of epileptic fit, and after a time it is discovered that he is paralyzed on one side of the body. The convulsions may be limited to one-half of the body, and this side is subsequently paralyzed; but if the convulsions occur after paralysis has become established the non-paralyzed side is the one which is attacked with clonic spasms. In some of the cases initiated by convulsions the patient may remain in a somewhat lethargic condition, with eyes closed or only half open, for a period of from three to six weeks and yet recover, but if deep coma continues for forty-eight hours the case generally terminates fatally.

(3) *Simple Mode of Onset.*—In the simple onset the patient may suddenly fall from paralysis, but without loss of consciousness. He generally complains of a feeling of "numbness" of the paralyzed side, but does not suffer pain.

The *temperature* is lowered immediately after an apoplectic attack, reaching in some cases 96.5° F., and in the fulminating forms it remains low until death. If life continue for from ten to twenty hours the initial sinking gives place to an elevation of temperature. In fatal cases the secondary rise may reach 108° F. or even higher before death, but

in favorable cases it continues to oscillate for some days between 99° F. and 100.5° F. If the case is to terminate favorably the temperature now gradually sinks to the normal, but if a fatal termination is to take place the more or less stationary temperature of the first few days is followed by a rapid and continuous rise, just as occurs after the period of initial lowering.

Conjugate deviation of the eyes, with rotation of the head and neck, occurs as a temporary symptom in all cases of severe cerebral hemorrhage, the deviation being directed away from the paralyzed side when the lesion is situated in the hemisphere and towards it when it is situated low down in the pons. The eyes are usually fixed, but occasionally exhibit slight nystagmus. The rotation may completely disappear when the patient falls asleep and the symptom usually lasts only a few days.

The *permanent symptoms* of cerebral hemorrhage are the various forms of hemiplegia with rigidity of the paralyzed muscles which will be subsequently described.

Vaso-motor disorders are generally observed in the paralyzed limbs soon after the attack; the paralyzed limbs are redder and warmer than the corresponding healthy limbs, the difference in temperature varying from a fraction of a degree to 2° F., and they may also become swollen from a slight degree of subcutaneous œdema. The temperature of the paralyzed limbs gradually decreases and is eventually lower than that of the sound side, and when the œdema disappears the skin becomes dry and scaly. Congestions and even hemorrhage of the lungs, and extravasations into the pleura, endocardium, mucous membrane of the stomach, the suprarenal capsules, and the kidneys frequently accompany cerebral hemorrhage, and are often limited to the paralyzed side of the body.

The *trophic disorders* consist of acute bedsores, which may appear on the gluteal region of the paralyzed side soon after the attack. Acute inflammation of one or more of the large joints, or chronic arthritis of the small joints, may begin on the paralyzed side generally in from three to six weeks after the onset. In some cases the patient complains of pain on pressure in the paralyzed limb, which is especially well marked along the course of the principal nerve trunks, and Cornil has shown that it is most probably caused by an inflammatory hypertrophy of the nerves or of their sheaths. In some exceptional cases the paralyzed muscles undergo early and rapid wasting, and when the hemiplegia occurs in childhood, the affected limbs grow more slowly than the corresponding healthy ones, and the former are consequently smaller in all their dimensions than the latter. The skin on the paralyzed side sometimes feels thicker than on the healthy side, while the hair some-

times grows better on the affected than on the unaffected side. The nails on the paralyzed side become yellowish, marked with ridges, brittle, and curved.

The mental condition of the patient is seldom fully restored. In most cases the intellectual condition is permanently enfeebled, so that the patient feels unequal to any unusual intellectual effort, while in other cases the intellect undergoes progressive declension, and the patient is reduced to a state of childishness or pronounced dementia. At other times the patient becomes peevish, whimsical, irritable, and gives way to outbursts of passion. The disorders of speech which occur in cerebral hemorrhage will be subsequently described.

2. Meningeal Hemorrhage.

Etiology.—The most frequent causes of meningeal apoplexy are injuries of the skull producing rupture of arteries, veins, or sinuses, rupture of aneurisms of larger arterial branches, and thrombosis of the sinuses. Meningeal hemorrhage may occur in the course of acute infectious diseases, chronic dyscrasiæ, and progressive paralysis of the insane, while the meningeal apoplexy of newborn children is caused by certain accidents attending childbirth.

Symptoms.—Meningeal hemorrhage caused by rupture of an aneurism forms the least complicated form of the disease, inasmuch as hemorrhages of traumatic origin are usually complicated by other cerebral symptoms. In severe cases of uncomplicated meningeal hemorrhage the patient becomes suddenly apoplectic without any warning, or with only slight premonitory symptoms, such as headache, dizziness, and vomiting. The paralysis commonly affects all four extremities, and hemiplegia is only rarely met with. The onset is often marked by epileptiform convulsions and vomiting, profound coma supervenes, and the patient dies in a few hours or at most in a few days. In less severe cases the patient may recover consciousness after a few hours, and he then complains of headache and is often delirious, and after a short period of somnolence he finally becomes comatose. In a few cases the symptoms creep on gradually; the patient complains at first of headache, dizziness, and numbness or weakness of the extremities on one or both sides, and after a time he falls into a condition of stupor and ultimately dies comatose.

If an aneurism of considerable size has existed for some time before the occurrence of hemorrhage, it may give rise to the symptoms of an intracranial tumor, which will be subsequently described.

In the meningeal hemorrhages of the newborn, the children are either born dead, or in a condition of asphyxia, and often die soon afterwards. If respiration is established the infant lies in a comatose condition for some days or weeks, and during this time he suffers from frequent attacks of convulsions, which are generally most pronounced on one side of the body. In such cases the disease may prove fatal by coma or the patient may gradually recover so far as life is concerned, but he remains the subject of one or other of the forms of spastic paralysis of infancy already described.

Prognosis.—The case will terminate fatally at an early period if the patient cannot be roused at all, and if the reflex action of the conjunctiva is abolished, the urine and feces are passed involuntarily, and there is well-marked stertor, while the persistence of even a slight degree of these symptoms without abatement is a sign of great gravity. A quick, irregular pulse with labored respiration, and decided difficulty of deglutition and articulation, are also very unfavorable signs. A marked and persistent depression of the pulse is of fatal significance, while a marked and persistent rise of temperature is an almost equally grave sign. A sudden depression of temperature with increase or renewal of a preëxisting comatose condition is also of serious import, indicating, as it does, the occurrence of a fresh hemorrhage. Acute bedsores on the buttock of the paralyzed side is a fatal sign.

Treatment.—The treatment of cerebral anæmia must vary according as the disease is acute or chronic, and as it is limited to the brain or affects the entire body. In an ordinary *fainting fit* the patient should be as speedily as possible placed in the recumbent posture, and recovery usually takes place without further treatment. If the symptoms are more persistent, some form of local irritant must be employed, such as sprinkling the face with cold water, or flecking it with the corner of a towel dipped in cold water, the application of the faradic current to the surface by means of the metallic brush, the inhalation of ammonia to stimulate the olfactory and trigeminal nerves, or a stimulating enema. As soon as the patient can swallow, stimulants, such as coffee or brandy, must be given, especially if the heart's action be feeble. In severe cases of cerebral anæmia, after profuse hemorrhage, the body of the patient ought to be covered with warm clothing and surrounded by bottles containing hot water, and in order to increase the flow of blood towards the brain the head ought to be kept in a low position, while pressure is maintained over the abdominal and axillary arteries. In cases of severe and prolonged anæmia from loss of blood transfusion ought to be tried as a last resort. The cerebral anæmia which arises during the course of acute diseases must be treated by the judicious use

of wine and nourishing diet, and if the case admit of it by such tonics as quinine, iron, and arsenic. When delirium or other form of cerebral excitement accompanies the anæmia, the great aim of treatment should be to procure sleep, and a full dose of chloral or better still an opiate should be administered. Hydrocephaloid disease should be treated on the same general principles as other forms of cerebral anæmia and in addition the diarrhœa or other disease which has caused the anæmia must be attended to.

Occlusion of cerebral vessels must be treated on the same general principles as hemorrhage from them, but in embolus the cardiac complication must be subjected to treatment, while in thrombosis the presence either of syphilitic endarteritis or of atheromatous degeneration of the arteries must be borne in mind and the appropriate treatment adopted for each.

Congestion of the brain must be treated by rest in the recumbent position with the head raised, ice to the head, and active purgation. The diet should be plain and unstimulating, but nourishing. Moderate doses of ergot are supposed to have a favorable influence on the disease.

In cerebral hemorrhage the aims of treatment are (1) to avert a threatened attack; (2) to treat the apoplectic condition; (3) to allay excitement during the inflammatory reaction; and (4) to improve the permanent symptoms.

(1) *Prophylactic Treatment.*—When a person complains of any of the usual premonitory symptoms of apoplexy the treatment must vary according to the state of the general health. The condition of the heart and bloodvessels must be carefully explored, and the urine must be tested for albumen and sugar, and the treatment must be adapted to meet any disorder of the general health which may be discovered. Saline purgatives are generally useful by lowering the arterial tension, and during the continuance of threatening symptoms the patient should be kept quiet in a darkened room and cold applied to the head.

(2) The apoplectic attack must be treated by absolute rest, and cold to the head, and if the arterial tension be high a moderate bleeding may do good, but the severe general bleeding practised a few years ago is quite inadmissible. When there are signs of cardiac failure, or when the breathing is intermittent or assumes the Cheyne-Stokes character, then a stimulating treatment must be adopted, but the common practice of applying blisters to the shorn scalp or to the nape of the neck, or mustard plasters to the calves of the legs, is useless and ought to be strongly condemned.

If the attack is preceded or accompanied by recurring epileptiform attacks, or if the patient is restless and there is more or less of delirious

wandering, bromide of potassium may be given. If the bowels are constipated an enema containing castor oil or oil of turpentine should be administered, or two drops of croton oil may be given in a suitable vehicle or placed on the back of the tongue. The state of the bladder should be carefully examined and a catheter used if necessary.

(3) If the patient survive the first shock of the apoplectic attack, the less we interfere during the first days the better. He must be kept as quiet as possible both in body and mind, his diet should consist of liquid nourishment, and the state of his secretions and excretions should be carefully regulated. Cold should be applied when the symptoms of inflammatory reaction appear, and if the patient complain of headache, or there be persistent wakefulness or delirium, a full dose of bromide of potassium, or even an opiate or chloral should be given. During this period great care must be taken to prevent bedsores on the paralyzed side by paying constant attention to the state of the bedding and securing extreme cleanliness. In severe cases the patient should, when practicable, be placed from the first on a water bed.

(4) The most effectual method of promoting the improvement of the paralyzed nerves and muscles is a thorough attention to the general health of the patient, and the treatment proper for each case will depend upon his age, habits, and constitution, and on the presence or absence of any concomitant disease. The general principles of treatment are to take care that the patient has a sufficiency of easily digestible and nutritious food; that all circumstances which might cause mental excitement are avoided; and that he has a due amount of repose and sleep. In the hemiplegias of elderly people, in which miliary aneurisms may be presumed to exist, care must be taken that the circulation is not subjected to any sudden strain, and it is, therefore, necessary to keep the bowels moderately open lest straining at stool should induce another attack. The patient should take open air exercise by walking on level ground, or in a chair or carriage, and much good may be done by sponging with salt water. When there is advanced arterial degeneration or high arterial tension the water ought to be tepid, and hemiplegic patients, as a rule, should only use baths of moderate temperature. Iodide of potassium is the most likely drug to promote the absorption of the clot, and its administration may be commenced immediately after the febrile symptoms of the inflammatory reaction have subsided. In ordinary cases, from three to five grain doses three times a day will suffice; but if the hemorrhage is caused by premature degeneration of the arteries from syphilis, it will be advisable to give from fifteen to twenty grain doses three times a day, and in some cases it will be necessary to give mercury in some form along with the iodine salt.

The local treatment of the paralyzed limbs consists of passive movements, and friction of the skin by means of a flesh brush, flannel, or the palm of the hand. After a time the patient should be directed to move the paralyzed limbs by voluntary effort, and various gymnastic exercises may be adopted to bring the paralyzed muscles into separate and combined action. The constant current may be employed by placing one electrode on each mastoid process, or one on a mastoid process and the other on the nape of the neck, but in this method only feeble currents should be used, and the duration of the application should not exceed three minutes. According to another method, the current is passed through the paralyzed nerves and muscles in the usual way. The faradic current has been employed in contractures for the purpose of acting, not on the contracted muscles, but upon their antagonists, but this method has not been productive of much good. The methods used to promote the recovery of the paralyzed muscles are also the best remedies for the sensory disorders which are often present in cases of hemiplegia.

CHAPTER XVI.

ENCEPHALITIS.

Etiology.—The most frequent cause of acute inflammation and abscess of the brain is recent injury. When air gains access to the interior of the skull through an open wound, encephalitis becomes complicated with meningitis, but contusion of the brain may give rise to it in the absence of any perforating wound. Encephalitis may be set up by diseases of the bones of the skull, such as caries, or inflammatory action may be transmitted to the brain from diseases of the nose, antrum of Highmore, and orbit, while abscess of the brain frequently results from disease of the middle or internal ear. Multiple cerebral abscesses occur in connection with acute febrile affections, such as typhoid and scarlet fevers, and in putrid bronchitis and bronchiectasis, ulcerative endocarditis, and other pyæmic affections. Focal diseases of the brain like tumors, hemorrhage, thrombosis, and embolism, often set up surrounding inflammation in the brain, and in some cases encephalitis may be of idiopathic origin.

DIFFUSED OR GENERAL ENCEPHALITIS.

Cases of uncomplicated general encephalitis are not very common, most of the cases which have been reported as examples of this disease being examples of cerebral meningitis, or, more strictly speaking, of meningo-encephalitis. The symptoms of simple encephalitis are pyrexia, a busy and talkative but not violent delirium, passing on to somnolence and finally to coma. The presence of spasms would seem to indicate a complication with meningitis, while the appearance of partial or complete hemiplegia would show that the disease is more pronounced on one side than on the other, and therefore that the inflammation is partial.

PARTIAL OR LOCAL ENCEPHALITIS.

Local encephalitis may occur as 1, an idiopathic affection in children, when it has been named *poliencephalitis acuta infantium;* 2, as the result of an injury; 3, as a consequence of pyæmic infection; and

4, as a secondary result of a focal disease like (*a*) abscess, (*b*) hemorrhage, (*c*) softening from occlusion of vessel, and (*d*) tumor.

1. Poliencephalitis Acuta Infantium.

Acute inflammation of the brain occurs in infancy between the ages of one and six years. The attack may follow measles or scarlet fever, or be set up by a blow on the head, but in the majority of cases no exciting cause can be assigned, and the subjects of the disease do not even seem to belong to neuropathic families. The attack begins suddenly with fever, vomiting, and convulsions, and the child falls into an apoplectic condition which may last two or three days, occasionally one or two weeks, or even longer. The spasms are generally more pronounced on one side of the body than on the other, and are often strictly unilateral; after a longer or shorter time the convulsed limbs become more or less completely paralyzed. It is possible that many of these cases are fatal, but in other cases the child gradually recovers, except that he is the subject of a permanent hemiplegia. The further progress of this affection has been already described under the name of spasmodic hemiplegia of infancy, of which unilateral atrophy of the brain is the anatomical counterpart.

2. Traumatic Local Encephalitis.

When a contusion in the interior of the brain has taken place, the patient first suffers from the usual symptoms of concussion, and it is only when these have disappeared that the usual symptoms of a local encephalitis can be recognized. The patient lies in a semiconscious condition, and when roused complains of headache and dizziness, and staggers on attempting to walk. The pupils are variable in size, but they are generally equal and react slowly to light. The countenance is usually suffused, but at times it turns pale, and the pulse, which was frequent and irregular during the stage of concussion, sinks to sixty or seventy beats, but the thermometer may indicate the existence of fever of remittent type. When the encephalitis is situated in the frontal, temporal, or occipital lobes, the symptoms may be so slight that the patient feels quite well in a few days, or suffers for a week or two from very indefinite symptoms. Suddenly, however, more pronounced symptoms become manifest; the fever, which still continues of irregular type, becomes more intense; dizziness and headache become more marked; vomiting is not infrequently present; the pupils are dilated

and fixed; the pulse is slow; and the patient falls into a condition of stupor, which may be accompanied by delirium, or may pass directly into complete unconsciousness.

As the case progresses the symptoms of a localized disease may appear, consisting at first of spasmodic phenomena, such as rolling of the eyes, twitchings of the face or hands, or more decided clonic convulsions of the limbs, or even a general convulsion, but these are soon followed by paralysis of the abducens, motor oculi, or facial nerve, and in a few cases by hemiparesis or hemiplegia. The stupor now grows deeper, the urine and feces are passed involuntarily, the previously slow pulse becomes quick and irregular, and death takes place in coma. The course of the temperature is variable, but a continuous elevation until death is exceptional.

The duration of local encephalitis varies greatly, and an abscess of the brain has occasionally been found as early as the fifth day after the injury, and when air gains access to the brain through an open wound, and suppuration occurs, the disease may terminate fatally at a much earlier period. The patient may occasionally be restored to comparative health after an attack of traumatic encephalitis. The inflammatory focus may be transformed into a relatively innocuous condition, but even in those favorable cases chronic changes of a diffused character are occasioned which give rise to more or less permanent symptoms. The symptoms consist generally of some form of mental deterioration, such as melancholia, chronic mania, or a condition resembling general paralysis. Injuries of the skull are often followed by epilepsy, and may be the starting-point of intracranial growths, while a fall on the back of the head sometimes gives rise to diabetes.

3. Acute Pyæmic Encephalitis.

Acute pyæmic encephalitis is frequently ushered in by rigors, but these being symptomatic of the general disease, do not indicate the approaching complication. The first signs of the brain affection are afforded by severe headache, which is usually frontal; dizziness; delirium or somnolence; unilateral convulsions; and formication or numbness of the extremities. The disease makes rapid progress and the headache now becomes intense, the dizziness is so great that the patient is unable to sit up in bed, the mind is confused, and the delirium or somnolent condition of the first stage soon gives place to profound coma, which proves rapidly fatal. The intensity of the febrile symptoms is variable, but in many cases there is a considerable elevation of temperature.

4. Encephalitis Complicating Pre-existing Lesions of the Brain.

a. Acute Abscess with Encephalitis.

The symptoms caused by the acute encephalitis which accompanies caries of the petrous bone are often obscured by coexisting meningitis and thrombosis of the lateral sinuses. An abscess in the temporal lobe may attain a considerable size, and cause general symptoms of compression before giving rise to symptoms of local disease, inasmuch as this lobe does not contain any direct sensory or motor conducting tracts. Acute abscesses of the temporal lobe are consequently seldom recognized during life. The symptoms of acute abscess, from otorrhœa, or from caries of the other bones of the skull, begin with severe headache, vomiting, ringing in the ears, confusion of ideas, loss of memory, and delirium. As the disease advances the headache becomes more and more intense, the delirium gives place to stupor, epileptiform convulsions supervene, and the case soon terminates fatally amidst profound coma. Fever of variable type is usually present, the pulse is slow, and the pupils are contracted and sluggish. In some cases of abscess of the temporal lobe the general symptoms are complicated by those of a localized disease, and in these cases the abscess, which has rapidly attained to a considerable size, compresses the fibres of the internal capsule or the cerebral peduncle, and thus causes an incomplete hemiplegia with various sensory disorders, as well as paralysis of the oculomotor nerve, and occasionally of the facial. A few cases are associated with acute meningitis or thrombosis of the lateral sinus.

b. Chronic Abscess with Encephalitis.

Chronic abscess of the brain may be divided into (*a*) *primary*, and (*b*) *secondary* chronic abscess.

(*a*) Primary Chronic Abscess.

Primary chronic abscess is usually caused by some injury of the brain. All the symptoms, or nearly all, may disappear soon after the injury, and a period relatively free from symptoms may follow, forming the latent stage of chronic abscess. The average duration of the latent stage is, according to Lebert, from one to two months, but the period may vary in individual cases from a few days to years. When once a chronic abscess is formed, the symptoms caused by it are more or less

similar to those of cerebral tumor, and when the former is situated in the motor areas of the cortex and centrum ovale, or injure the sensory peduncular fibres, the symptoms of a local lesion are present from the beginning. The symptoms may be divided into those of (1) the *latent*, and (2) the *terminal* stages.

(1) *Symptoms of the Latent Period.*

The symptoms of the latent stage are those of a moderate degree of intracranial pressure. These consist of constant headache with paroxysmal exacerbations, and slight febrile disturbance, dizziness, nausea, and occasionally vomiting. The headache may be limited to the spot where the injury was received, or correspond to the part of the brain where the abscess is situated, the latter being often at a point of the brain exactly opposite the seat of injury. Paroxysmal exacerbations of the headache are indicative of congestion around the abscess, and when these frequently recur the abscess is likely to prove fatal within a brief space of time. In some cases monospasms, monoplegia, or other indications of a local disease in the brain are superadded to the general symptoms just described.

In other cases the patient has intervals of comparative freedom from all cerebral symptoms, but in the midst of comparative health he may suddenly complain of intense headache, and soon fall into a deep but transitory coma of several hours' duration, from which he rapidly recovers, such attacks being probably due to sudden pressure on the brain from congestion.

(2) *Symptoms of the Terminal Period.*

When once the terminal period begins, abscess of the brain generally leads to death in a few days. The symptoms of this period differ widely in individual cases, and the following groups may be distinguished:

(*a*) *Terminal œdema of the brain* is the most usual mode of termination of chronic abscess. In the majority of cases the tissues of the brain are compressed to such an extent that death by coma results in two or three days, while in a few cases the course of the symptoms is characterized by temporary improvements and aggravations, so that the fatal issue may be delayed for some time. In some cases there may be a transitory initial stage of irritation, characterized by mental irritability, restlessness, illusions, violent delirium, and a slight elevation of temperature. The irritative symptoms soon give place to those of depression, the patient complains of headache, the temperature falls, the pulse is slow, the pupils are dilated and react feebly to light, there is mental

confusion, and the patient falls into a somnolent condition, from which he may be roused temporarily when pressed by questions; but in a short time coma supervenes, and the case soon terminates fatally, the pulse becoming small, quick, and irregular before death.

The symptoms of local disease which may have been present during the latent stage of the abscess are variously modified in the terminal stage. Partial convulsions, which may have been present, become more violent and are soon followed by paralysis, or they may become transformed into a series of epileptiform attacks terminating in coma. General convulsions are sometimes caused by abscess situated in the latent regions of the hemisphere, and these are usually followed by profound coma.

(*b*) *Rupture of the Abscess on the Surface of the Brain.*—When abscesses make their way to the surface of the brain, an acute and rapidly fatal meningitis generally results. The symptoms of irritation are at first predominant, but the patient soon becomes unconscious, and dies comatose in a short time.

(*c*) *Perforation of the Abscess into the Ventricle.*—This occurrence causes a group of symptoms which may be recognized, if the existence of an abscess have been previously suspected. A sudden cerebral attack, attended by bilateral, but more or less partial convulsions, such as spasms of both legs, or of the facial muscles on both sides, is an indication of rupture into the ventricles, provided the patient be not already in an unconscious condition. General convulsions have sometimes been observed. Clonic spasms of the ocular muscles soon appear, caused probably by irritation of the corpora quadrigemina. The patient becomes rapidly unconscious, hemiplegia and death in profound coma take place generally in from four to twenty-four hours after the rupture of the abscess.

(*d*) Abscess of the cerebellum may terminate suddenly from arrest of the respiratory functions produced by pressure on the medulla oblongata.

(*e*) Occasionally the brain is found in a condition of remarkable anæmia, and in such cases the immediate cause of death is not evident.

(*b*) Secondary Chronic Abscess of the Brain.

Secondary chronic abscesses are generally caused by affections of the inner ear. The diagnosis of the presence of chronic abscess of the brain is difficult, inasmuch as only a small proportion of such cases give rise to characteristic symptoms. When the abscess is encapsulated it may remain latent for a long time, so that no disease of the brain is suspected until the terminal period. Even the terminal symptoms present varieties which tend to obscure the diagnosis, these symptoms

sometimes resembling those of diffuse meningitis, and at other times those of thrombosis of a sinus.

Varieties.—The following varieties of chronic abscess secondary to disease of the ear may be distinguished: (1) Chronic abscess with distinct typical course; (2) Chronic abscess with terminal stage alone distinct; (3) Chronic abscess with thrombosis of the lateral sinus; (4) Chronic abscess complicated during the terminal period by meningitis.

(1) In affections of the inner ear abscess may form in the temporo-sphenoidal lobe. In a long-standing case of disease of the internal ear, where rigors and other general inflammatory symptoms are associated with severe pain in the head, vomiting, convulsions, and other cerebral symptoms, the formation of an abscess in the brain may be suspected. These symptoms may pass off, and the patient enjoy apparent health for months, with probably occasional headaches. The terminal stage is announced by intense headache and dizziness soon followed by loss of consciousness and stertorous breathing. Consciousness may be partially restored in a few hours, and the patient then suffers from intense headache and vomiting. After a short time the patient lapses a second time into a semiconscious condition, and convulsions, generally unilateral, supervene. Spasm followed by paralysis of the ocular muscles is not an unfrequent symptom, and when the abscess is so large that it extends to the lenticular nucleus and compresses the internal capsule, or the fibres of the pyramidal tract in the crusta, a certain degree of hemiplegia may be present.

(2) Chronic abscess of the brain is sometimes observed in cases of caries of the petrous bone, in which the terminal symptoms have not been preceded by those indicative of irritation or encephalitis.

(3) Chronic abscess of the brain sometimes precedes, at other times succeeds to thrombosis of the lateral sinus. The chief initial symptoms are, besides those of the ear affection, dizziness, intense headache, and occasionally transitory delirium, followed by somnolence. The patient suffers from frequently repeated rigors if the temperature of the body be raised, and the fever assumes a remittent type. In the further progress of the case the symptoms may pursue either of two directions. The symptoms may be those of progressively increasing pressure upon the brain, ending in coma, or the general symptoms indicative of compression may be associated with those of localized disease, provided the abscess has attained a sufficient size to press upon the internal capsule. General convulsions may occur immediately before death.

(4) Chronic abscess of the brain may be complicated during the terminal period by meningitis, and when the initial stage of the former is latent, the terminal symptoms may be so similar to those of primary

acute meningitis that the two affections cannot be distinguished from one another during life.

c. Encephalitis Secondary to Hemorrhage.

The patient may have made a good recovery from the early symptoms of an apoplectic attack, but several days afterwards there is a secondary elevation of temperature, and the pulse becomes hard and frequent. The patient complains of headache, or he may suffer from slight wandering and confusion of ideas or fall into a somnolent condition. The general are soon followed by local symptoms, consisting usually of the well-known secondary contractures. Some patients may manifest only a slight tremor of the paralyzed limbs; in others the flexors are in a state of contracture; and in a third series of cases these conditions alternate. The temperature of the paralyzed side is often considerably elevated, and anomalies in the secretion of sweat are observed. The somnolence may now increase to a deep sopor which lasts several days, and may pass into a profound or fatal coma. In cases that recover symptoms frequently persist which show that a chronic encephalitis is established. The patient suffers from persistent headache, and frequent attacks of dizziness, while he is subject to congestive attacks each of which may cause new convulsions in the paralyzed limbs. The paralyzed limbs are generally subject to pains of variable character, which may be situated in the joints, bones, skin, or muscles. Secondary encephalitis is also the chief cause of the atrophy of the brain found in many of these patients and which is always associated with profound psychical disturbances.

d. Encephalitis Secondary to Thrombosis and Embolism.

After the formation of softening from occlusion of a vessel in the brain, a febrile condition, attended by a drowsy delirium or somnolence, often continues for some time, and either develops into permanent imbecility, or gives place to partial restoration of the mental faculties. When partial recovery takes place the patient is affected with weakness of memory, irregular and causeless outbursts of temper, and a disposition to shed tears or to laugh without adequate cause. The patient is liable to congestive attacks which cause temporary unconsciousness, and during these new foci of softening may be developed in the brain.

In senile encephalomalacia the symptoms of secondary encephalitis are caused by an increase of the intracranial pressure on the one hand and irritation of the surrounding parts on the other. The symptoms of inflammatory reaction are slight, and when a certain degree of senile

atrophy of the brain had existed previous to the occurrence of the attack, the mental functions become progressively abolished without being preceded by symptoms of active irritation or by those which indicate a gradual compression of the brain.

e. Encephalitis Secondary to Tumor.

The symptoms which may, with probability, be ascribed to encephalitis during the growth of a cerebral tumor, are the occurrence of sudden apoplectiform attacks, the rapid conversion of slight muscular weakness into complete paralysis, partial convulsions followed by paralysis, general convulsions, the sudden appearance of sensory disorders in the paralyzed or partially paralyzed limbs, and the gradual development of coma. When, however, coma is suddenly developed it is more likely to be caused by hemorrhage or sudden œdema than by encephalitis. Every attack of encephalitis is attended by violent headache, but the symptom may be caused by a congestive swelling of the tumor itself.

Morbid Anatomy.—In encephalitis the affected tissue assumes a reddish color, and is studded with a number of capillary extravasations, which are followed by more or less œdema. The affected portion of the brain becomes voluminous and the cut surface rises above the level of the surrounding tissues. An encephalitis of slight intensity and small extent may undergo complete repair. At other times the focus undergoes destructive changes and its contents become converted into a thick emulsion, colored brownish or yellowish by blood pigment. After a time a focus of yellow softening forms which gradually becomes more colorless, and at last may be transformed into a cavity which is filled with a thin milky fluid. Later, the focus manifests a delicate stroma consisting of connective tissue supplied with fine bloodvessels, and the interspaces of which are filled by a thin turbid fluid. In other cases a local encephalitis leads to the production of a firm sclerotic cicatrix. These cicatrices are situated usually near the surface of the brain and only rarely in its interior; they are of a dirty white color, tough and firm, and the tissue surrounding them becomes atrophied so that the affected hemisphere is less than the other. In a large number of cases the encephalitic focus is transformed into a collection of pus, forming either an acute or chronic abscess. An acute abscess has a tendency to spread in every direction, and presents an irregular cavity in the substance of the brain, the walls of which have a rough and shaggy surface. As the pus in the central cavity accumulates, so much pressure is exerted on the surrounding tissue that the circulation is arrested and this leads to further destruction and to advance of the abscess. Chronic abscesses of the brain possess a fibrous capsule which

may attain to a thickness of several millimetres. The internal surface of the limiting membrane is smooth, and has an opaque yellowish-white appearance owing to a continuous layer of cells which are in a state of fatty degeneration. The pus of the abscess is of a greenish color, greasy consistence, odorless, and gives an acid reaction. After a time the abscess enlarges so as to raise considerably the general intracranial pressure, and extensive yellow softening may occur in the surrounding tissues from arrest of their nutrition. Both acute and chronic abscesses may give rise to perforations on the surface of the brain and into the ventricles, extensive œdema or anæmia of the brain from increase of the intracranial pressure, and chronic internal hydrocephalus when the abscess is situated in the cerebellum in such a position that it lessens the cavity of the fourth ventricle or of the Sylvian aqueduct.

Morbid Physiology.—Acute encephalitis gives rise to general symptoms like headache, dizziness, and vomiting, most probably through an interference with the general circulation of the brain. Local irritative symptoms like monospasms and unilateral convulsions form by no means so prominent a feature of acute encephalitis as of acute meningitis, and in the former the symptoms which indicate a general compression of the brain, such as somnolence and coma, appear at an earlier period than in the latter. The clinical course of chronic abscess of the brain is similar to that of an intracranial tumor, and the symptoms which are present in the former depend like those of the latter upon the locality of the lesion.

Treatment.—The physician's advice may be sought to determine questions of cerebral localization and other delicate points of diagnosis which are likely to arise in the progress of abscess of the brain, but the decision with regard to the treatment to be adopted must rest with the surgeon.

The secondary inflammation which is liable to supervene in the course of necrotic softening, cerebral hemorrhage, and intracranial tumors, is the form of encephalitis which is most likely to come under the care of the physician. This form of encephalitis is best treated by complete rest in a darkened room, mild purgation, and cold applied to the head. Active measures like bleeding and blistering are worse than useless. In chronic abscess of the brain the general health of the patient must be attended to, and if definite localizing symptoms be present the pus ought to be removed by operation. Paroxysms of severe headache may sometimes be relieved by chloride of ammonium, while more active symptoms like delirium may be combated by bromide of potassium, either alone or in combination with chloral, and an opiate or cannabis indica may sometimes be found useful.

CHAPTER XVII.

ATROPHY, HYPERTROPHY, AND TUMORS OF THE BRAIN.

I. ATROPHY OF THE BRAIN.

1. Atrophy of the Corpus Callosum.

The corpus callosum may be arrested at any period of its development so that it may be entirely wanting, or union may fail to take place in the middle line, either wholly or partially. Deficiency of the corpus callosum has generally been found associated with idiocy or imbecility, but the mental defects do not present anything characteristic, and some cases have been reported in which there was no mental defect, and consequently the condition cannot be recognized during life.

2. Atrophy of the Cerebellum.

Atrophy of the cerebellum has sometimes been met with as a primary disease from arrest of development, but atrophy of one lateral lobe of the cerebellum occurs most frequently as a secondary result of acquired atrophy of the opposite hemisphere of the brain. The symptoms most frequently observed in the primary disease are a staggering gait, along with a tendency to fall backwards; persistent or temporary disturbances of speech; analgesia and other sensory disorders; epileptiform convulsions; and weakness of mind, or idiocy.

3. Atrophy of the Cerebral Hemispheres.

The cerebral hemispheres are arrested in their development in cases of microcephalic and other forms of idiocy, and partial defects of the brain, either from arrest of development or from destructive lesions, are met with in parencephalus and unilateral atrophy of the brain, already described as giving rise to some forms of the spasmodic paralysis of infancy.

II. HYPERTROPHY OF THE BRAIN.

1. General Hypertrophy of the Brain.

Etiology.—General hypertrophy of the brain usually appears to be congenital. Several of the reported cases were associated with peripheral multiple neuroma, and both of these conditions are frequent accompaniments of idiocy or delayed mental development. A few cases appear to have developed subsequently to an injury to the head, while the disease appears to have been a result of chronic lead poisoning.

Symptoms.—The symptoms almost always appear soon after birth or in early infancy, and consist of epileptiform convulsions, local spasms, attacks of laryngismus stridulus, and tremors. The pulse is usually retarded, but it may occasionally be much accelerated. The affection in children is sometimes associated with premature development, but in other cases there is more or less weakness of mind, amounting even to the highest degree of idiocy. The tongue is often so much increased in size that it protrudes from the mouth. Drowsiness is an occasional but by no means constant symptom. Some of the affected children are liable to fall frequently, being overbalanced by the great weight of the head. Disturbances of the nerves of the general or special senses are comparatively rare. Death results from an attack of convulsions, from cerebral compression with coma, or from an intercurrent disease.

2. Partial Hypertrophy or Heterotopia of Brain Substance.

This condition was first described by Virchow, and has hitherto been principally of interest to the morbid anatomist. Virchow observed in one case an apparently new formation of gyri within the white substance of the posterior lobe, and a hyperplastic malformation of the caudate nucleus, while Simon met with small accessory gyri on the summit of the convolutions. Klob found a mass of white cerebral substance, the size of a bean, hanging from a pedicle between the optic nerves.

These conditions have hitherto been found in epileptics, idiots, or in persons otherwise mentally affected, but their clinical significance is somewhat doubtful. All authors regard these malformations as congenital.

III. INTRACRANIAL TUMORS.

Etiology.—Tumors of the brain arise from similar causes to those which originate tumors in other localities, and these need not be described here.

Symptoms.—Headache is one of the earliest and most striking of the initial symptoms of intracranial growths, and it is present in about two-thirds of all cases. The headache of intracranial tumor consists of an acute lancinating or severe boring pain which is very violent, and may continue for many weeks without intermission, although it is liable to paroxysmal exacerbations of great severity. The pain is aggravated by light, noises, and all movements of the head, and it may occupy the occipital, frontal, or temporal regions, but its seat has no necessary relation to the situation of the tumor. The skull may, however, be found tender to percussion at a point corresponding to the situation of the tumor. The profound headache of cerebral tumor may also be accompanied by neuralgic headache caused by irritation of the fifth nerve in its course along the base of the brain. The pain of trigeminal neuralgia occurs in paroxysms, and it is usually associated with numbness, formication, itching, a feeling as if the part was swollen, or neuroparalytic ophthalmia. When sensation is diminished on the painful side, or when all three branches of the nerve are simultaneously affected, the presence of tumor may be suspected, and the diagnosis is rendered more certain if disorders of other cranial nerves are present.

Another frequent initial symptom of intracranial tumor is *dizziness*, which may, along with paroxysms of violent headache, be the only symptom of a growth which presents itself for many months. *Vomiting* is, however, apt to be associated with headache and dizziness in the early stages of a cerebral growth, and it is often a very distressing symptom.

Sensory disorders are generally ushered in by hyperæsthesia or some other irritative symptoms, which are, after a time, followed by anæsthesia. Wandering pains, formication, tingling, and numbness alternate with one another before there is a distinct diminution of sensation, and they do not entirely cease until complete anæsthesia is established.

The motor disorders consist at first of tremor of one extremity or of one-half of the body, or of cramps which may vary from slight spasmodic twitches of a special group of muscles to persistent tonic, clonic, or choreiform spasms of the muscles of some of the extremities. The clonic spasm is liable to assume the form of unilateral epileptiform con-

vulsions which may or may not be accompanied by unconsciousness. After a longer or shorter time the irritative motor symptoms give place to paralysis, which often creeps on gradually and may not be complete for a comparatively long time. The most usual form of paralysis in cerebral tumor is hemiplegia, which only differs from that of cerebral hemorrhage by the fact that it creeps on gradually in the former, instead of being suddenly produced as in the latter, and that the spastic condition of the limbs is not generally so pronounced in the hemiplegic tumor as in that of hemorrhage.

One or more of the cranial motor nerves may be compressed at the base of the brain or at their point of origin in the crus cerebri, pons, and medulla oblongata. Paralysis may be preceded by spasmodic twitchings of the muscles supplied by these nerves.

Various forms of reeling and staggering may be present when the tumor is situated near the cerebellum or its peduncles, but these disorders belong to the localizing symptoms and will be subsequently described.

The sense of sight is frequently affected in the course of intracranial growths, Calmeil having found amblyopia in two-fifths, and Ladame amaurosis in one-fifth of the cases analyzed by them. The field of vision may be altered in various ways and it is very important for regional diagnosis to examine for the different varieties of hemiopia. On ophthalmoscopic examination the fundus of the eye may present the usual signs of optic neuritis or of the "choked disk," the latter being by far the most important sign of cerebral tumor, inasmuch as it is seldom present except when the intracranial pressure is increased in degree by a solid growth. The pupils may be contracted or unequal, but as the intracranial pressure becomes great by the increasing size of the tumor the pupils are dilated and react feebly to light.

The disorders of the *sense of hearing* consist of rustling noises in the ears, and dulness of hearing, but complete deafness is only occasionally observed. Paralysis of the labyrinthine fibres of the auditory nerve may cause vertigo and motor disorders similar to those observed in Ménière's disease.

The disorders of the *sense of smell* consist of subjective sensations or loss of smell in one or both nostrils, but this sense is not often affected.

The disorders of the *sense of taste* consist also of subjective sensations or loss of taste, but Ladame found that this function was only mentioned as having been affected seven times out of all the cases he had collected.

The *organic functions* are always impaired in the course of intracranial growths. The intense headache from which the patient suffers

occasions a continued wakefulness which reacts upon the general health, and when vomiting recurs frequently the general nutrition is greatly impaired. The patient often suffers from constipation, but this condition may alternate with diarrhœa. Polyphagia is an occasional symptom of cerebral tumor, but it does not prevent the progressive emaciation. This symptom is occasionally accompanied by diabetes mellitus. Polyuria with or without saccharine urine is not an infrequent symptom of cerebral tumor. In the early stages of cerebral tumor the action of the heart may be irregular and slow from irritation of the vagus, but towards the end the pulse becomes extremely frequent from paralysis of that nerve. Respiration is not often affected, but its rhythm may be quickened by irritation and rendered slower by pressure of the brain.

Fever, although not a usual symptom of cerebral tumor, may be present during complicating attacks of encephalitis.

The psychical disturbances which are present in greater or less degree in about one-half the cases, consist at first of mental excitement and emotional displays like those of hysteria, ideas of grandeur, hallucinations, delusions, and outbursts of passion which may amount to maniacal fury. The irritative symptoms, however, give place after a time to those of depression, which consist of drowsiness, apathy, loss of speech, and imbecility. The terminal symptoms consist of extreme emaciation, widely spread anæsthesia, blindness and impairment or loss of one or more of the other special senses, motor paralysis which often implicates all the extremities, imbecility, and deep and enduring coma.

Morbid Anatomy.—The morbid anatomy of the atrophy and hypertrophy of the brain is a wide subject, which cannot be further discussed here. With regard to intracranial tumors, it will suffice to remind the reader that the brain is surrounded by unyielding osseous walls and that consequently all growths which gradually encroach upon the space occupied by the brain must give rise to nearly the same kind of symptoms. The tumors themselves being so variable in their nature, possess no affinity with each other and the symptoms to which they give rise from their situation afford the only grounds for grouping them together. Intracranial tumors may be divided into, *a*, new formations; *b*, vascular tumors; and *c*, parasites.

Morbid Physiology.—The tumor at first may give rise to few or no symptoms, but as it gradually increases in volume the whole brain is subjected to pressure, the cerebro-spinal fluid is first displaced, the blood is then squeezed out and the whole brain becomes anæmic; the functions of the brain become gradually diminished and ultimately lost, and the patient dies comatose. But in addition to this general pressure the tissues surrounding the growth are subjected to a special pressure

which soon leads to their destruction. Destruction of tissue is at other times caused by ischæmic softening from the compression of an artery, while loss of function may be caused by effusion of serum from compression of veins. But although the direct tendency of a tumor is to destroy the surrounding tissue, its indirect effect is often irritation, and consequently the depression symptoms are often preceded or accompanied by phenomena of irritation. Still greater complications in the symptoms are caused by the fact that on the one hand irritation of a higher centre may produce an inhibitory action on a lower centre, while on the other hand destruction of a higher centre may leave the functional activity of a lower centre more unrestrained. The symptoms which are caused by the localization of the tumor do not differ essentially from those caused by other focal diseases, and these will be afterwards considered. The intensity of the symptoms depends to some extent upon idiosyncrasies of the patient, the locality of the lesion, and in still greater degree upon the rapidity of the growth of the tumor, slow growing tumors often attaining great size before giving rise to any distinctive symptoms, and fast growing tumors causing very active symptoms at an early period.

Treatment.—In the earlier stages of cerebral tumors the symptoms of irritation and local congestion predominate, and these must be treated by cold to the head, purgatives, the use of flying blisters, and bromide of potassium either alone or combined with chloral hydrate. The intense headache of cerebral tumor must be treated by the application of cold to the head, and subcutaneous injection of morphia. Chloride of ammonium in half drachm doses may occasionally be found useful. Epileptiform convulsions must be treated by large doses of bromide of potassium. With the view of promoting the absorption of the growth iodide of potassium has been given in large doses, and as much as from half a drachm to a drachm may be given three times a day. In progressive growths like cancer, sarcoma, and glioma this treatment cannot of course lead to any permanent benefit, but it may be useful in aneurism, and is by far the most hopeful treatment in parasites. If syphilis is suspected, an energetic antisyphilitic treatment by means of mercury and iodide of potassium must be undertaken. It is possible that hereafter surgeons will be able to undertake the removal of a considerable number of cerebral tumors; even already a few successful cases have been reported.

CHAPTER XVIII.

APHASIA.

APHASIA comprises certain disorders of the faculty of language, but inasmuch as every form of disorder is not to be included under the term it is necessary to subject the faculty itself to a preliminary analysis in order to determine which belong and which do not belong to this group. Language, taken in its evident sense, consists of the various means by which animals indicate mental states to one another, this faculty comprising a power of expressing one's own states and of appreciating the states of others. Mental states may be divided into feelings, cognitions, and volitions. In one sense language may be said very often, if not always, to indicate volitions, but inasmuch as voluntary actions are always determined by motives, or, in other words, by the feelings and cognitions, the language of volition merges into that of the other two mental states. Language may, therefore, be divided into that of the feelings, or *emotional language*, and that of the cognitions, or *intellectual language*, or *speech*. The distinction between the language of the emotions and speech is by no means clear and trenchant. When a man delivers an oration, for instance, only a small part of his utterances and gestures are to be regarded as belonging to intellectual language. All the variations of tone, the melodious voice, the graces of attitude and gesture, the charm of elegant and rhythmical language, and the thousand other ways by which a great orator knows how to sway and influence his audience, belong to emotional and not to intellectual language. The pleasure we derive from looking at a clearly printed volume, and especially from looking at an illuminated text, or at a picture rather than at a diagram, shows that there are different means whereby the feelings may be engaged, and even such methods as accents, italics, and notes of exclamation, by means of which inflection, emphasis, and wonder are indicated, the rhythm of metrical language, and the diction and imagery of poetry, all belong to emotional and not to intellectual language. The language of emotional and of intellectual gesture are also by no means readily separated. The gestures of those who retain the full use of spoken and written language are in great part indicative of the feelings, but that gestures can be made subservient to intellectual expression is shown by the importance it assumes in the

training of the deaf and dumb. But although it is not always easy to discriminate between emotional and intellectual language, we must repeat that it is disorders of speech or intellectual language alone that are comprised under the term aphasia.

But we shall now see that aphasia does not include every form of disorder of intellectual language. Speech is the instrument of the social state, and that it may be the means of communication between animals it must possess to each a *subjective* and an *objective* value, or must fulfil an *impressive* and an *expressive* function. Each individual of a social community, in order to become an effective member, must be able to comprehend the mental state of others from watching their gestures and listening to their various vocalizations, and must also be able by his gestures and vocalizations to render his own mental state intelligible to the others. The *subjective* or *impressive* function of speech may be divided into *receptive* and *regulative* functions.

The *receptive* department is represented structurally by the various peripheral sense-organs and the centripetal fibres which conduct impression upon these sense-organs upwards to the cortex of the brain. Complete loss of speech from disease of the receptive apparatus is unknown. The vocal speech of a person born blind is almost entirely unaffected either in its subjective or objective aspects, while the patient may, by the device of raised letters, be taught to understand written language. The deaf-mute is taught both to understand and to give expression to a complicated speech by gestures, and in recent times such patients have been taught not only to use their vocal organs for expression in speech, but also to understand the vocal speech of others by closely observing the movements of the muscles of articulation.

The structural counterpart of the *regulative* function consists of that part of the cortex of the brain in which the centripetal impulses are reduced to such order as is necessary to render them the correlatives of the cognitions. Now the cognitions express the relations between our feelings, and every cognition must be expressed in a proposition. The mode of expression may not always assume a distinct propositional form, but it must at least possess a propositional value if it convey distinct knowledge. If I pronounce the word "orange" in the hearing of another, it may or may not convey to him distinct information, but if any information be imparted the word must convey to the listener the idea that the object named "orange" belongs to a class of objects already known to him under that name, and the word in this sense possesses the value of a distinct proposition. If the listener has never had any experience with the object named "orange," it is clear that the utterance of the name will convey no meaning, but if he has

had experience of fruits and colors, distinct information may be conveyed to him with regard to the object by saying "an orange is a yellow fruit." The listener will be able to associate the general properties of fruit and a distinct color with the word in future, but the information has been imparted by means of a formal proposition. The activity of the regulative cortical centres of speech has for its functional correlative the arrangement of the presentative and representative cognitions into the form of distinct mental propositions.

The *objective* or *expressive* function of speech may be divided into *emissive* and *executive* departments.

The *emissive* department is represented structurally by that organization in the cortex of the brain in which the regulative impulses are finally coördinate before being conducted to the executive department.

The *executive* department is represented structurally by groups of nerve cells in the central gray tube, and by the nerves and muscles concerned in vocalization, articulation, the manual operations of writing, and various gestures. Complete loss of speech from disease of the executive apparatus is probably unknown. The patient, for instance, may lose his voice in different diseases of the larynx, but he can still articulate; he may lose both voice and power of articulation in bulbar paralysis, but he is generally able to make his wants known in writing, and when unable to write from want of a previous education, he can make his ordinary requirements known by gesture.

Our further remarks must be limited to the derangements of speech caused by disease of the cortex of the brain, and which are grouped together under the name of aphasia. These disorders consist of affections of the regulative department of the impressive function, and of the emissive department of the expressive function; the former of these being named *sensory* and the latter *motor* aphasia. In addition to these two chief varieties there are cases in which the ingoing and outgoing functions of speech are both affected, and inasmuch as the lesion in such cases is situated between the sensory and motor cortical centres Wernecke proposes to group them under the name of *commissural* aphasia, but here we shall treat of them under the name of *mixed sensory and motor* aphasia. As motor aphasia is the simplest form of the affection, we shall deal with it first.

MOTOR APHASIA.

In cases of motor aphasia the patient is unable to communicate his thoughts by words (motor aphemia), or by writing (motor agraphia), while there is loss or impairment of his intellectual pantomime (motor

amimia). The patient can often utter words, but they do not, if the motor aphasia is complete, possess any intellectual value; in the language of Dr. Hughlings-Jackson, the patient is *speechless* but not *wordless*. The words which the patient can utter continue, as a rule, the same for the same patient—"recurring utterances," or the patient may under excitement swear, or even utter a phrase appropriate to the surrounding circumstances, such as "good-bye" when a friend is leaving. It will be readily seen that "recurring utterances" like "yes" and "no," which are repeated on all occasions whether appropriate or not, do not possess any intellectual value, while of the occasional utterances swearing is a purely emotional expression, and even the phrase "good-bye", must be regarded as expressing a state of mental regret rather than a purely intellectual appreciation of the surrounding conditions. In some cases, in addition to the usual recurring utterances of "yes" and "no," the patient repeats such phrases as "come on to me," or "I want protection." The man whose recurring utterance was "come on to me" was a railway signalman, and had been taken ill on the rails in front of his box, while the man who could only say "I want protection," had his left cerebral hemisphere injured in a brawl. Dr. Hughlings-Jackson makes the very probable supposition that in these cases the recurring utterances constituted the last words spoken, or which were in a state of mental preparation for utterance when the damage occurred to the brain. It is not improbable that words uttered or about to be uttered during a period of great excitement may leave permanent traces on the organization of the brain which will render them liable to be subsequently uttered as interjectional phrases during emotional states. That all these words and phrases must be regarded as expressive of emotional rather than intellectual states is shown by the fact that the patient is frequently unable to repeat his favorite oath or his formula of leave-taking, or perhaps "yes" or "no," when asked to do so.

The patient is not always completely speechless; he may, for instance, retain the full use of the words "yes" and "no," and even when he uses "no" to express assent as well as dissent he may be able, by the aid of pantomime, to make it known in which sense he intends the word to be understood. All forms of intellectual expression are not affected to the same degree. A patient may have lost the use of vocal speech completely and yet retain to a greater or less extent the power of writing, and conversely a patient may retain or reacquire the full use of vocal speech while the power of writing is permanently lost; in other words, motor aphemia may be present without agraphia, or agraphia without aphemia. The faculty of intellectual pantomime is also often reacquired long before that of vocal speech in cases of motor aphasia;

but none is known, so far as I am aware, in which the former was permanently lost while the latter was regained.

In motor aphasia the patient understands all that is said to him and remembers what is read to him or what he reads himself; his vocal organs act normally; the muscles of mastication and deglutition are not paralyzed, and he can sing, laugh, smile, and frown as usual. He will point to objects named and recognize drawings of them, provided they were known to him before his illness. He is able to play at cards and other games, and recognizes handwriting. He can copy writing, provided his hand is not paralyzed, but in aggravated cases the patient copies printed words in printed characters and written words in written characters. If the right hand is paralyzed, the patient on attempting to use his left hand writes from right to left instead of from left to right as usual, the result being what is known as "mirror-writing," so named because it looks like ordinary writing only on being reflected from a mirror.

SENSORY APHASIA (AMNESIC APHASIA).

The purest examples of sensory aphasia consist of the affections known as *word-blindness* and *word-deafness*.

In *word-blindness* the patient is totally unable to comprehend written or printed language, but he is at the same time not blind, inasmuch as he avoids obstacles in walking, and takes notice of everything that passes around him. His sight is, however, sometimes defective, right-sided bilateral homonymous hemianopia being often if not always present, but this defect does not prevent the patient from reading fairly well when the lesion is situated in the optic tract. The patient can write spontaneously or to dictation, but he cannot copy printed or written words and is quite unable to read what he has himself just written. The patient is not only totally unable to read aloud, but singularly enough he may also be quite unconscious of his disability. A patient of mine on being presented with a letter which I had just received making inquiries about a patient attending as an out-patient at the infirmary, read without much hesitation: "Manchester Royal Infirmary. Dear Sir: You are requested to bring this paper with you the next time you come to the Infirmary." Not a single word of this corresponded with the note which he pretended to read. His wife told me that at home he pretended to read the newspaper aloud, but she added "it's all stuff out of his own head." One night he stopped in the midst of a paragraph in which he was apparently interested and asked

his wife to finish the remainder of it. She read the paragraph aloud as it was printed, and in great surprise he asked, "Is that what it says?" and on being assured that such was the case he added, "Well! I must be an idiot."

In *word-deafness* the patient is unable to understand the vocal speech of others. The subjects of this disorder hear as well as ever a knocking at the door or the tick of a watch, but are totally unable to comprehend the language of others, or, in other words, spoken words fail to call up in the mind of the patient the images corresponding to the persons, things, events, and relations indicated by the words. When word-deafness is complete, the subject of it fails to do anything requested of him. But even in such cases the patient is quick at interpreting signs, and if the physician ask him to put out his tongue he may do so not because he understands the spoken request, but because he notices that the eyes of the observer are directed to his mouth. In most cases, however, the disorder is partial, and the patient is then able to comprehend familiar words and phrases, and may put his tongue out, close the door, and perform a number of other simple and common actions when asked. A very good test of word-deafness is the one proposed by Dr. Hughlings-Jackson, namely: Ask the patient some absurd question, such as "Are you a hundred years of age?" If the patient comprehends the question it will excite astonishment and amusement. A patient of mine when asked the question says readily, "No, I don't think I am so old as that," and evidently the question does not cause him any surprise, but when 100 is presented to him in written figures he then bursts into laughter and says, "Pooh! Nothing like it."

Word-deafness is almost always associated with a certain degree of word-blindness. Speech is organized for the first time in connection with the sense of hearing, and when some time afterwards written speech comes to be organized, the visible characters are understood through the medium of the already organized audible characters. The connection between the written and spoken words varies greatly in different cases. In uneducated persons written speech is only understood by being read aloud or translated into spoken speech, and in such cases word-deafness would entail complete word-blindness. Educated persons, however, understand the meaning of written words directly, so that injury of the auditory centre would not damage much the understanding of written speech. A patient of mine, who has received a tolerable education, is suffering from word-deafness, and he can read aloud without committing many mistakes, while he apparently understands what he reads, but he is unable to name a single object presented to him. The sight of the object does not call up its name, and yet there is every ground for be-

lieving that the lesion is situated in the auditory cortical centre, and that the visual centre is free from disease. In this case the patient is totally unable to name his own fingers, but when the word is written down he immediately identifies it as indicating the finger of his own hand. Another patient with hemiopia and word-blindness, fails to recognize the written word, but readily names his finger or any other object when presented to him. The disorder of the expressive faculty of speech in word-deafness is quite as interesting as the disorders of the impressive faculty. When the patient speaks he misplaces words, as calling the "window" a "fender," or he uses a word kindred in its meaning with the one intended, as "worm powder" for "cough medicine," or in its sound, as "parasol" for "castor oil." This condition, which has been named by Kussmaul *paraphasia*, may be present in word-blindness as well as in word-deafness, and possibly also in the incomplete form of motor aphasia. A condition which may be named *paragraphia* is also often present.

A patient of mine on being asked to write his own name, "William Abson," wrote down "Wuagageag Abreaghrer," and on being asked to copy a passage beginning "with deep feeling," he wrote with great care and deliberation, and in a good hand, "weeth deap fineearer." As the patient wrote each letter he named one aloud, but the written and spoken letter never corresponded with one another. When asked to read a passage beginning with "Oh, those dreams of my childhood and my youth," he read "On through depth of my shouded and my youth." This condition has been called *paralesia*.

A profounder degree of disorder than paraphasia is found in those cases just mentioned, in which the presentation of persons and things to the patient fails to call up their names. The patient already mentioned as being unable to name his own finger, has only the use of three or four concrete nouns, which he employs on all occasions, yet he has a large vocabulary of words expressive of events, dates, and relations, as well as the use of some abstract nouns. On being pressed for the name of his thumb, he called it "the public house," the index finger "the first one," and on being pressed still further for a name, "the first halfpenny;" the middle finger, "the second halfpenny;" the ring finger, "threepence;" and the little finger, "the fourth halfpenny." Asked the name of a watch, he called it "a glass of beer," and to indicate his appreciation of the difference between a gold and silver watch presented to him, he pointed to the first as being "the best beer." The only names of things in his possession are public house, glass of beer, and the names of the coins in ordinary circulation, but which he seldom uses correctly. He can count up to any number

when he begins from one, but it is only on rare occasions that he is able to break in on the series and begin with, say six. This patient is almost completely word-deaf, but will show his appreciation of most requests and statements made in writing. A still more aggravated disorder of the expressive faculty of speech is found in those cases in which the vocal speech of the patient is an unintelligible jargon. Such patients fail to comprehend either written or spoken language, and it is probable that the sensory aphasia is also accompanied by a motor aphasia. A patient of mine takes a book in his hand and begins to read aloud with much apparent relish, and uses variations of accent, inflection, and pauses to give emphasis to his reading, but his vocalizations are quite unintelligible, and consist of such sounds as "pluf, plon, lep," etc. The intervals between his pauses seem to depend upon his breathing capacity, and to have no relation to the length of the sentences on the printed page before him. When, however, the page is presented to him upside down he immediately turns it the right side up, and with an expression of great impatience.

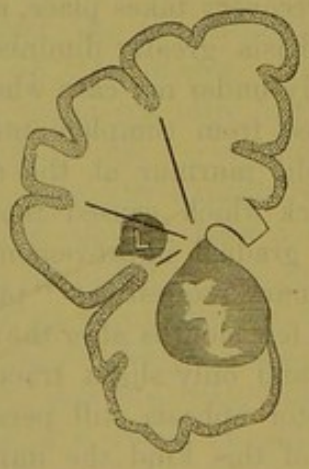
MIXED MOTOR AND SENSORY APHASIA.

In cases of right-sided hemiplegia with aphasia the patient is frequently unable either to express a single word or to comprehend any question addressed to him in written or spoken language. There is at first total motor aphasia and total sensory aphasia. In cases of this kind, however, gradual recovery takes place, and after a time either the motor or the sensory aphasia greatly diminishes or wholly disappears. A young boy was recently under my care who, after an attack of right-sided hemiplegia, suffered from complete motor and sensory aphasia. The presence of a systolic murmur at the apex of the heart showed that the apoplectic attack which caused the paralysis was of embolic origin. After a time a gradual improvement took place; the patient began to obey such ordinary requests as "put out your tongue," and "close your eyes," and a few months after the attack the sensory aphasia completely disappeared, and only slight traces of the hemiplegia could be detected, but the motor aphasia still persists six months after the attack. In some cases of this kind the improvement appears to take place on the motor side. Those aggravated cases of word-deafness and word-blindness in which the speech is mere jargon are frequently if not always accompanied by hemiplegia, and in such cases the motor as well as the sensory aphasia is at first more or less complete, and even when partial recovery takes place it is probable that these cases

are to be recognized as a mixed motor and sensory, rather than a pure sensory aphasia.

Morbid Anatomy and Physiology.—The faculty of language was placed by Gull in the supra-orbital lobes of the brain, but the first serious attempt to determine the localization of this faculty by means of morbid anatomy was made by Bouillaud, who, in 1825, came to the conclusion that the faculty of speech was situated in the anterior cerebral lobes. In 1836, Marc Dax advanced the opinion that the organ of language was situated in the left hemisphere of the brain near to the Island of Riel, but this opinion was neglected and almost forgotten until 1861, when Broca localized the faculty of language in the third left frontal convolution. In the subsequent two years Broca collected seventeen cases of aphasia with post-mortem examinations, and of these the lesion was situated sixteen times in the posterior part of the third left frontal convolution and once in the temporal lobe and Island of Riel. Soon afterwards cases were published by Parrot, Fernet, and Charcot, in which a destructive lesion of the posterior part of the third right frontal convolution had not given rise to any disorder of speech, these cases affording the negative side of the proof that the faculty of language is organized in the left hemisphere. It was suggested by Bouillaud and Broca that the faculty of speech is organized in the same hemisphere of the brain as the mechanism which regulates the special movements of the hand, and in proof of this supposition it has been found that aphasia

FIG. 176.



does coexist with left-sided hemiplegia in left-handed people, several cases of this kind being now recorded by different observers. The lesion in aphasia may consist of a growth, of a depressed bone, or of a hemorrhage, but occlusion of the Sylvian artery or of one of its branches is by far the most frequent cause of aphasia.

Lesions of the centrum semiovale which damage the fibres of the corpus callosum which connect the third frontal convolutions of the two hemispheres, and the fibres which connect the third left frontal convolution with the internal capsule, occasion an aphasia which is as permanent as that caused by destruction of Broca's convolution itself (Fig. 176, L). In sensory aphasia the lesion is localized in the area of distribution of the parieto-sphenoidal and sphenoidal branches of the Sylvian artery (Fig. 177, 4 and 5). In word-deafness the lesion is situated in the first

FIG. 177.

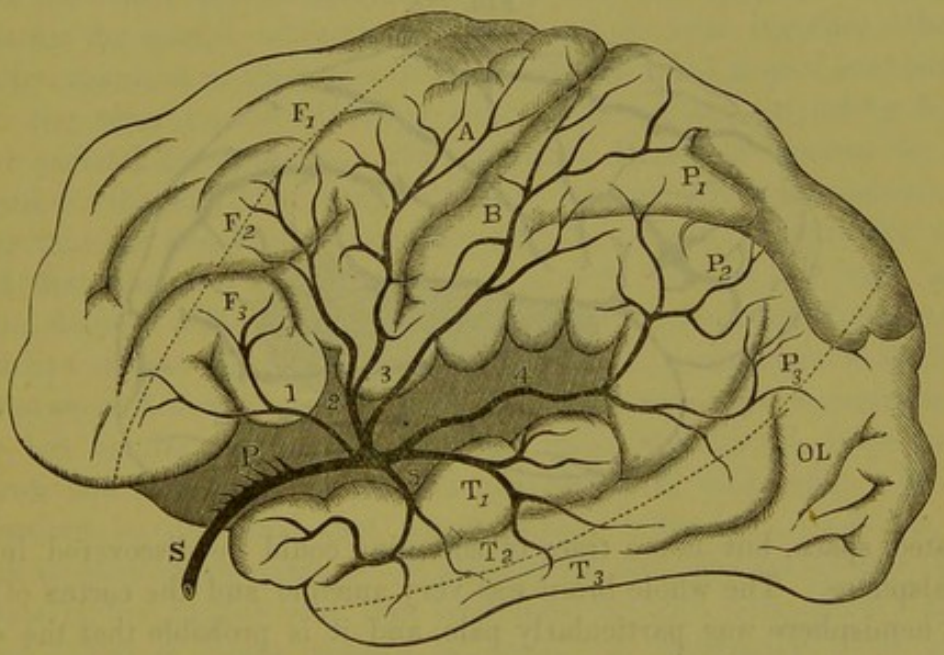


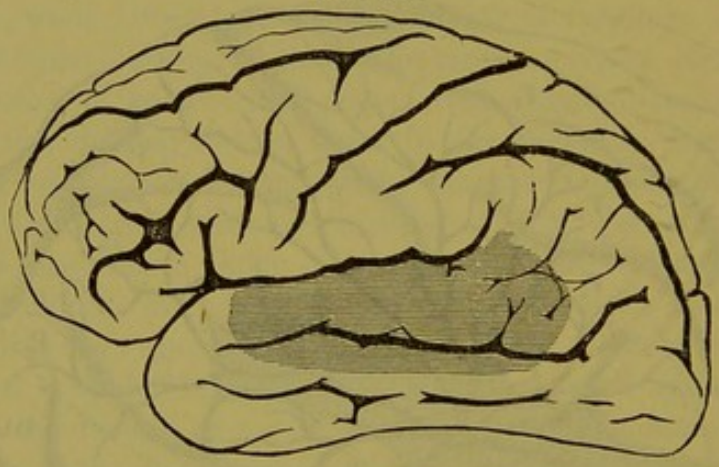
DIAGRAM SHOWING THE AREA OF DISTRIBUTION OF THE MIDDLE CEREBRAL ARTERY.

S, Sylvian or middle cerebral artery; P, Perforating branches; 1, Inferior frontal branch; 2, Ascending frontal branch; 3, Ascending parietal branch; 4 and 5, Parieto-sphenoidal and sphenoidal branches; A, Ascending frontal convolution; B, Ascending parietal convolution; F_1, F_2, F_3, First, second, and third frontal convolutions; P_1, P_2, P_3, First, second, and third parietal convolutions; T_1, T_2, T_3, First, second, and third temporo-sphenoidal convolutions; OL, Occipital lobe.

and second temporo-sphenoidal convolutions (the acoustic centre), and in word-blindness in the angular gyrus and adjacent parts of the occipital lobe (the visual centre). The shaded portion in Fig. 178 shows the position of the lesion in a case of word-deafness reported by Wernicke. The lesion may also be situated in the white substance subjacent to these convolutions. In mixed sensory and motor aphasia the Sylvian artery is occluded immediately outside the nutritive arteries which pass into the anterior perforated space, and consequently the circulation is arrested through all the cortical branches of the vessel. The result of occlusion of the vessel at this point is that hemiplegia is associated with aphasia, and the sensory as well as the motor centres are partially

damaged. It must, however, be remembered that the terminal cortical branches of the Sylvian artery anastomose to some extent with the terminal branches of the anterior and posterior cerebral arteries, and occlusion of the vessel is not always followed by extensive softening, especially in young people. A young girl came under my observation who was suffering from mitral disease, and who died a fortnight after an attack of right-sided hemiplegia with complete motor and sensory aphasia. At the autopsy an embolus was discovered in the left Sylvian artery immediately outside the nutritive arteries to the anterior per-

FIG. 178.



forated space, but not a trace of softening could be discovered in the hemisphere. The whole brain was very anæmic and the cortex of the left hemisphere was particularly pale, and it is probable that the cells had undergone fatty degeneration, but there was no softening, and it is possible that had the patient lived the cortex of the central convolutions would have completely recovered. In older people, however, softening of some portion of the cortex always takes place after occlusion of the Sylvian artery. The softening may sometimes take place in the area of the posterior, and more frequently in the area of the anterior branches, while at times the intervening portion of the cortex is more or less disorganized, and thus it is that a disorder of speech which at first is a complete motor and sensory aphasia may recover, so that it becomes a simple motor or a simple sensory aphasia, according to the locality which is permanently softened, or may remain permanently a combined motor and sensory aphasia in case a wide area of the cortex undergoes softening.

Motor aphemia is caused by destruction of the posterior part of the third left frontal convolution for the same reason that paralysis of the right hand is caused by destruction of the ascending frontal and parietal

convolutions. It may be objected that in motor aphemia the muscles of articulation are not paralyzed, inasmuch as there is no sign of a local paralysis of the tongue or lips. But notwithstanding that the general movements of the tongue and lips are unaffected, there is a paralysis of the special combination of muscular movements which are required to produce articulate sounds, and in motor agraphia there is a paralysis of the particular combination of movements by which writing is effected, although the general movements of the hand may be unimpaired. The reason that motor aphemia and agraphia are so frequently associated is that the centre for the special movements of the tongue and lips, and that for the special movements of the hand lie near together; the one being organized in the posterior portion of the third frontal convolution, and the other in the adjoining inferior part of the ascending frontal and parietal convolutions. But although these two centres lie near together, the fact that they still form separate centres explains why it is that the one kind of disorder may be present without the other, and that the two are only seldom present in any one case in an equal degree. The reason that the general movements of the tongue and lips are not paralyzed in lesion of the left hemisphere is that they are organized in both hemispheres, and that when the organization in the left hemisphere is destroyed the right hemisphere takes up the action and maintains the general movements of both sides. It may, therefore, be presumed that the recurring utterances which are met with in cases of complete motor aphasia are also like the general movements of the tongue and lips, organized in both hemispheres of the brain. But if motor aphasia is caused by destruction of a motor centre in the cortex of the left hemisphere of the brain, why is it that aphasia is not caused by destruction of the conducting path which connects this centre with the bulbar nuclei? The answer is, that injury of the conducting path in the knee of the internal capsule does cause an aphasia, although it does not remain permanent. A right-sided hemiplegia caused by hemorrhage into the lenticular nucleus is accompanied by a motor aphasia at first, but the disorder of speech passes off in a few weeks or months at most. The explanation which Dr. Broadbent has given of this fact is generally accepted. He believes that when the direct conducting path which connects the third left frontal convolution with the bulbar nuclei is interrupted, a new path is gradually opened first from the third left to the corresponding third right frontal convolution, through the corpus callosum, then through the pyramidal tract of the right hemisphere to the bulbar nuclei. But when the right pyramidal tract is injured subsequently to an injury of the left tract, there not only is then more or less aphasia, but the general move-

ments of the lips and tongue become paralyzed, and the patient presents all the phenomena of a bulbar paralysis. But if motor aphasia is a paralysis of certain special movements, we shall now see that sensory aphasia is an anæsthesia to certain properties of matter, while the appreciation of the general properties is retained. The condition of patients who are word-deaf and word-blind is comparable to the mental state to which Goltz reduced dogs by washing out large portions of the cortex of the brain. The animal operated upon recovers to a greater or less extent from the paralysis which follows immediately upon the injury, but it is found that the psychical capacity is very seriously impaired. He is not blind, because in walking about the laboratory he avoids obstacles, but he fails to recognize his usual food as such, and the vessel in which it is contained is avoided as an obstacle.

The dog still retains the knowledge which connects the falling of a shadow upon the eye with the near proximity of a resisting body, which is the most general of all knowledge, but he has lost the knowledge which is only a little less general than it, that a particular substance possesses the specific properties of taste and smell, which constitutes its food to be eaten and not an obstacle to be avoided. On the side of the auditory sense the dog is equally deficient; he is, indeed, like the word-deaf: he still hears noises, but fails to respond to his own name. To make the similarity between the dogs operated upon by Goltz and the word-deaf and word-blind more complete, a post-mortem examination of the dog exhibited at the International Medical Congress at London, showed that the cortex was chiefly injured in the occipital and temporo-sphenoidal lobes, or in the sensory areas of the cortex. A more or less similar mental condition to that of the dog operated upon, so far as regards sight, was observed by Fürstner in cases of general paralysis of the insane, in which the occipital lobes were specially involved in the disease. The patient could still see large objects and avoid them as obstacles, but was unable to count correctly small objects, and lost all knowledge of the special properties of matter. A coin, for instance, he recognized as made of metal, and as having a certain form and size, but he had no knowledge of the special properties which gave it value as a money tender. The word-deaf and the word-blind are not reduced to so low a condition of intelligence as this, but their disability is the same in its essential features; there is a loss of the more special and least organized knowledge, and a preservation of the more general and best organized knowledge. To elaborate this idea would require much more time and space than I can afford, but it will suffice to say that, speaking generally, the best organized knowledge is that which is most frequently repeated in the experience of any individual, and of the race. As I

look around me, I see a number of objects which I recognize as books, and one would think that the association between the sight of a book and its name is well organized in me, and so no doubt it is. But of each book I can say that its color, its size, its weight, and all its other innumerable properties, is the same as the color, size, weight, and other properties which I know in other objects. Wherever I turn, and whatever object I look at, I cannot get rid of the fundamental relations of sameness and difference, and consequently the name of these simple relations must be much more profoundly organized than the names of things.

In dissolution of the impressive faculty of speech, therefore, the names of things disappear first and of the simple relations last. Now of all words the individual names are the least organized, and it is within the experience of every one how very easy it is to forget the names of one's friends, while such simple predicates as *is*, *may*, and *shall*, or *being*, *go*, *run*, and *know;* adverbs like *here*, *there*, *now*, *before*, and *after;* adjectives like *good* and *bad*, and *hard* and *soft*, are undoubtedly much more frequently repeated in speech than the names of persons and things, and the former are, therefore, more likely to be preserved than the latter in cases of dissolution of the impressive faculty of speech. In those cases of aphasia in which the names of persons and things are almost entirely lost, let it not be thought that the other parts of speech have suffered no damage. The patient whose case I have already mentioned, who named every object presented to him, including his own fingers, "public house," "glass of beer," or "halfpenny," was accustomed to describe his previous occupation in the following terms: "When gentlemen got into trouble I brought an action against them and they were all right then." He was a messenger in the bankruptcy court and had to serve processes on defaulting debtors. He must, therefore, have been very familiar with such words as "to liquidate," and with such phrases as "to compound with one's creditors," but he now never uses such complex words and does not give any evidence that he understands them when uttered in his presence. It must, therefore, be presumed that in him all other parts of speech as well as the nouns have suffered damage, inasmuch as he is only able to make use of those words and phrases which express the simplest relations, and qualities, and the events most frequently repeated in his experience. In dissolution of the nervous mechanism of the impressive faculty of speech the names of persons and things disappear first because they are the least organized. The facility with which proper nouns are lost has been experienced by most of us in the readiness with which we forget the names of our most intimate friends when the brain is fatigued by

overwork or loss of sleep. Most educated men have also experienced the strong tendency which the technical language of a newly acquired science has to fade from the memory. But although the loss of the nouns is the most marked feature of sensory aphasia it must be admitted that this disability is accompanied by the loss of all words which express complex relations and qualities, and the words which remain to the last are those which express the relations and qualities which have been most frequently repeated in the experience of the race, and the events which have been most frequently repeated in the experience of the individual. It must, however, be remembered that frequency of repetition is only one factor in producing degrees of organization in speech. The strength of the impression which formed the association between the word and the thing signified by it, as well as various other circumstances, must be taken into account. "Burnt children dread the fire," and doubtless the word fire becomes deeply fixed in the organization from a very limited experience of its action. But as it is impossible to pursue the subject further in this place, the student must be left to think it out for himself. That word-deafness, and word-blindness are examples of partial anæsthesia is well illustrated by recorded cases in which a person, while suffering from one of these affections, suddenly becomes completely deaf or blind without having any local disease in the ear or eye, and at the autopsy a spot of softening is found symmetrically placed in each hemisphere of the brain on a level with the first and second temporo-sphenoidal convolutions, or with the angular gyri.

CHAPTER XIX.

DISEASES OF THE MEMBRANES OF THE BRAIN.

I. DISEASES OF THE DURA MATER.

Inflammation of the dura mater may be divided into (I.) external, and (II.) internal pachymeningitis.

(I.) External Pachymeningitis.

Etiology.—The chief causes of external pachymeningitis are injuries which detach the dura mater from the skull and give rise to hemorrhage between the membrane and bones; perforating injuries of the skull; and extension of inflammation from neighboring tissues, as from the temporal bone in caries of the petrous portion. The external layer of the dura mater is subject to chronic thickening in old age.

Symptoms.—In traumatic external pachymeningitis the patient may recover from the immediate effects of the injury, but at the end of two or three weeks he suffers from pain in the head and is feverish, and should there be an external wound it assumes an unhealthy aspect. If the inflammation spread further the headache increases in intensity, and the patient suffers from vertigo, nausea, and vomiting, while monospasms, or unilateral convulsions may occur if the motor area of the cortex is affected. As the inflammatory process extends inwards through the pia mater to the cortex the previously convulsed limbs become paralyzed, delirium supervenes, and the patient soon dies in coma.

When an abscess forms between the bones of the skull and the dura mater the symptoms may be those of gradual cerebral compression. In the pachymeningitis of old age the symptoms consist of a persistent dull headache, and in many cases the presence of the disease is only discovered post mortem.

(II.) Internal Hemorrhagic Pachymeningitis (Hæmatoma of the Dura Mater).

Etiology.—Hæmatoma of the cerebral dura mater is generally met with in old age, and is either caused by internal injury, or results from

cachectic diseases like scorbutus, chronic alcoholism, general paralysis, and senile atrophy of the brain.

Symptoms.—The symptoms are variable, but the most constant is a dull headache, which is generally diffused, but may be more pronounced on one side than the other. The *motor disturbances* consist of unilateral or bilateral muscular twitchings, which are followed by a persistent rigidity. In other cases paresis, first of the one and then of the other extremity of the same side occurs, which gradually increases until a decided hemiplegia is established. In some cases the paralysis extends to the other side, showing either that the inflammatory action has spread to the other side or that a fresh hemorrhage has occurred, forming a new focus of disease. The gait is often uncertain, and the patient experiences difficulty in writing and speaking, while a conjugate deviation of the eyeballs towards the side of the lesion is not infrequently present. Patients sometimes complain of formication and numbness of the paralyzed side, but impairment of sensation in the absence of paralysis never occurs.

Psychical disturbances are sometimes present as part of the primary disease, as when hæmatoma occurs in general paralysis, but at other times they result directly from the hæmatoma. These disorders consist at first of great mental irritability, abnormal sensitiveness to light, and ringing in the ears, but as the disease advances the signs of increased irritability give place to those of a gradual compression. In other cases drowsiness is the chief psychical symptom observed, and when it is present in old people in conjunction with chronic headache and contracted or dilated pupils, hæmatoma should be suspected. The pupils are contracted and unirritable to light during the irritative stage of the disease, but dilatation predominates when the symptoms of compression supervene. In most cases the pulse is slow during the early stages of the disease, but becomes frequent and irregular before death. The course of the disease is very variable. It usually begins with more or less acute symptoms, which are followed by an interval of comparative health; after a time a fresh acute attack occurs, which may cause death or is followed by another interval. The majority of cases terminate fatally, although it is not impossible that partial recovery may occasionally take place.

II. DISEASES OF THE PIA MATER.

(I.) Tubercular Meningitis (Acute Hydrocephalus).

Etiology.—Most of those who suffer from tubercular meningitis belong to families in which the tubercular diathesis is distinctly marked. The largest proportion of cases of tubercular meningitis should occur in crowded populations and large cities, and amongst the poorest and most neglected part of the population. Season does not appear to exert any influence in the production of the disease. Tubercular meningitis may set in at any age, but it is much more frequent between the ages of two and seven years. The numbers diminish from the seventh to the tenth, and in still greater proportion from the tenth to the fifteenth year. In adults it is most common between the ages of twenty and forty, and occurs very exceptionally after the forty-fifth year. The male sex appears to be more frequently affected than the female. In adults the proportion is 51.5 men to 48.5 women, and the proportion of males affected in children is still greater.

Symptoms.—Premonitory symptoms manifest themselves for a variable period of weeks or months before the development of the distinctive phenomena of tubercular meningitis, the most constant of these being a gradual loss of flesh without perceptible cause, which is more noticeable in the trunk and limbs than in the face, loss of appetite, constipation or diarrhœa, and an undefined feeling of illness and weariness.

As the invasion approaches the patient, when a child, loses his vivacity, becomes sad, fretful, taciturn, irritable, and wants to be left alone, and in the midst of play leaves his companions in order to give vent to his distress in tears. He sleeps with eyes half open, and grinds his teeth, while his sleep is disturbed by muscular twitches and horrible dreams, so that he frequently starts and cries out.

On awakening he complains of headache, and after a time a slight febrile accession is observed towards evening without obvious cause.

In some cases the brain symptoms make their appearance without any warning, and the first symptoms which indicate an affection of the pia mater may be violent headache, vomiting, or facial paralysis, followed quickly by unconsciousness.

The disease itself presents (*a*) an initial period in which symptoms may be referred to excitation of the cortex; (*b*) a period in which symptoms caused by excitation are mixed with those due to progressive pressure caused by effusion and to lesions at the base of the brain; and (*c*) a

final period in which symptoms due to pressure caused by the ventricular effusion greatly predominate.

(*a*) *The Period of Invasion.*—In some cases the invasion is ushered in by a chill, accompanied by a rapid rise of temperature, while in others there are distinct shiverings, violent headache, and attacks of giddiness. Vomiting is, however, the most common special symptom of the period of invasion. It varies greatly in frequency, but as a rule occurs only twice in twenty-four hours during the first two or three days of the disease. In some few cases, however, the child vomits incessantly, whether food is taken or not, but when once the tendency to vomit has ceased for twenty-four hours, it does not ordinarily recur. Constipation is present, as a rule, throughout the whole course of the disease. Although there are occasional exceptions, the constipation is not often obstinate, and it is generally easy to procure action of the bowels by ordinary means.

Headache, of a dull, heavy, or lancinating character, is usually an urgent symptom while consciousness is retained, and its temporary exacerbations are made known by moaning or shrieks, while even after unconsciousness has set in the patient puts his hand to his head, wrinkles his forehead, and distorts his face as if from pain. Vertigo is always present. Patients feel as if they were falling in bed, or as if surrounding objects were revolving around them, and the gait is often reeling and unsteady, but the rapid development of severe symptoms soon prevents all attempts at station and locomotion.

The *motor disturbances* consist of spasmodic movements in the form of partial convulsions, giving rise to tremor and conjugate deviation of the eyeballs, with rotation of the head and neck, strong convergent and divergent squints, grinning contortions of the muscles of the face, and grinding of the teeth. It is probable that the hydrocephalic cry, which so commonly occurs during this period of the disease, which Trousseau says is "single and loud, like the cry of a person frightened by some sudden danger," is due to spasmodic action of the respiratory and laryngeal muscles, and is quite independent of consciousness. The muscles of the neck and back are often persistently contracted, and the abdomen is retracted, owing to spasm of its muscles, so that it presents a characteristic boat-shaped depression. In children epileptiform convulsions are not uncommon, but they are rare in adults, and towards the end of this stage slight paralysis of the ocular and facial muscles may be present, as declared by inequality of the pupils, ptosis, strabismus, or slight facial paralysis.

The *sensory disturbances* are often obscured by loss of consciousness, but at times a general or partial hyperæsthesia of the surface of the

body may be observed at the beginning of the disease, while general or partial anæsthesia is not infrequent at an advanced period of the disease, and has occasionally been observed as a premonitory symptom. Intolerance of sound and light is a prominent symptom; the child is impatient of the slightest noise, and avoids the light by lying with the face buried in the pillow or turned towards the wall, with the knees drawn up towards the abdomen, and the eyelids firmly closed.

Psychical disturbances are not always present in the early period of the affection. The patient cannot, however, form consecutive trains of ideas, and children soon become somnolent, lie with their eyes closed, and reply to questions curtly or merely by a nod. When raised up, they complain much, knit their brows, throw back their heads, and slip down in bed; they cannot bear the slightest disturbance, and will clench their teeth against food. Delirium is of frequent occurrence when the patient is half asleep; and, in the case of children, the hydrocephalic cry is not infrequently heard at this time, being accompanied by starting up in terror. At other times the patient may spring from bed, or make defensive movements in consequence of hallucinations of sight and hearing. The somnolence soon increases, the eyes stare without expression into vacancy, and the patient ceases to speak to his attendants, but he talks senselessly with himself, or laughs, sings, whistles, or shouts, and performs meaningless movements, such as plucking at imaginary objects, picking the bedclothes, blowing, spitting, and grimacing. After a time these disturbances cease, and the mental faculties may again become completely or partially clear, but after a longer or shorter period, new and more profound disturbances supervene, which increase until the fatal termination.

Disorders of vision are seldom absent in this stage so long as the patient is tolerably conscious, and an ophthalmoscope may reveal the presence of double optic neuritis, or tubercles of the choroid.

The pulse is, as a rule, slow and full during the early stage of the affection, but it is subject to rapid variations during muscular exertion of every kind and psychical excitement, especially in the case of children. The temperature is very inconstant, and does not conform to any type. The skin is usually dry, and even in those cases where the tubercular meningitis is associated with phthisis, the sweats of the latter disease usually cease when the meningitis manifests itself. It has also been observed that the cough, dyspnœa, and expectoration of phthisis cease, and in the case of intestinal tuberculosis the diarrhœa is arrested. The injection of the face and conjunctivæ varies frequently without obvious cause; lividity of the face belongs to a later period, or is dependent on lung complications.

The average duration of the first stage is about eight days, but it varies from two days to two or three weeks, and is not unfrequently followed by a remission which leads the friends of the patient to hope for a favorable termination.

(*b*) *The second stage* is now ushered in with the evidences of loss of functional activity. The signs of excitement become less frequent, and somnolence and mental torpor more prominent, but even in the midst of sopor adults give evidence of suffering from severe headache by groans and gestures, while in children the hydrocephalic cry is frequently heard at this stage.

The presence of muscular spasms is declared by combined movements of the eyeballs or nystagmus, inequality of the pupils, winking and facial contortions, and grinding of the teeth with munching movements of the jaws, while in some cases one or more of the limbs are agitated by tremors or choreic movements, and in rare cases the muscles of the trunk and limbs are maintained in a state of cataleptic rigidity.

The stiffness of the muscles of the nape of the neck becomes more intense, so that the head is drawn back and thrust into the pillow. The rigidity may also extend to the muscles of the trunk, and the body is maintained in a condition of tetanic rigidity, the opisthotonos either appearing in paroxysms and lasting only a few minutes at a time, or remaining continuous until death. Paralyses of various extent and distribution now make their appearance. Paralysis of the oculo-motor nerve is common, and gives rise to divergent squint, ptosis, and dilatation, with fixity of one or both pupils. The trochlear nerve may be paralyzed along with the third, but is never affected alone. Paralysis of the abducens may, however, occur as a separate affection. The paralytic form of conjugate deviation of the eyes and rotation of the head and neck may appear at this period of the disease, and decided paralysis of one side of the face is often present, while the tongue is drawn towards the paralyzed side on protrusion. It is not uncommon for aphasia to occur, especially in adults, in this stage of the disease. The disorder of speech may either be a sensory or a motor aphasia, or a sensory aphasia may appear first, then there is a rapid improvement, but soon a motor aphasia is superadded and in a few days the patient dies comatose after suffering probably from severe general convulsions.

Hemiplegia and hemiparesis are rare, but monoplegiæ are frequent. At times one arm or one leg is paretic or completely paralyzed; while occasionally the paralysis assumes the paraplegic form, or a considerable diminution of strength may occur in all four extremities.

The *sensory disorders* consist of partial anæsthesia which may be limited to one or other extremity or to the side of the face, but as the

disease progresses the anæsthesia becomes more profound, and distributed over the whole body. The optic disks are markedly swollen in this stage, and retinal hemorrhages are a frequent complication.

Vomiting is an occasional symptom of this stage, the constipation continues, and the urine is generally passed in bed or there is complete retention. The pulse becomes less frequent, and may sink to sixty or still lower, and is at the same time fuller; but the slightest excitement may raise the number of beats to more than one hundred per minute, and it is apt to become irregular. The temperature varies, and although it usually conforms to the remittent type in children which is so characteristic of tuberculosis of other organs, yet it should be remembered that in adults it may remain almost completely normal throughout this stage of the disease.

The respiration is sometimes normal, at other times irregular, and it occasionally assumes the character of the Cheyne-Stokes respiration. Neuro-paralytic œdema of the lungs is liable to occur at this period of the disease. This period may last about a week, and then the third stage of the disease is ushered in by the appearance of persistent coma.

(*c*) *The Third Stage.*—The patient is now completely insensible to most external excitants, and most reflex actions are extinguished, although he may still respond to a loud voice close to the ear.

Isolated paralyses are not now so readily observed, inasmuch as there is general relaxation of the whole muscular system, but rigidity of the masticatory muscles and of the muscles of the back and extremities is sometimes still present. Convulsions are rare in adults; but in children partial convulsive spasms may occur, and immediately before death the patient may be seized with general convulsions. The electric excitability of the muscles to both constant and induction currents is normal. Every form of sensibility is now completely abolished. The pupils are dilated and fixed, the upper lids fall inert and paralyzed over the eyeballs, and an ophthalmoscopic examination reveals signs of marked œdema of the optic disks. The pulse during this period becomes more and more frequent and may be irregular, and in the case of children the pulsations of the fontanelles become weaker and weaker, and may disappear before death.

The respiration is irregular, and at times so superficial and feeble that it may escape observation, while at other times it is forced and deep. Rapid emaciation occurs, especially in the case of children; the skin is generally dry and rough, and it may become slightly livid with the decreasing force of the heart; and occasionally a bedsore appears. The temperature during this period varies greatly. In some cases it is below normal, and immediately before death may sink as low as 95° F. or

even 93° F. In another series of cases a state of moderate fever continues up to the time of death; while in a third group the fever greatly increases before death, and may even continue to increase for some time afterwards.

The urine is generally scanty with high specific gravity; its quantity may be increased or normal; the proportion of chlorides varies greatly; and the phosphates are said to be increased. A small quantity of albumen is frequently present, but sugar is rare. The duration of the paralytic stage is generally three or four days, and seldom reaches a week.

The course of tubercular meningitis is very variable. In some cases it runs a rapid course and terminates fatally in five or six days. In other cases the disease is very protracted; the invasion is slow and insidious, and the disease may last from thirty to fifty or even sixty days.

Chronic Hydrocephalus.

The etiology of chronic hydrocephalus is not well known; but hereditary predisposition appears to exert some influence in its production, for more than one child may be affected in the same family. Congenital syphilis is probably the most important predisposing cause, and it is possible that too much importance has been attributed to rickets in its production. The disease is always congenital or acquired soon after birth, except when it results from intracranial tumors or from occlusion of one or more of the lateral sinuses. Of the exciting causes little is known. Chronic hydrocephalus is sometimes preceded by an attack resembling acute hydrocephalus.

Symptoms.—In congenital hydrocephalus cerebral symptoms, such as daily recurring convulsions, strabismus, or rolling of the eyeballs, are apparent from the infant's birth, while in a few days or weeks the head is observed to undergo progressive enlargement.

Impairment of the general nutrition is one of the first symptoms; the child may seem eager for food and sucks well, yet it loses flesh and strength, and the skin hangs in loose folds on its attenuated limbs. The bowels are generally constipated, or diarrhœa may alternate with constipation, and the evacuations are always unhealthy. The child is restless and may be drowsy during the day, but wakeful and fretful during the night. The fontanelles and sutures are now unusually open, the anterior fontanelle is tense and pulsates strongly, and the child is subject to paroxysms of restlessness, during which there is increased heat of the head.

The sutures become gradually wider with the increase of effusion, the fontanelles increase in size, the head assumes a globular form, and the physiognomy of the child soon acquires the characteristic features of chronic hydrocephalus. As the fluid accumulates within the cranium it presses equally in all directions, and the cavity of the skull must enlarge in the direction of least resistance. The greatest increase in the size of the head is effected chiefly by enlargement of the anterior fontanelle and by widening of the sagittal suture, these being the points which are the last to be ossified, and at which the bones of the skull are less firmly fixed. The frontal bones are consequently pushed forwards, rendering the forehead round and prominent, the parietal bones are pressed backwards and outwards, and the occipital bone downwards and backwards, sometimes even so far that it assumes an almost horizontal position. The head is, as a rule, globular and flat at the top, although it occasionally assumes a conical form. Its size varies, and it has been known to measure two or even three feet in circumference. The orbital plates of the frontal bones are pushed from the horizontal to an oblique, or it may be almost vertical position, and thus encroach upon the cavities of the orbits. The eyeballs are consequently pressed forwards and rendered prominent; they are at the same time rotated downwards, so that the sclerotic coat appears below the upper lids, while the pupils are half hidden beneath the lower lids. On placing the hand over the open fontanelles and sutures they are felt tense and fluctuating. The hair grows scantily over the head, and the skin is tense and shining, differing in this respect from the wrinkled condition of that of the rest of the body; distended veins ramify over the scalp; and the enlarged head contrasts strikingly with the small infantile face. The child has a dull and stupid expression, and being unable to hold his head up he is obliged to remain in the recumbent posture or to assume a half-sitting position, while his head is supported with his hands or propped up with pillows. The most usual cerebral symptoms which appear during the progress of the case are convulsions; attacks of laryngismus stridulus; paralyses with contractures of varying distribution; rolling of the eyeballs; and amblyopia progressing to amaurosis. Hearing, as a rule, remains unaffected until near the fatal termination. Chronic hydrocephalus lasts usually from one to two years, but in exceptional cases the patient has been known to live to middle or even old age. The disease is almost always fatal, death being caused by laryngismus stridulus, general convulsions, or some intercurrent disease.

(II.) Meningitis of the Convexity of the Brain.

Etiology.—Simple *primary* meningitis of the convexity of the brain is a rare affection. It may occur at all ages, but chiefly attacks infants under two years of age, when it is named *leptomeningitis infantium.* It occurs with decreasing frequency from this age to puberty, when it becomes more frequent, but it is only very rarely met with in advanced age. In adults men are more frequently attacked than women. The exciting causes of the disease are not well known.

Secondary meningitis of the convexity may be produced by inflammation of the bones of the skull, the usual causes of the latter being external injury, scrofula, and syphilis. Gummata may also give rise to this inflammation. Otorrhœa, especially when complicated by caries of the temporal bone, is one of the most frequent causes of purulent meningitis, and the affection may result from puriform softening of a thrombus in one of the sinuses, erysipelas of the head leading to osteophlebitis of the bones of the skull, carbuncles of the face and neck, suppuration of the eyeball, and all intracranial diseases like tumors, abscesses, or necrotic softening.

Symptoms of Primary Meningitis of the Convexity.—The course of acute meningitis may be divided into three stages: (1) The period of excitement, (2) the period of transition, and (3) the stage of collapse.

(1) *The Period of Excitement.*—Obscure premonitory symptoms are sometimes observed, consisting usually of a feeling of heaviness in the head along with paroxysms of violent cephalalgia, sleeplessness, irritability of temper, and general malaise. As a rule, however, the disease begins suddenly by a well-marked rigor, intense headache, vomiting, fever, and delirium, while in children it is ushered in by an attack of general convulsions.

The headache may be diffused or referred to the forehead, temples, vertex, or occiput; it is intensified by light and sound, and by all movements of the head. The patient consequently shuns the light, and holds his head between his hands, in order to prevent it from moving. The headache is continuous, but is marked by exacerbations of intense severity, during which the patient, especially if a child, may utter a loud and piercing cry.

Vomiting is a very constant symptom of meningitis, and is, like the vomiting symptomatic of other cerebral diseases, unattended by nausea, and epigastric pain or tenderness. It recurs frequently during the first forty-eight hours, and may then cease or occur at intervals throughout the course of the affection.

The motor disturbances in this stage consist of strabismus, slight twitching of the muscles of the face and limbs, and tonic spasms of those of the neck and back, while the patient staggers if he attempts to walk. The pupils are usually contracted or unequal, but react readily to light.

The sensory disturbances consist of buzzing in the ears, flashes before the eyes, and intolerance of light and sound. Cutaneous hyperæsthesia is not infrequently present, so that the slightest touch on the skin may cause pain, and the reflex excitability is increased.

The psychical disturbances are well marked from the first. The patient is extremely irritable, and fierce delirium is apt to occur, the patient shouting and violently struggling with his attendants. At other times he is morose, and buries his head under the bedclothes, obstinately refusing to answer questions. The temperature of the body is elevated, the pulse beats from one hundred and twenty to one hundred and forty or more, and the respirations are increased to thirty or forty in the minute.

(2) *The Period of Transition.*—During this period the furious delirium of the first stage becomes quieter, the patient lies on his back, with his fingers picking at the bedclothes or catching imaginary flies in the air.

The motor symptoms of this period consist of strabismus, conjugate deviations of the eyeballs, with rotation of the head and neck, twitchings of the face; grinding of the teeth and trismus; distortions of the tongue; the hydrocephalic cry; subsultus and tremor of the hand; convulsive movements of the limbs, and retraction of the head and neck with opisthotonos.

These convulsive symptoms are followed by paralysis, which is very variable in its distribution, some groups of muscles being paralyzed while others continue convulsed.

The sensory disturbances consist of dimness of vision and of hearing, ending in blindness and deafness, while the general cutaneous hyperæsthesia of the first stage is replaced by anæsthesia. The bowels are constipated throughout, and the abdominal walls are often retracted as in tubercular meningitis. The respirations are irregular, the pulse frequent and thready, and there is retention of urine.

(3) *The Stage of Collapse.*—The third stage of the affection now becomes established, the convulsive phenomena give place everywhere to paralysis, and the patient passes into a profound and fatal coma.

Symptoms of Secondary Meningitis.—The symptoms of secondary meningitis differ considerably according to the cause of the inflammation, but, inasmuch as inflammation from caries of the petrous bone is the most usual form of the affection, it will be useful to describe it first.

The affection may be ushered in by chilliness or a distinct rigor and feverish symptoms, but intense headache, either continuous or marked by remissions and exacerbations, is the first symptom to direct attention to the brain. The headache may be fixed to a point in the vicinity of the diseased ear, or shoot from one ear to another, while at other times it is diffused over the whole head. If the local affection be attended by pain, the commencement of the meningitis is marked by a great increase of its intensity, and the onset of the latter may sometimes be completely masked by an increase of the local inflammation in the ear. Attacks of dizziness now supervene, accompanied by nausea and vomiting; the patient complains of noises in the head, general painful sensations diffused over the body, and obscuration of the special senses, while he often becomes actively delirious.

The motor symptoms are rigidity of the muscles of the nape of the neck, convulsive twitching of the facial muscles on the affected side, trismus, grinding of the teeth, and occasionally spasms of the extremities.

The spasmodic phenomena, however, soon give place to paralysis affecting first the facial, hypoglossal, and glosso-pharyngeal nerves, and should the inflammation extend forwards along the base of the skull, the third, fourth, sixth, and probably the fifth nerves on the same side as the lesion. The state of the pupils is variable, and liable to frequent changes during the course of the disease, being generally contracted or unequal at first, and dilated and fixed when effusion has taken place. Paralysis of the extremities is rare, but the patient has an unsteady, staggering gait.

The *sensory disturbances* consist of marked hyperæsthesia of the skin, joints, bones, and muscles, so that every movement is painful.

Vomiting generally continues throughout the whole course of the disease, the bowels are constipated, and the abdominal muscles are tender to the touch and retracted. The temperature in acute cases is usually high, but remits in the morning, although it remains constantly high in some cases.

The pulse, as a rule, rises and falls in frequency along with the temperature, except in the cases where symptoms of compression of the brain occur during the first days of the disease.

The urine is often albuminous, and this may or may not be associated with amyloid disease of the liver, spleen, and kidneys.

The optic disks usually present the same appearances as those observed in tubercular meningitis. The psychical symptoms are very variable, consisting of jactitation, restlessness, and confusion of ideas, especially towards evening, when the temperature rises. After a time the patient falls into a somnolent condition, from which he can at first be readily

roused by a loud question, but this state soon gives place to profound and fatal coma.

(III.) Simple Meningitis of the Base of the Brain (Basilar Meningitis).

Etiology.—Inflammatory processes at the base of the brain are caused by fissures, tumors, abscesses and other lesions at the base of the brain, but the form to be described at present arises spontaneously, and occurs in persons from sixteen to thirty years of age.

Symptoms.—When primary basilar meningitis is diffused and general, the affection begins by languor, mental depression, chilliness or even rigor, thirst, and the usual symptoms of fever. The patient complains of intense cephalalgia and giddiness, and these are followed by severe attacks of vomiting.

Motor disturbances may be completely absent throughout the whole course of the affection, but when present they consist of spasmodic rigidity of the muscles of the back of the neck, with retraction of the head, and rarely of rigidity or clonic twitchings of certain groups of the muscles of the extremities. The patient grinds his teeth during sleep, and in the later stages of the affection trismus and hiccough have been observed. Paralysis of the abducens is not uncommon, but paralysis of the oculo-motor nerve is rare. Paresis of the facial or hypoglossal nerves may occur temporarily during the course of the affection, and subsequently disappear, but complete paralysis of them has not been observed. The power of deglutition may be impaired during the course of the affection and be afterwards regained, or it may increase to complete dysphagia before death. Paresis of the extremities is occasionally observed, but never complete paralysis.

The sensory disturbances consist of cutaneous hyperæsthesia, especially in the region of distribution of the fifth nerves, ringing in the ears, scintillations before the eyes, and occasionally hallucinations. The psychical disturbances are more variable than in any other form of meningitis. In some cases the mental faculties are unaffected throughout the whole course of the disease, while in others they are early involved. The mental symptoms usually consist of a mild delirium; but in exceptional cases this may be more active, the patient being restless, quarrelsome, capricious, and irascible. Active delirium is usually temporary and soon gives place to a milder form; the patient after a time becomes somnolent, but may be temporarily aroused by a loud question addressed to him; in a short time he fails into a state of complete in-

sensibility, during which the urine and feces are passed involuntarily. Vomiting continues to distress the patient throughout the course of the disease, the bowels are constipated, but the abdomen is not retracted as in tubercular meningitis.

The temperature may rise in the first stage as high as 104° F. in the evening and approach to the normal in the morning, but in the later stages the temperature remains low, being sometimes subnormal. The pulse, as a rule, follows the temperature, being very frequent in the initial period, and sinking in the course of the disease to below sixty beats in the minute. Towards the end of life it again increases, and becomes very frequent, irregular, and intermittent; the patient is covered with bedsores, much emaciated, and dies in a state of marasmus.

The chronic forms of basilar meningitis may give rise to localized inflammatory products at the base of the brain, which cause symptoms scarcely to be distinguished from those of tumors occupying the same situation. The symptoms are variable in such cases, the most characteristic being paralyses of the various cranial nerves. In addition to the headache and dizziness, there are anosmia, amaurosis, or hemianopsia, ptosis, paralysis of the motor nerves of the eyeball, sensory disturbances in the region of distribution of the fifth nerve, masticatory paralysis, paresis of the seventh nerve, and occasionally paresis of one or more of the extremities. If the inflammation extend to the lower end of the pons, bulbar paralysis, dysphagia, and dyspnœa may be present.

(IV.) Metastatic Meningitis.

Metastatic meningitis comprises certain varieties of the affection, which occur as terminal phenomena in the course of acute diseases.

The diseases with which meningitis is most frequently associated are pneumonia, ulcerative endocarditis, acute rheumatism, purulent pleurisy, pericarditis, diphtheria, and the acute exanthemata. Although chronic Bright's disease is liable to be complicated by inflammation of the serous membranes, meningitis is rare.

Symptoms.—The extent and intensity of the inflammation vary greatly in different cases; in some there is little or no effusion into the ventricles, and the symptoms of compression are absent; the inflammation is sometimes limited to the convexity, and at other times extends to the base and upper part of the spinal cord; and in the meningitis of acute febrile diseases the symptoms are obscured by the cerebral disturbance usually observed in all grave acute affections.

Varieties of Metastatic Meningitis.

Meningitis with Pneumonia.—Meningitis may appear in the course of pneumonia from the third to the eighth day, or even later. The most usual symptoms are chilliness, intense headache, rapidly developed and mild, or occasionally furious delirium, a fresh accession of fever, and hyperpyrexia before death. The delirium gives place at an early period to somnolency, ending in coma. A slight degree of rigidity and pain in the neck is always a valuable sign of meningitis, and vomiting is a frequent occurrence. The pupils are generally contracted at first, and may subsequently become unequal. If the base of the brain be affected, paralysis of the oculo-motor and other nerves at the base of the skull renders the nature of the complication more apparent.

Meningitis with Ulcerative Endocarditis.—The cerebral symptoms in ulcerative endocarditis are caused by multiple hemorrhagic infarctions of the cortex of the brain or of the pia mater, and the symptoms produced are more or less like pyæmic encephalitis.

Rheumatic meningitis has been described, but post-mortem evidence of its existence is wanting.

(V.) Traumatic Meningitis.

Etiology.—This form of meningitis or of meningo-encephalitis may appear during the period of reaction from concussion, or follow a contusion of the brain. Injury of the scalp, with subsequent inflammation of the bones of the skull and dura mater, may also give rise to inflammation of the pia mater and brain. At other times the inflammatory process is set up by a perforating injury of the skull either with or without extravasation of blood between the dura mater and the bone; the effects in such cases being intensified by the admission of air containing germs into the open wound. In other cases the meningitis is a secondary result of osteitis, thrombosis of the sinuses probably playing an important part in its production in such cases. The meningitis at other times may result after necrosis of the bone has taken place. Hutchinson thinks that in fractures of the petrous portion of the temporal bone the inflammation extends along the sheath of the seventh nerve, and in this way gains access to the subarachnoidal spaces.

Symptoms.—This affection may be divided into two varieties: (*a*) *acute*, and (*b*) *chronic or subacute* traumatic meningo-encephalitis.

(*a*) *Acute Traumatic Meningo-encephalitis.*—The symptoms of the onset of the acute form of the affection are modified by the fact that

the inflammatory process usually attacks a patient already suffering from the symptoms of concussion, compression, or contusion of the brain. It is impossible to distinguish between the symptoms of the reaction period of concussion and those of the early or congestive stage of true inflammation, so that we are unable to determine when the symptoms of the former terminate and those of the latter begin. At the onset of the inflammatory attack the patient complains of severe and continuous cephalalgia; the carotids beat forcibly; the face is suffused and the scalp hot; the pupils are contracted; and there are intolerance of light and sound, spectral illusions, noises in the ears, and general hyperæsthesia to external impressions. The patient likewise suffers from the usual symptoms of pyrexia; the pulse is full and bounding, and there are restlessness and wakefulness with delirium of a violent character. These symptoms may, under proper treatment, gradually subside until health is reëstablished; but more commonly the symptoms of the stage of irritation develop into those of the stage of compression.

During the transition period between the early stage of excitement and that of compression of the brain, the symptoms of a localized disease may make their appearance. Clonic or tonic spasms, followed by paralysis, may occur in particular groups of muscles. Rigidity of the muscles of the nape of the neck, with retraction of the head, is usually present at this period, and may also extend to the muscles of the back and give rise to tetanic seizures. Hemiplegia of the side opposite to the injury is, according to Hutchinson, a constant symptom of direct traumatic meningo-encephalitis. The abdominal muscles are usually retracted and the bowels constipated. When the meningitis is situated at the base of the brain, the cranial nerves in their passage along the base of the skull may become implicated. The most usual symptoms produced are ptosis, strabismus, paralysis of the facial muscles or of half of the tongue, and difficulty of deglutition.

The symptoms of compression of the brain now become rapidly developed; the delirium is replaced by stupor, from which the patient is roused with difficulty; the pupils are dilated and insensible to light; the breathing is slow and stertorous; the pulse, retarded at first, becomes feeble and frequent towards the end; the skin is hot and bathed in perspiration; and convulsive twitchings or jerkings of the limbs are observed; but these soon give place to general muscular relaxation, and the patient dies in profound coma.

(*b*) *Subacute Traumatic Meningo-encephalitis.*—This form of meningo-encephalitis may come on a few days after the injury or not until months have elapsed. The patient has often apparently recovered from

the original injury, but, as a rule, some of the consequences of concussion remain. The patient in the interval has complained of headache, impairment of sight and hearing, confusion of ideas, or mental irritability. The symptoms of the inflammatory attack may be ushered in by an aggravation of the symptoms which have persisted during the interval, or by an epileptic attack. The pupils are contracted, dilated, or unequal; there are intolerance of light and sound, convulsive twitchings of the limbs and face, strabismus, delirium, and the ordinary phenomena of symptomatic fever. After a time the symptoms of compression supervene, and the patient dies comatose.

Morbid Anatomy.—In *external pachymeningitis* the dura mater is at first congested and presents punctiform extravasations, while at a more advanced stage the membrane becomes swollen and infiltrated with numerous white blood-corpuscles. If the disease has been acute, the white corpuscles become more numerous and make their way inwards, so that a purulent internal pachymeningitis is superadded to the external pachymeningitis. If, however, the disease has been chronic, the white blood-corpuscles become transformed into spindle-cells and ultimately develop into connective tissue. In long-standing cases portions of the membrane may become calcified.

Hemorrhagic pachymeningitis appears to begin with hyperæmia of the dura mater in the area of distribution of the middle meningeal artery. The inner surface of the dura mater assumes a rosy color, and after a time a loose yellowish coating or false membrane forms, which is dotted with a number of confluent or separate hemorrhagic points. The false membrane become very vascular and from time to time some of them rupture, giving rise to hemorrhages of variable quantity. The clots which form become partially organized and the delicate capillaries which develop in them become the source of new hemorrhages, so that in this way a large quantity of blood may be poured out between the thickened membranes. Hæmatoma is most frequently found in the upper part of the brain along the falx cerebri and thence it spreads down the curved portion of the frontal and occipital lobes and laterally towards the Sylvian fissure. The bones of the skull have been found sometimes abnormally thin and at other times unusually thick, and the brain itself is compressed by the hæmatoma, while it is often the subject of atrophy independently of the disease in the membranes.

In *tubercular meningitis* the changes in the pia mater are (1) those which are directly connected with the formation of tubercles, (2) those caused by the inflammation surrounding them, and (3) those which arise from the effusion into the ventricles.

(1) The pia mater is studded with tubercles which appear as grayish-white granulations distributed along the course of the vessels, the territory supplied by the Sylvian artery being particularly liable to be affected.

(2) The inflammatory changes in the pia mater vary greatly according to the amount of effusion which has been present. Evidences of suppuration are found at the base of the brain, and cloudy streaks of exudation may be observed passing along the vessels. In some cases a purulent or fibrinous effusion is observed in the pia mater of the convexity, which may extend backwards over the anterior surface of the pons and medulla and find its way over the entire surface of the cerebellum. In some cases the choroid plexuses and the velum interpositum are covered with a yellowish purulent exudation.

(3) The ventricles are generally distended with serous or sero-purulent fluid, and the brain is compressed, so that the cortex and white substance are dry and anæmic, but when effusion is absent those parts are congested and œdematous.

The cortex is often studded by punctiform hemorrhages caused, according to Rindfleisch, by tubercular degeneration of the nutritive arteries. When the exudation spreads along the base of the brain the cranial nerves are implicated in the disease, and even the spinal cord is not unfrequently affected.

Essentially the same phenomena are met with in the other forms of acute meningitis as in the tubercular variety, so far as the appearances of inflammation and the effusion are concerned, and it is, therefore, unnecessary to describe the morbid changes of these diseases in detail.

In *chronic hydrocephalus* the general ventricular cavity of the brain contains serous fluid, which varies in quantity from a few ounces to many pounds, and consequently the hemispheres of the brain are compressed and flattened, so that the sulci and convolutions almost entirely disappear.

Morbid Physiology.—The premonitory symptoms of all the acute forms of meningitis are such as are met with in all severe inflammatory affections, and the purely nervous phenomena of the period of invasion are caused by irritation of the cortex of the brain, and the student who is acquainted with the physiology of the cortex will be able readily to explain the various sensory disorders and the spasmodic affections which occur at this period of the disease. In the second stage of the disease portions of the structure of the cortex are being destroyed, so that the hyperæsthesias and spasms of the first period become mixed up with various forms of anæsthesia and paralysis. Direct implication of the cranial nerves in the inflammatory process at the base of the brain explains some of the local forms of anæsthesia and paralysis which

occur at this period. As the effusion increases in quantity the brain is subjected to compression and its functions are gradually or suddenly abolished, and the patient dies comatose.

Treatment.—The treatment of all the acute forms of meningitis must be conducted on the same general principles, by the application of ice to the shaven scalp, rest in a darkened room, and moderate evacuation of the bowels by a saline purgative. Local bloodletting often relieves the severe headache and gives at least temporary relief.

In *external pachymeningitis* the question of trephining will arise when an abscess forms or an extravasation of blood takes place between the dura mater and bone, but such cases come under the care of the surgeon.

In *hæmatoma* the treatment will depend greatly upon the underlying affection, and a careful examination must be made into the habits of the patient, and of all the other organs, in order to discover upon what disease the meningeal affection depends.

In all the acute inflammations of the pia mater mercury may be given in frequently repeated small doses; in the tubercular disease this treatment can do no harm, and in the simple varieties it may do good. Iodide of potassium has also been given with the view of promoting absorption of the effusion but the results of this treatment are doubtful. When the headache is very severe, morphia in suitable doses should be administered subcutaneously, and chloral hydrate and bromide of potassium either separately or combined will be found useful when the convulsive symptoms are severe. Blisters or croton oil liniment to the shaven scalp have been much used in the treatment of meningitis, but I cannot too strongly condemn this practice.

In chronic hydrocephalus partial success has been obtained by withdrawing a few ounces of fluid at a time by means of an aspirator, while the skull is at the same time supported by bandages.

CHAPTER XX.

PARALYSIS AGITANS, MULTIPLE SCLEROSIS, AND CHOREA.

I. PARALYSIS AGITANS.

Etiology.—Paralysis agitans occurs generally in advanced age, although a few cases have been observed in young people. Men are more frequently attacked than women. The disease is frequently met with amongst persons living in damp and unhealthy situations, and some authors believe that there is a causal relation between it and rheumatism. It is sometimes caused by great emotional disturbance, and many cases follow wounds and other injuries.

Symptoms.—The clinical history of paralysis agitans may be divided into three stages: (1) The period of invasion; (2) the stationary period; and (3) the terminal period.

(1) *The Period of Invasion.*—Paralysis agitans may begin in a slow and progressive manner, or more or less suddenly. In the *slow* mode of invasion the characteristic phenomena of the disease are preceded by premonitory symptoms, consisting of sleeplessness, mental irritability, rheumatoid or neuralgic pains, a remarkable feeling of fatigue, and a transitory feeling of weakness in the limbs. After a longer or shorter time the disease declares itself by a trembling in one of the extremities, generally in the small muscles of one hand. At first the tremor is only occasionally present, but as the disease advances it becomes persistent during waking hours, but ceases with sleep, and it can be arrested by a strong voluntary effort. When the hand is the first part affected, the "patient," to quote Charcot, "closes the fingers on the thumb, as though in the act of spinning wool, at the same moment the wrist is bent by rapid jerks on the forearm and the forearm on the arm." As the disease advances, the tremor increases in intensity and persistency, and invades by degrees parts which have hitherto remained sound. The order in which the tremor invades the different groups of muscles is somewhat variable. In some cases the tremor is restricted for a long time to one-half of the body, the tremor begins in the right hand, and after months or years the lower extremity on the same side becomes affected, and after another variable period the left hand and foot are

successively invaded. In other cases both lower extremities are first affected, and in a few cases the upper extremity of the one side, generally the right, is first attacked, and then the lower extremity of the opposite side.

In the *sudden* mode of invasion the tremor appears suddenly either in one extremity or in all the limbs at once; it may soon diminish or disappear, but soon recurs, and after a series of alternate exacerbations and remissions, becomes permanent. The duration of the initial stage varies from one to three years.

(2) *Stationary Period.*—When the disease is fully developed the trembling becomes almost incessant, although it varies in intensity. It is aggravated by emotional excitement, cold, and voluntary effort, and becomes less during repose, and ceases during sleep. The different segments of the fingers and hand undergo involuntary and rhythmical oscillations, which closely resemble voluntary movements. "Thus in some patients," says Charcot, "the thumb moves over the fingers as when a pencil or paper ball is rolled between them; in others the movements are more complicated, and resemble what takes place in crumbling a piece of bread." The handwriting now assumes special characteristics. At an early stage of the disease the writing at the first glance presents little change, but when examined with a magnifying glass slight inequalities are perceived, and as the disease advances the up-strokes become markedly tremulous.

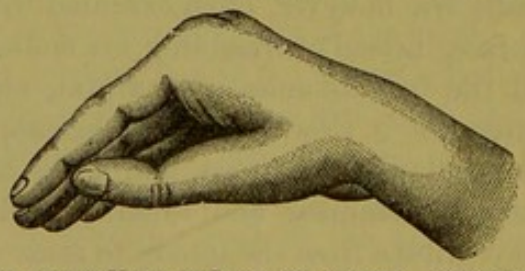
The muscles of the head and neck, as already stated, usually remain unaffected. The muscles of the eyeballs are also exempt from tremor, and consequently nystagmus, which is so prominent a symptom of disseminated sclerosis, has no existence in paralysis agitans. The movements of the eyeballs are, however, often executed with great slowness. The muscles of the face, instead of trembling, are motionless; the features become fixed; and the face assumes a mournful, stolid, or vacant expression. The utterance is slow, jerky, and accomplished with great apparent effort, soon inducing weariness, and if the tremor of the body be intense it becomes tremulous and broken, while in old-standing cases the saliva may dribble from the mouth to some extent.

After a longer or shorter time the muscular power becomes gradually weakened. In many cases, however, motor weakness is more apparent than real, the phenomena depending upon the great slowness with which voluntary movements are executed, the immense effort which all voluntary actions, even speaking, entail, and the readiness with which fatigue is induced. But although the muscular power, when measured by the dynamometer, is often retained much longer than might be expected, yet after a time motor power becomes gradually diminished, and

paralytic symptoms supervene. The paralysis, however, almost always remains partial, and is irregularly developed in different groups of muscles, and, as in various other forms of paralysis, the extensors of the limbs are affected to a greater extent than the flexors. The trembling often abates in the muscles as paralysis increases. The bladder and rectum are only very exceptionally involved in the paralysis. The muscles react normally to both the faradic and galvanic currents.

After a time the muscles of the extremities, trunk, and neck become the subjects of *rigidity*, at first temporary, but ultimately becoming permanent, the flexors being affected to a greater extent than the extensors. The rigidity of the muscles produces characteristic alterations in the attitudes of the body. The rigidity of the anterior cervical muscles causes the head to be strongly bent forwards, and the patient cannot raise it or turn it to either side without great difficulty. The body is also inclined forwards when the patient is standing. The elbows are habitually held somewhat removed from the chest, the forearms are slightly flexed on the arms, and the hands are sometimes flexed, sometimes slightly extended on the forearms, and rest on the epigastrium. The fingers are flexed at the metacarpo-phalangeal articulations, the index and middle fingers are extended, but the remaining fingers are slightly flexed at the phalangeal articulations, all of them are slightly inclined to the ulnar border of the hand, and the thumb is extended and opposed to the index finger, so that the attitude of the hand and fingers closely resembles that assumed by them in holding a pen (Fig. 179). In some cases the fingers are alternately flexed and

Fig. 179.

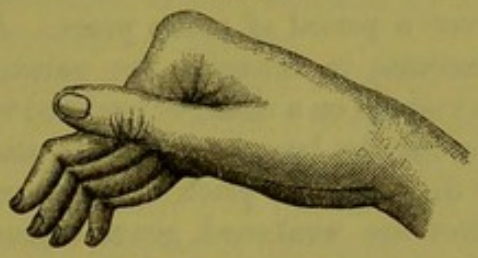


Attitude of the Hand in Paralysis Agitans. (After Charcot.)

extended at their several articulations so as to resemble the distortions of *arthritis deformans* (Fig. 180). In paralysis agitans, however, the joints are not swollen and stiff, and passive movement of the articulations does not give rise to the creaking sounds observed in the rheumatic affection. The rigidity of the muscles of the lower extremities is sometimes so great as to resemble paraplegia with contracture. The spasm

of the adductors of the thighs and muscles of the calf predominates over their antagonists, so that the knees are drawn inwards, the leg is slightly flexed on the thigh, and the foot assumes the well-known position of *talipes equino-varus*. The toes are extended at the metatarso-phalangeal and flexed at the phalangeal articulations (*Griffe des Orteils*).

Fig. 180.



Attitude of the Hand in Paralysis Agitans simulating that of Arthritis Deformans. (After Charcot.)

In the advanced stage of this disease the patients move all of a piece, as if their joints were soldered together, and the head and body are kept inclined forwards, a position which no doubt largely contributes to produce that tendency to fall forwards manifested when walking.

The gait is now characteristic. The patient gets up slowly and with difficulty from his seat, and hesitates for a few moments before starting; when once he has begun to walk, he is compelled to run forwards, in order to save himself from falling. In the language of Trousseau, he looks as if pursuing his own centre of gravity. This gait has been called *paralysis festinans* or *procursoria*, or simply *propulsion*.

The forward running or propulsion is the usual gait of paralysis agitans, but some patients manifest a strong tendency to run or to fall backwards, although their bodies are inclined forwards, a tendency which has been named *retropulsion*. Graves mentions the case of a patient who, if arrested in his forward movement, immediately began to run backwards, and Charcot could excite the impulse to move backwards in a female patient by slightly pulling her back by the dress when she was standing. It has already been mentioned that a few cases of paralysis agitans are ushered in by rheumatoid or neuralgic pains, but in the majority of cases pains are absent. The patient is, however, distressed by disagreeable sensations, such as cramps and sensations of tension and traction in the muscles, along with a general feeling of weariness and discomfort. These sensations render the patient restless, and cause him to seek frequent changes of posture. He complains of a constant sensation of *excessive heat*, although the thermometer shows that the temperature of the body is normal. In order to relieve this

feeling the patient throws off the bedclothes at night, and only retains the lightest garments in the daytime. This sensation of heat is especially felt in the epigastrium and back, but may affect the face and limbs. It is liable to remissions and exacerbations, and seems to attain its maximum after a paroxysm of trembling; it is often accompanied by profuse perspiration.

(3) *Terminal Period.*—The course of the disease is very protracted, and may extend over a period of many years. As the tremors and muscular rigidity increase in intensity, the patients are compelled to remain the whole day seated on a chair, or confined to bed. The general nutrition suffers, the muscles become atrophied, and the paresis of the second stage of the disease gives place to pronounced paralysis. The memory and intellect are weakened, general prostration sets in, the urine and feces are passed unconsciously, bedsores appear on the sacrum, and the patient dies from nervous exhaustion and marasmus. The tremors disappear entirely a few days before death, and paralytic symptoms become predominant. In the majority of cases, however, death results from some intercurrent disease, such as pneumonia or pleurisy.

II. MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD (DISSEMINATED OR INSULAR SCLEROSIS).

Etiology.—Hereditary transmission has been traced in several recorded cases of multiple sclerosis, although it is always indirect rather than direct. It is commonly observed in youth and middle age, and a considerable proportion of the cases recorded in England have been in children under ten years of age. It is probably more frequent in women than in men, but the disproportion between the sexes is probably not great. The exciting causes are exposure to cold and damp, excessive mental or bodily exertion, profound emotional disturbances and traumatic influences, as blows on the head and concussion from railway accidents.

Symptoms.—Multiple sclerosis may be divided into three varieties: (1) the *cerebro-spinal*, (2) the *cerebral*, and (3) the *spinal* form. Of these the cerebro-spinal variety is by far the most frequent and important.

(1) *The Cerebro-spinal Form.*—This form of multiple sclerosis may develop gradually and insidiously or suddenly. In cases, the development of which is gradual, the initial symptoms are very obscure, and may be referred either to the spinal cord or brain. The spinal symptoms which usher in the disease consist of paresis of the lower extremities, with a slow and trembling gait, or ataxia with various paræsthesiæ, neuralgic pains, other disturbances of sensibility in the limbs, and

cardialgic attacks accompanied by urgent vomiting. The more usual cerebral symptoms observed in the beginning of the disease are vertigo, headache, staggering gait, tremors on voluntary effort, impairment of speech, vision, or hearing, paresis of the muscles supplied by one or more of the cranial motor nerves, and various psychical disturbances. When the disease begins abruptly, the symptoms are ushered in by a convulsive or apoplectiform attack, followed by diplopia, amblyopia, nystagmus, and disturbances of speech.

The first motor symptom to attract attention is usually paresis or paralysis of certain muscular groups. Weakness generally begins in one leg, and subsequently extends to the other leg and to the arms, but the order in which the paralysis of the different muscular groups is developed presents every imaginable combination.

The gait is usually of the spastic variety, muscular contractures set in, and the legs are held like rigid bars in the position of extension and adduction, just as in primary lateral sclerosis. In the later stages of the disease flexion of the different segments of the lower extremities may predominate over extension. The paralysis rarely becomes so well marked in the upper as in the lower extremities. When the upper extremities are, however, affected with paralysis and contracture, they are maintained in a position of forced extension, and closely applied to the sides of the body. The affection sometimes begins with ataxia, but in these cases it may often be noticed that characteristic symptoms of true locomotor ataxia are absent, while others are present which do not usually belong to it.

Intermittent muscular tremor constitutes one of the most characteristic symptoms of this affection, although it has been found absent in a few isolated cases. This tremor appears almost exclusively during voluntary movements, and disappears during repose. So long as the patient remains seated quietly the tremor is either entirely absent, or at most there is only a trifling shaking movement of the head, or a slight oscillation of the trunk. As soon, however, as he attempts to seize anything with his hand the tremor begins, and increases in violence in proportion to the effort made to execute the movement. Several devices may be used in order to bring into prominence the characteristic tremor of multiple sclerosis, such as asking the patient to seize small objects with his fingers or to stretch out his arms horizontally before him.

The tremor of this affection differs from that of paralysis agitans, not only in being intermittent instead of continuous, but also in having a much wider sweep than that of the latter affection. It holds an intermediate position between the extensive jerking movements of chorea, and the small and frequent oscillations of paralysis agitans.

The true characteristic of the tremor of multiple sclerosis is best elicited by asking the patient to convey a glass of water to his mouth. As the glass is being carried to the mouth it oscillates from side to side in the patient's hand, these oscillations appearing to increase in extent and frequency as the mouth is approached. In aggravated cases the contents of the glass are spilt in every direction; but in milder cases the patient is able, by moving his head downwards in order to meet the glass, to apply it to his lips, and then the trunk, head, and arms begin to tremble violently, so that the edge of the glass rattles against the teeth, and the contents are spluttered over the patient's face. When the patient rises and attempts to walk the tremor involves the entire body, which may be shaken with such violence that he is unable to proceed or even to remain standing. As soon as the voluntary effort is relaxed the tremor diminishes, and as long as the patient is in the recumbent posture no trace of it can usually be detected; occasionally the tremor has been known to persist during repose.

The *sensory* disturbances are somewhat variable and not always well marked. They may assume the form of facial neuralgia, lancinating or diffused pains in the extremities, hyperæsthesia or anæsthesia of variable distribution, girdle pains, and various paræsthesiæ felt in different parts of the body, the latter being the most frequent of all the sensory disorders.

The *reflex* actions are variously affected in different cases. The cutaneous reflexes remain for a long time unaffected; but the tendon-reactions are usually exaggerated, especially in the lower extremities. In consequence of the increase of the tendon-reactions in the lower extremities, the knee-phenomenon and ankle-clonus are usually exaggerated, and the limbs may be thrown into the state of trembling named *spinal epilepsy*. This condition must, however, be carefully distinguished from the characteristic tremor of multiple sclerosis.

Trophic disturbances are generally wanting for a long time, but in the later stages various nutritive disorders usually make their appearance. The sclerotic nodules may encroach on the anterior gray horns of the spinal cord, and then muscular atrophy results as in progressive muscular atrophy. Muscular atrophy may present itself in the upper or lower extremities, neck, face, tongue, or indeed in any part of the body. The electrical reactions of the nerves and muscles remain normal until the muscular atrophy begins, and then the electric irritability of both becomes gradually diminished.

During the terminal period of the disease bedsores appear over the sacrum and other parts subjected to pressure, and general nutrition fails.

The *bladder and rectum*, as a rule, remain unaffected for a compara-

tively long time, but their functions are ultimately interfered with as in chronic myelitis. The disorders of the sexual functions are somewhat variable. In some patients sexual desire appears to be increased at an early period of the disease, while in others it is completely abolished. In the majority of cases the sexual functions remain normal for a comparatively long time.

Bulbar Symptoms.—Some of the phenomena caused by implication of the pons and medulla oblongata in the morbid process are amongst the most important and characteristic symptoms of the disease. The speech is slow and hesitating, while each syllable is separately pronounced, presenting a mode of articulation which has been named the *syllabic* or *scanning*. The voice is weak, low, sometimes whispering, and monotonous, while it breaks readily when forced efforts are made. Laryngoscopic examination shows that the vocal cords move normally, but their tension is diminished and frequently changes. The acts of laughing and crying are often represented by peculiar noisy inspirations. After a time symptoms of true bulbar paralysis supervene, the movements of the lips and tongue are impaired, and by-and-by mastication and deglutition become increasingly difficult, the velum palati is paralyzed, speech becomes inarticulate, and the mouth remains permanently paralyzed, while the saliva dribbles out.

Diplopia with strabismus is a not unfrequent symptom, although it may subsequently disappear as in locomotor ataxia.

Nystagmus is, however, the most important of all the ocular symptoms, being present, according to Charcot, in about half the cases. The movements of the eyeballs may be persistent or occur only during forced accommodation, or when movements are performed by the extremities. At other times the nystagmus may not be apparent during the ordinary movements of the eyeballs, but when the patient is asked to look upwards and outwards so as to strain the ocular muscles, slight oscillatory movements may be observed.

Amblyopia or *amaurosis* is a frequent symptom. Of fifty cases observed by Guanck, defects of vision were found in twenty-eight cases, and of these cases various degrees of amblyopia were present in eight, amblyopia and restriction of the field of vision with dyschromatopsia in five, and amblyopia and defects of the field of vision, with morbid changes in the optic disks, in fifteen cases. Of the fifteen cases in which the optic disks were found altered, complete white atrophy of both disks with blindness was found in two cases. Such cases are rare, the rule being that atrophy of the optic nerve in multiple sclerosis is unilateral. In ten cases partial white atrophy was observed, the change in color was strictly limited to the temporal halves of the fields of vision in four cases, while

the nasal halves were affected, but in a much less degree than the temporal halves, in six cases. In the remaining three cases hyperæmia of the disks with optic neuritis was present, caused, most probably, by a retrobulbar neuritis. The senses of smell, taste, and hearing are impaired in some cases, but these disorders are rare.

Psychical disorders are always observed in multiple cerebro-spinal sclerosis. They consist of mental irritability, emotional excitement causing the patient to laugh or to shed tears without apparent motive, and impairment of memory and intelligence. At other times the mental disorder assumes the form of distinct unsoundness of mind. In such cases there may be melancholia, monomania, with ideas of persecution or of grandeur, and the patient may fall into a state of complete dementia.

In the course of the disease, the patient suffers from attacks of vertigo. This symptom usually comes on at an early period, and continues to distress the patient throughout. The patients feel as if they themselves were being turned round, or as if surrounding objects were whirling around them. They suffer greatly from sleeplessness and violent headache.

Apoplectiform or *epileptiform seizures* have been observed in a small number of cases; they are apparently analogous to the apoplectiform attacks which occur in general paralysis of the insane. They are characterized by the development of grave cerebral symptoms, and are accompanied by a considerable elevation of temperature. After slight premonitory symptoms, such as a feeling of pressure in the head, there is a partial loss of consciousness, which in a few hours may develop into coma. The face is red and hot, the pulse is quick, and the temperature of the body rises to 104° F. or 105° F. In most cases hemiplegia with muscular flaccidity, and on rare occasions rigidity, is present from the outset of the seizure. After one or two days the temperature falls, the patient sinks into a quiet sleep from which he may be readily roused, and he feels, on awaking, comparatively well. Hemiplegia, however, persists for a few days longer, and then gradually disappears. These attacks may be repeated several times in the course of the disease, recurring in some cases every few months, but each is followed by an aggravation of the general symptoms, and death sometimes occurs during an attack.

(2) *Cerebral Multiple Sclerosis.*—In this form of the disease, which is rarely observed, the psychical disturbances are predominant. The tremor is said to precede the paralytic manifestations, but in other respects the course of the affection does not differ greatly from the cerebro-spinal variety.

(3) *Spinal Multiple Sclerosis.*—The spinal form of the affection is characterized by absence of the cerebral symptoms, particularly nystagmus, tremor on voluntary effort, vertigo, apoplectiform attacks, and psychical disturbances. The symptoms of the spinal form of multiple sclerosis often simulate those of a primary lateral sclerosis, amyotrophic lateral sclerosis, or locomotor ataxia, but in the first disease symptoms are usually superadded which do not belong to the symptoms of the classical forms of the last three diseases. The course of multiple sclerosis is somewhat variable, the average duration being from five to ten years. The first stage extends from the commencement of the symptoms up to the appearance of marked paralysis with contractures. This stage occasionally begins with cerebral symptoms, consisting of headache, vertigo, and unsteady gait, but most commonly it commences with paresis of the lower extremities like that caused by a primary lateral sclerosis, and in such cases the nature of the disease may not be recognized until the appearance of the characteristic tremor. In other cases the disease begins by an apoplectiform attack or gastralgic disturbances, while paralyses, disorders of coördination, tremor, and other symptoms are added in quick succession. The progress of this stage is often interrupted by remissions or improvements, but the nature of the disease is essentially progressive, and at the end of from two to six years the second stage of the disease is constituted by the appearance of marked and persistent paralysis with contractures. During the second stage, which lasts from four to six years or more, the nervous symptoms remain more or less stationary, and the general nutrition is but little impaired. In the third stage of the disease the patient loses his appetite and becomes emaciated, the bladder is paralyzed, cystitis and bedsores appear, and the patient dies from septic fever. In other cases death is caused by a bulbar paralysis, or by some intercurrent disease like pneumonia, pleurisy, or phthisis.

III. CHOREA.

Etiology.—Heredity plays an important part in the production of chorea, but the transmission is probably always indirect. The disease chiefly attacks children, often occurring about puberty, although children at the breast and young women are sometimes attacked. Girls are much more frequently affected than boys. Everything which augments the excitability of the nervous system during the period of sexual development, such as premature excitement of the sexual passion, onanism, or any undue emotional disturbance, increases the tendency to

chorea. But the most usual predisposing causes in adults are pregnancy and menstrual disorders, and chlorosis. That some causal relationship exists between articular rheumatism and chorea has been known since the beginning of the century, but the true nature of this relationship is not yet accurately ascertained. Out of two hundred and fourteen cases of chorea analyzed by Dr. Moury, thirty-three had suffered from rheumatic fever and twenty-three from rheumatic pains, while in nine cases the history of rheumatism was very doubtful, these figures giving from twenty-nine to thirty-two per cent. of cases in which a rheumatic history could be traced. Chorea occurs frequently after scarlet fever, a fact which may probably be explained by the frequency with which scarlet fever is followed by rheumatism. Chorea sometimes occurs during pregnancy. It is most frequent during first pregnancy, but it is sometimes repeated in the same patient in subsequent pregnancies. It appears more frequently during the first than the second half of pregnancy, but it sometimes begins in the later months, and may continue up to the time of delivery or beyond it. The majority of those affected are from twenty to thirty years of age. The disease is more prevalent in large cities than in the country, and race appears to influence the production of the disease, inasmuch as in South America the black is much less frequently attacked than the white population. Of the exciting causes of chorea, the most frequent and important are emotional disturbances, such as fright, sorrow, and discontent. Hysterical girls and those who are strongly predisposed to chorea, or who have already suffered from an attack, may acquire the disease from imitation of others suffering from it.

Symptoms.—The development of the characteristic phenomena of chorea is generally preceded for a variable period of days or weeks by various premonitory symptoms. The most usual of these are afforded by changes in the character and disposition of the patient, who becomes forgetful, inattentive, fretful, and discontented or apathetic, while the intellectual powers are impaired. The irregular spasmodic muscular contractions of chorea are not unfrequently ushered in by a slight dragging of one of the lower extremities, with a tendency to walk in a curved line and liability to let objects fall from the hand. These manifestations of the approaching disease are probably due, in some degree, to irregular muscular contractions, but are largely dependent upon muscular weakness, and in some cases paralysis may be so pronounced that the case may be mistaken for hemiplegia. The characteristic choreic movements generally begin in the small muscles of the face and in those of a hand. They consist at first of grimaces and other contortions of the face, and slight jerking movements of the fingers and at

the wrist-joint, with pronation of the forearm, when the patient is conscious of being observed or is excited from any other cause; these soon increase in intensity and persist during repose.

The irregular contractions soon extend so as to involve all the voluntary muscles, when the affection may be called *general chorea*, or they remain more or less limited to the muscles of one-half of the body, when the disease is called *unilateral chorea* or *hemichorea*.

General Chorea.—When once the disease is fully established the symptoms are quite characteristic, and it would be difficult to find phrases more expressive of the disorderly muscular movements than "insanity of the muscles," adopted by Bellingham, and "folie musculaire" by Bouillaud.

The features undergo every variety of contortion. The brow is knit and immediately expanded; the eyebrows are elevated and the next moment depressed, or one may be elevated while the other is lowered; the eyelids open and close alternately; the eyeballs are quickly rotated in different directions; and the labial commissures are suddenly drawn outwards, and as quickly retracted. These opposite movements succeed one another with such rapidity that the face presents in quick succession the most contradictory expressions, such as those of delight, vexation, and anger.

The tongue is thrust out of the mouth, and quickly retracted and rolled about from side to side; the jaws are separated and closed, it may be with so much violence that teeth are broken, or the tongue and cheeks are severely bitten; lateral displacements of the lower jaw are frequently observed, and the head is jerked suddenly from one side to the other, while the facial grimaces by which the movements of the jaws, tongue, and head are accompanied add to the comical appearance presented by the patient. The pupils are usually dilated and their reaction to light is diminished.

The superior extremities execute every variety of movement, the shoulders are elevated, then lowered, and immediately afterwards drawn backwards or forwards; the arm and forearm are moved at the shoulder- and elbow-joint in every possible direction; the hand is alternately pronated and supinated, flexed and extended; and the fingers are at one moment extended and spread apart and at the next flexed. These movements are combined in such varied ways that a gesticulatory agitation is produced which defies description.

The muscles of the trunk are implicated, and their unequal disorderly contractions produce sudden lateral and antero-posterior deviations of the vertebral column, which in certain cases may be so violent that the patient is thrown from his chair or out of bed. The muscles of the

lower extremities also undergo irregular contractions, causing eversion and inversion of the foot and various contortions of the toes, as well as movements at the larger articulations. The respiratory rhythm becomes irregular and jerky, and on laryngoscopic examination the vocal cords have been observed to act in an irregular and disorderly manner.

Choreic movements may persist during repose, and are much exaggerated during voluntary effort and when the patient is under observation or excited in any way, but they cease during sleep, and also under the influence of chloroform, in all but very aggravated cases. In cases of moderate intensity delicate manual operations, such as those required for writing, sewing, and playing upon musical instruments, become alone impossible; while operations, like eating, requiring less complicated adjustments for their performance, are still effected, although in an imperfect and roundabout manner, and after frequent interruptions from the involuntary contraction of antagonistic muscles.

In aggravated cases it becomes impossible to execute almost any intended movement. When the patient endeavors to carry anything to his mouth, such as a glass of water, the progress of his arm is arrested by a series of jerks and contradictory movements which may scatter the contents of the glass in every direction; the patient cannot button or unbutton his clothes; the maintenance of the erect posture is difficult or impossible; and even in the recumbent posture he is not free from the danger of being thrust out of bed; his clothes and linen become worn out by constant rubbing; and the skin over the prominent bones becomes erythematous and may ulcerate.

On the patient's being asked to show the tongue, he protrudes it with a jerk, the mouth being opened to an unnecessary extent; the tongue is immediately withdrawn, while the mouth and jaws close upon it with violence. When the patient endeavors to speak, the convulsive action of the facial muscles becomes aggravated; his articulation is irregular, jerky, drawling, or stammering; his voice is monotonous; and in aggravated cases his speech is so disordered as to be almost if not entirely unintelligible. Spasmodic contraction extends to the muscles of mastication and deglutition, and consequently these functions are performed imperfectly and with difficulty.

Hemichorea.—The spasmodic phenomena are sometimes limited to the muscles of one-half the body, the unilateral variety occurring in about one-fifth of all cases. Disorders of speech are usually more marked when the right than when the left side is affected with spasm, although speech is sometimes disordered when the left side is solely affected. Broadbent asserts that the muscles bilaterally associated in their actions, and which are comparatively spared in hemiplegia, are

affected to some extent on both sides in hemichorea. The other symptoms of hemichorea are the same as those of general chorea, and do not require separate description.

Although spasmodic motor disturbance constitutes the most characteristic feature of chorea, it must not be forgotten that a certain degree of muscular weakness is always present, this being easy of recognition in cases of hemichorea. Indeed, towards the termination of the affection or during its course, the choreic movements may be replaced by a more or less complete hemiplegia or paraplegia, and we have already seen that paralytic symptoms may precede the development of the characteristic movements.

The electric excitability of the nerves and muscles is said to be increased to both currents, a fact more readily proved in hemichorea than in the bilateral variety.

The reflex excitability is said by some authors to be increased and by others to be diminished.

Sensory disturbances are not frequently observed in chorea. Painful points have been found at times in the course of the nerve trunks of the affected region, while tenderness on pressure over the spinous processes of some of the vertebræ is occasionally met with. At other times cutaneous hyperæsthesia or hyperalgesia distributed over half or the whole of the body has been observed, but anæsthesia of like distribution is more common.

Vaso-motor and *secretory* disturbances are wanting, there are no special trophic changes, and the general health does not suffer, except in aggravated and chronic cases, in which the constant agitation and want of sleep induce a condition of anæmia and general marasmus.

Psychical disturbances are invariably observed in chorea. The mental depression and irritability with which the disease begins usually increase during its course. The patient is obstinate, taciturn, and even violent towards parents and attendants. He suffers from impairment of memory, incapacity for thinking, and general intellectual weakness. At times there may be hallucinations of sight, especially at night, succeeded by a maniacal delirium, and according to the observations of Marcé, half of the cases in which this delirium supervenes terminate fatally.

The pulse may be irregular, and the patient suffers from palpitation, while a physical examination of the heart generally reveals the presence of endocardial murmurs, either arising from disease of the valves or of functional origin. Chorea is not usually accompanied by pyrexia, but in severe cases, when there is violent muscular action, elevation of tem-

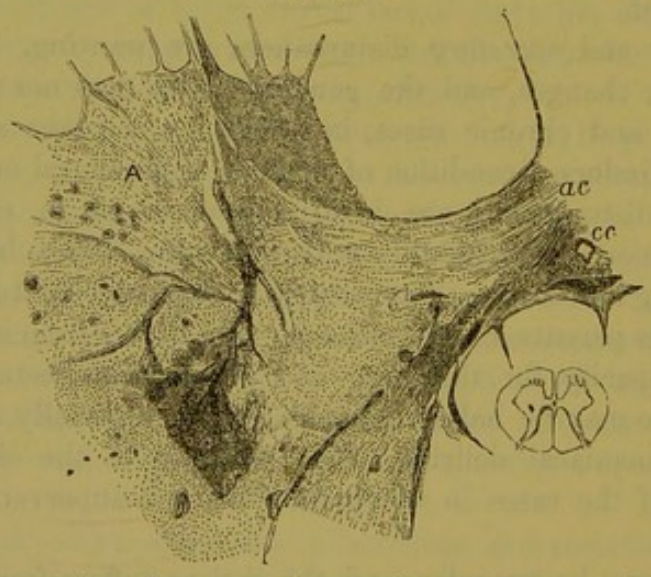
perature is not uncommon. When chorea is associated with acute rheumatism more or less fever is necessarily present.

Chorea usually runs a chronic course, the majority of cases lasting from six to eight weeks, and aggravated cases four to five months. Chorea frequently recurs, a fresh attack being excited by a slight emotional disturbance, pregnancy, or the presence of an acute disease. Chorea usually terminates in complete recovery. The disease is not often fatal in children, but a fatal termination may occur from a complication of rheumatism and endocarditis, or from cerebral symptoms, consisting of collapse with complete muscular relaxation and involuntary evacuations, and a final coma.

Morbid Anatomy.—Post-mortem investigation has not thrown much light upon the nature of paralysis agitans. The chief changes which have been discovered consist of obliteration of the central canal of the spinal cord from increase of the epithelium of the ependyma, and pigmentation of the ganglion cells of the column of Clarke, anterior horns, olivary body, and corpus dentatum of the cerebellum. Slight evidences of sclerosis were found in the lateral columns of the cord as well as in the corpora striata and the hemispheres of the brain.

Multiple sclerosis consists of a chronic inflammation which appears in well-defined nodules, and is widely distributed throughout the

FIG. 181.

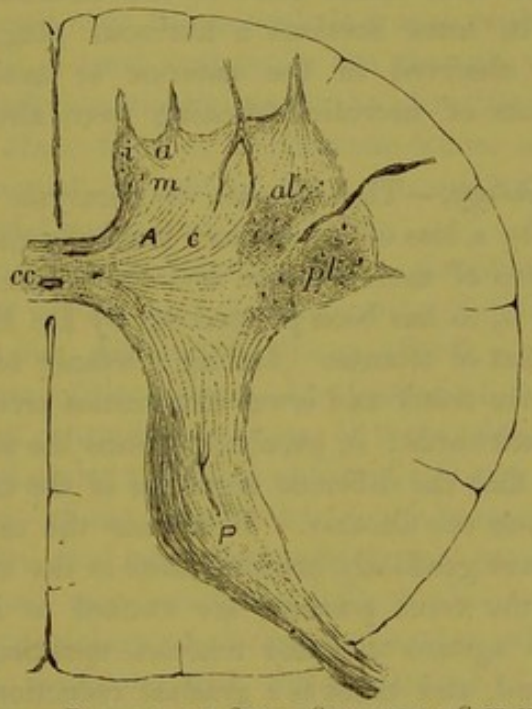


SECTION OF THE ANTERIOR GRAY HORN OF THE CERVICAL ENLARGEMENT OF THE SPINAL CORD FROM A CASE OF CHOREA THAT DIED ON THE FOURTH DAY OF SCARLET FEVER. (BURY.)
cc, Central canal; *ac*, Anterior commissure; A, Anterior horn.

nervous system. In the spinal cord these nodules vary from the size of a hemp-seed to that of a bean, but they often become confluent and appear to attain a much larger size in the brain. On section the

nodules appear gray or grayish-yellow, and when exposed to the air change to a salmon color; they are translucent or opaque, irregular or oval in shape, generally isolated and circumscribed, but occasionally confluent, and are in consistence dense, tough, even cartilaginous, but on rare occasions semifluid and gelatinous. The distribution of the nodules is very variable. On making successive transverse sections of the cord the nodules will appear in one or both the lateral columns at one level, in the posterior columns at another, in the gray substance at a third, and in the whole transverse area of the cord at a fourth. The

FIG. 182.



SECTION OF THE CERVICAL REGION OF THE SPINAL CORD FROM A CASE OF CHOREA. (YOUNG)
cc, Central canal; A and P, Anterior and Posterior horns respectively.

favorite sites in the cerebral hemispheres are the white substance of the centrum ovale, the septum lucidum, the corpus callosum, the basal ganglia, and the walls of the lateral ventricles. In the cerebellum the nodules are generally found in the white substance. The cortices of the cerebellum and cerebrum are singularly free from nodules. The anterior and posterior roots of the spinal nerves have been found diseased, and the cranial nerves, in their passage along the base of the skull, are particularly liable to be affected. The number of nodules which are present in one case is very variable, a few only being observed in some cases, while in others hundreds may be counted.

In most fatal cases of chorea which have been examined after death, small fibrinous vegetations have been found studded along the edges of

the mitral valve. Various changes have been described by different observers as having been met with in the nervous system. These changes, according to Dr. Dickinson, consist of dilatation of the medium sized arteries and veins throughout the substance of the brain and spinal cord, exudation, small hemorrhages, and patches of necrotic softening, or, in chronic cases, of sclerosis in the neighborhood of the vessels. These changes were more pronounced in the corpora striata, optic thalami, the anterior perforated spaces, and at the junction of the posterior gray horns and central column of the spinal cord (Fig. 181). In a fatal case of chorea examined by myself, all the vessels of the spinal cord were found more or less distended with red blood-corpuscles, and in some sections a fibrinous plug, which distended the vessel, was observed in the anterior or antero-lateral arteries (Fig. 182). Spots of necrotic softening were also observed in the basal ganglia.

Morbid Physiology.—The tremors of paralysis agitans are most probably caused by a loss of the balance normally existing between the regulative functions of the cerebrum and cerebellum. The attitude of paralysis agitans is, as has been pointed out by Dr. Hughlings-Jackson, the opposite of that of tetanus. During a tetanic seizure the action of the extensors of the trunk and lower extremities predominates, and the body is arched backwards; in paralysis agitans the action of the flexors predominates, so that the different segments of the trunk and extremities are flexed upon one another. In tetanus the muscles, the actions of which must have gradually predominated in the course of evolution so as to attain the erect posture, are excited to increased activity, while in paralysis agitans the same muscles, speaking broadly, become relatively paralyzed, and there is a gradual reduction of the human to the animal posture. In the stage of propulsion the attitude of the patient is very similar to that of a dog attempting to walk on his hind legs. The phenomena of propulsion are caused partly by the forced attitude of the patient and partly by the great slowness with which his movements are executed. When once the heel is raised from the ground by contraction of the muscles of the calf, the patient on getting up must balance himself on his toes, but in walking the forward inclination of the body tends to make the line of gravity pass in front of the active leg. The position is to some extent the same as that assumed by a person in running. But a healthy runner is able to take rapid and long strides with the passive leg, and by the time it is brought to the ground it is well in advance ef the line of gravity, and the body is thus kept from falling.

In paralysis agitans, however, the rigidity of the muscles prevents

the patient from taking a long stride, and the slowness with which the movements are executed renders it impossible for him to plant the leg that is about to become active in front of the line of gravity, and consequently the patient tends to fall forwards at every step. In retropulsion the line of gravity ever tends to fall behind the ball of the toe of the active leg, and the other leg cannot be moved backwards with sufficient celerity to enable the patient to place it far enough behind the retreating centre of gravity in order to arrest the backward movement. It is difficult to connect the chief symptoms of paralysis agitans with the morbid changes which have been discovered after death, but it must be remembered that these changes are not limited to the spinal cord, but are widely diffused over the encephalo-spinal centres and conducting paths. There is at least nothing in the changes which have been discovered after death incompatible with the idea that paralysis agitans is a dissolution of those nervous mechanisms whose action must have gradually predominated in passing from the quadrupedal position of animals to the bipedal position of man.

In multiple sclerosis the nodules are widely distributed over different parts of the nervous system, and the implication of different parts of the nervous system, having different functions, affords a ready explanation of the chief symptoms of the disease. Implication of the lateral columns explains the paresis, contractures, excess of the tendon-reflexes, and the phenomena grouped under the name of spinal epilepsy, of the posterior columns, the ataxia; of the anterior horns, the muscular atrophy; and of the bulbar nuclei, the labio-glosso-pharyngeal paralysis which may be present in different cases, while the student who is tolerably well acquainted with the physiology of the nervous system will be able to connect the remaining symptoms with the appearance of those nodules in different parts of the cerebrum, cerebellum, pons, and crura. Charcot attributes the characteristic tremor of multiple sclerosis to the long persistence of the axis-cylinders in the nodules of sclerosis. Conduction will take place through the nerve fibres long after they are involved in the nodules of the disease, but when once the medullary sheath is destroyed the conduction will be so retarded that the impulses from the cortex do not pass in a sufficiently quick succession to cause a continuous contraction. It has, however, been pointed out by Erb, that the characteristic tremors are absent in purely spinal cases, and that in all cases in which tremors were present during life nodules were discovered after death in the medulla oblongata, pons, crura cerebri, and other parts of the brain. It would seem, therefore, that in this disease again the tremors are caused by a loss of balance between the regulative functions of the cerebrum and cerebellum.

In chorea the lesions are supposed, by Broadbent and Hughlings-Jackson, to be localized in the corpus striatum, optic thalamus, and cortex of the brain, but they are still more widely distributed over the nervous system. It was first suggested by Kirkes that chorea is caused by multiple embolism of the vessels of the brain and spinal cord, produced by fibrinous particles being washed off from the valves of the heart after endocarditis. This theory, which has been elaborated by Dr. Hughlings-Jackson and others, has many facts in its favor, and now there can scarcely be a doubt that chorea is often caused by multiple embolism. Choreiform movements have, indeed, been produced in animals by Dr. Angel Money, by injecting inert powders into the circulation. At the same time, it must be remembered that chorea may occur in persons who are free from suspicion of a cardiac complication, and it seems likely, therefore, that the affection may be caused by other conditions besides multiple embolism. It seems likely, indeed, that the symptoms of chorea may be caused in the children of neuropathic families by conditions which lower the nutrition of the nervous system.

Treatment.—Paralysis agitans being a progressive disease, its treatment can only be palliative. The internal remedies which have been used are carbonate of iron, chloride of barium, hyoscyamus, strychnine, ergot, belladonna, quinine, zinc, nitrate of silver, chloride of gold, arsenic, and phosphorus, and of these probably the last two are the most deserving of trial. Morphia and other narcotics are necessary adjuncts of treatment in the later stages of the disease, when the patient is harassed by restlessness and sleeplessness, and both chloral and bromide of potassium may then be of use.

Multiple sclerosis must be treated on the same general principles as chronic myelitis. The most promising method of treatment appears to be the persistent application of the galvanic current to the spine, hydropathy, nitrate of silver, phosphorus, cod-liver oil, and nourishing but unstimulating diet. The greatest improvement that I ever saw take place in this disease was in the case of a man who, acting on my advice, changed the sedentary occupation of a clerk in a factory for that of a farmer.

Chorea should be treated by the careful regulation of the diet, and by the removal of every source of reflex irritation, such as intestinal worms. If anæmia be present, iron may be given, and if rheumatism complicates the case, salicylate of sodium must be administered, while the hot vapor bath may be of service. The child should in all cases be removed from school, and all intellectual work ought to be suspended. Of internal remedies, arsenic and zinc are the most useful. Arsenic is best given in the form of the ordinary liquor arsenicalis, and of this

preparation the dose should be from two to five minims gradually increased up to from five to ten minims three times a day, according to the age of the patient. The sulphate is the most convenient salt of zinc to employ. A dose of two or three grains may be given at first, three times a day, and the dose should be gradually increased daily until the almost incredibly large quantity of from fifteen to twenty grains is taken three times a day. If nausea or vomiting is produced, the dose should be slightly diminished for a few days until tolerance is established. Bromide of potassium or chloral hydrate are useful when psychical disturbances and sleeplessness are prominent symptoms, but they do not appear to exercise a decided influence on the course of the disease. At the Cheadle Convalescent Hospital Dr. Wausburgh Jones is having marked success in the treatment of chorea, by rest in bed, seclusion, overfeeding, and massage, or by the S. Weir-Mitchell treatment.

CHAPTER XXI.

GENERAL DIAGNOSIS.

Before bringing the work to a close, it will be desirable to make a few remarks on diagnosis, and although much of what we shall have to say will be a mere repetition, yet it will prove useful to discuss the general principles of this important subject disencumbered from the details of clinical descriptions. The diagnosis of the diseases of the nervous system may be divided into: 1, the clinical; 2, the *topographical;* and 3, the pathological diagnosis.

1. CLINICAL DIAGNOSIS.

With the view of arriving at a satisfactory clinical diagnosis, which must form the basis of the other kinds of diagnosis, the patient must be subjected to a methodical examination. The various tests which have already been described must be systematically applied in order to discover the sensory, motor, vaso-motor, trophic, and psychical disturbances which may be present. At the end of the examination the observations which have been made are systematized, when it is concluded that the patient is suffering from local anæsthesia, paranæsthesia, hemianæsthesia, local spasms, monospasms, unilateral convulsions, general convulsions, atrophic paralysis, spasmodic paralysis, paraplegia, hemiplegia, and monoplegia, either separately or in various combinations. It should also be noted whether the disease is, in its mode of onset, fulminant, acute, or chronic, and its origin idiopathic, traumatic, syphilitic, or arises from any other cause. Our knowledge of many diseases is, indeed, so slender that they admit of only a clinical diagnosis. When, for example, general convulsions occur at irregular intervals and in association with various emotional manifestations, we call them hysteria; when they recur at regular intervals in the absence of such manifestations, we call them epilepsy; when they occur as an acute affection without showing a tendency to periodical recurrence, we call them eclampsia, these being subdivided according to the cause of the symptoms into toxic, puerperal, uræmic, febrile, and reflex convulsions; and when no cause for them can be assigned, we call them idiopathic convulsions.

2. TOPOGRAPHICAL DIAGNOSIS.

In a large number of cases we are enabled to pass readily from the clinical to the topographical diagnosis. In order to determine the locality of the lesion, it is all important to remember the course of the sensory conducting paths from the periphery up to their termination in the sensory centres of the cortex of the brain, and of the motor conducting paths from their origin in the motor centres of the cortex to their termination, first in the spinal nuclei and then in the various muscles. In reference to the sensory symptoms, it may be said that when the disorder is more or less limited to the area of distribution of the afferent branches of a particular nerve the lesion is situated in the peripheral course of the nerve, when it is distributed pretty uniformly over the lower half of the body and the lower extremities the lesion is situated in the spinal cord, and when it is distributed over one lateral half of the body the lesion is situated in the brain, the posterior third of the posterior segment of the internal capsule being the part usually affected. Similar remarks apply to the motor symptoms. When a paralysis, for example, is limited to the muscles supplied by the branches of a particular nerve, the lesion is likely to be situated in the peripheral course of the nerve; when the paralysis is pretty uniformly distributed over the lower half of the body the lesion is situated in the spinal cord, and when it is restricted to a lateral half of the body the lesion is localized in the brain. The division of the various forms of loss of motor power into atrophic and spasmodic paralyses is a most important one for diagnostic purposes. In atrophic paralysis the lesion is situated in the anterior gray horns of the spinal cord, in the fibres which connect the ganglion cells of these horns with the muscles, or in the affected muscles themselves. In the spasmodic paralyses the lesion is situated in the motor centres of the cortex or in the conducting paths which connect these centres with the motor ganglion cells of the anterior gray horns of the spinal cord. Loss of the reflexes indicates, as a rule, that the lesion is situated in the spinal reflex loops, while excess of them generally shows that it is situated in the motor centres of the cortex, or in the conducting paths which connect these with the spinal nuclei. General symptoms like headache, vomiting, optic neuritis, incoherence, delirium, and loss of consciousness, indicate that the brain is implicated, but they are of no further value in localizing the disease. Before proceeding further with our subject, it will be well to mention some general difficulties which present themselves in localizing the lesion. The first is that the degree of the symptoms does not

by any means always correspond to the size and extent of the lesion. A hemorrhage may, by suddenly rupturing some of the fibres of the conducting paths and compressing others, give rise to sensory and motor disorders out of proportion to the extent of the lesion, while a slow growing tumor may, by gradually displacing and stretching the fibres of the conducting paths, come to occupy the usual situation of these paths without causing but a slight degree of motor or sensory disorder. It may, indeed, be laid down as a general rule that acute lesions are often much smaller, and chronic lesions much larger than the degree and character of the symptoms would lead us to expect. The second difficulty is that a lesion not only causes direct symptoms from irritation or destruction of the nervous tissues in its immediate vicinity, but often produces marked indirect symptoms by excitation or inhibition of remote parts. Goltz, for example, found that section of the spinal cord in the cervical region in dogs causes immediate paralysis of the power of erection of the penis, but if the animal survive for a few days this function recovers, and an erection may be obtained by reflex excitation. Indirect symptoms, having a similar import, are frequently met with in disease, and this is particularly apt to be the case when the lesion is situated in the brain. A small irritative lesion, when situated in the cortex of the brain, may set up widely diffused discharges, and thus give rise to extensive unilateral spasms or even to general convulsions. A small hemorrhage in the substance of the brain may, partly through the shock communicated to the whole organ from the suddenness of the onset, and partly from the compression it exercises upon the nervous tissues by encroaching upon the volume of the cranial cavity, suddenly arrest all the functions of the organ, so that the patient lies in an unconscious condition, and with almost complete muscular relaxation. When the indirect symptoms are so predominant as in the example just given, it will not be found possible to recognize the direct symptoms or to interpret their significance. But in most cases the indirect symptoms tend to pass off in a few days, and then the localization of the lesion may be determined. Having come to the conclusion that the lesion is situated in the brain, the case must now be approached with the view of determining its exact seat. Ordinary hemiplegia, when persistent, is caused most frequently by disease of the anterior two-thirds of the posterior segment of the internal capsule or of the area of the lenticulo-striate artery. In the most usual form of hemiplegia the arm is more paralyzed than the leg, but in those comparatively rare cases in which the leg is more paralyzed than the arm, the motor paralysis is unaccompanied by hemianæsthesia, and the lesion is then situated in the posterior two-thirds of the pos-

terior segment of the internal capsule or in the area of the lenticulo-optic artery. In some cases the arm is more paralyzed than the leg, and yet the hemiplegia is accompanied by hemianæsthesia, and the lesion is then situated in the optic thalamus and partially involves the posterior part of the internal capsule. If the hemiplegia is transient, the lesion is situated near the motor tract without causing permanent damage to its fibres, most commonly in the lenticular nucleus. Monoplegiæ are caused by lesions in the motor area of the cortex, or in the centrum semiovale between the motor centres and the internal capsule. If the paralysis in such cases be preceded or accompanied by unilateral convulsions, the lesion is situated in or near the cortex. Monoplegiæ caused by disease of the motor cortex or of the underlying white substance, are often accompanied by considerable diminution of the muscular sense and cutaneous sensibility of the affected limb. The exact situation of the disease when the cortex or underlying white substance is its seat, may be determined by reference to Ferrier's diagrams and Pitre's vertical sections of the cerebral hemispheres. In persistent motor aphasia the lesion is situated in the posterior part of the third frontal convolution or in the white substance immediately underlying it; but if recovery takes place in a few weeks or months, it is in or near the knee of the internal capsule. In sensory aphasia the lesion is situated in the angular gyrus or first and second temporo-sphenoidal convolutions, while in mixed motor and sensory aphasia, which is accompanied by hemiplegia, it is widely distributed over the motor area of the cortex. A transient aphasia may precede an attack of migraine headache, or follow a minor attack of epilepsy. Hemianæsthesia, with implication of the special senses, is caused by disease of the posterior part of the internal capsule. In some of these cases the visual affection consists of an amblyopia of the eye on the opposite side to the lesion, whilst in others it consists of a bilateral hemianopsia. In hemianæsthesia, with bilateral hemianopsia, the lesion, which is always situated in the optic thalamus, reaches sometimes so far back as to implicate the external geniculate body, but at other times it is situated opposite the middle part of the posterior segment of the internal capsule, and much too far forwards to reach that body. A tumor in any of the basal ganglia may cause hemianopsia by compression of the underlying optic tract. Homonymous hemianopsia may also be caused by a lesion in the white substance or in the cortex of the occipital lobe. Cortical or centrum ovale hemianopsia may be suspected rather than tract hemianopsia if the blind is not separated from the sensitive area of each retina by a sharply defined straight line, if a quadrant only of each field is lost, or if there is a considerable restriction of the pre-

served halves of the fields of vision, especially on the side opposite the lesion. When the lesion is situated in the left hemisphere the hemianopsia is often accompanied by word-blindness. In convulsive attacks which begin with a visual aura consisting of colored vision or hallucinations of sight, it may be inferred that an irritative lesion is situated in or near the cortex of the occipital lobe. If the convulsion begins with rattling noises in the ears or hallucinations of sound, an irritative lesion is situated in or near the cortex of the first or second temporo-convolutions.

It is possible that a destructive lesion in this area may give rise to some degree of deafness of the ear on the opposite side, but this sign is not to be much depended upon. If the lesion, however, is situated in the left hemisphere the patient suffers from word-deafness, which is often accompanied by loss of smell in the nostril on the side of the lesion from softening of the external root of the olfactory tract. If a person who is suffering from word-deafness suddenly becomes absolutely deaf without there being any evidence of local disease of the ears, there are symmetrical lesions in both temporo-sphenoidal lobes on a level with the first and second convolutions.

Lesions in the remaining parts of the cortex of the temporo-sphenoidal lobe and in the underlying white substance do not give rise to definite localizing symptoms. A tumor of this lobe may by compressing the optic tract give rise to hemianopsia, and the internal capsule to hemiplegia. The Island of Riel is, as we have seen, the embryonic area of the cortex, and lesions of it are often unaccompanied by any localizing symptoms, but when situated near the junction of this lobe with the operculum they give rise to a motor aphasia. But to return to the internal capsule; disease of the anterior segment does not give rise to any definite symptoms. Charcot found softening of this segment in a case in which there was considerable psychical disturbance during life. Disease of the cortex of the prefrontal area of the brain with which the anterior segment of the internal capsule appears to be connected, is also often found in cases in which there had been considerable mental disorder during life. If a patient with the general symptoms of a cerebral disease lies in a somnolent condition without giving evidence of localized sensory or motor disorder, the lesion is most probably situated either in the cortex or the white substance of the prefrontal lobe. In cases of unilateral convulsions which begin by spasm of the side of the face, if loss of consciousness precedes the spasm, the lesion is most probably situated in the prefrontal lobe.

If paralysis of the limbs on one side is associated with paralysis of one of the cranial nerves on the other, the lesion is situated in or near

the pyramidal tract of the side opposite the paralyzed limbs somewhere in its course between the internal capsule and the crossing in the medulla oblongata. When the third nerve of one side and the limbs on the other are simultaneously paralyzed, the lesion is in the crus cerebri; when the fifth is the cranial nerve affected, the lesion is situated about the middle of the pons; when the sixth or seventh, the seat is about the lower or posterior end of the pons; and when the twelfth, it is localized in the medulla, but the last occurs only very rarely. In apoplectic lesions of the upper part of the pons the pupils are often strongly contracted from irritation of the nuclei of the third nerve. In partial anæsthesia of one side of the face and anæsthesia of the trunk and limbs on the opposite side, the lesion is situated in the restiform body in or near the ascending root of the fifth nerve. Unilateral lesions of the pons implicate the transverse fibres coming from the middle peduncle of the cerebellum, and consequently give rise to a tendency to fall to the side of the lesion.

Disease of the basal ganglia do not give rise to very definite symptoms, except when they are sufficiently large to compress or rupture the fibres of the internal capsule. If a patient has suffered from a slight apoplectic attack with right hemiplegia and aphasia, from which recovery takes place in a few weeks leaving only a slight exaggeration of the tendon reflexes at the wrist, the lesion is most probably a hemorrhage limited to the left lenticular nucleus. If the same patient has, some time afterwards, a second apoplectic attack with left-sided hemiplegia and aphasia, from which recovery also takes place in the course of a few weeks, leaving, in addition to a slight exaggeration of the tendon-reflexes at the left-wrist, bulbar symptoms like those of progressive labio-glosso-pharyngeal paralysis, both lenticular nuclei will most probably be found to have been destroyed by hemorrhage. Lesions strictly limited to the caudate nucleus or to the inner half of the optic thalamus cause no localizing symptoms. If, however, a person has a slight apoplectic attack which is at first attended with little or no loss of consciousness, and becomes after a few minutes or hours suddenly profoundly comatose with complete resolution of all the limbs, or paralysis of the limbs on one side and spasmodic contraction of those on the other, dilated and fixed pupils, and a great initial lowering followed by a rapid rise of temperature, a hemorrhage of the caudate nucleus or of the inner part of the optic thalamus has ruptured into the general ventricular cavity.

When an apoplectic attack causes hemiplegia with hemianæsthesia, which is followed by post-hemiplegic chorea or athetosis, the lesion is situated in the area of the posterior external artery of the optic thalamus, but it is not known whether the mobile spasms are caused by the damage

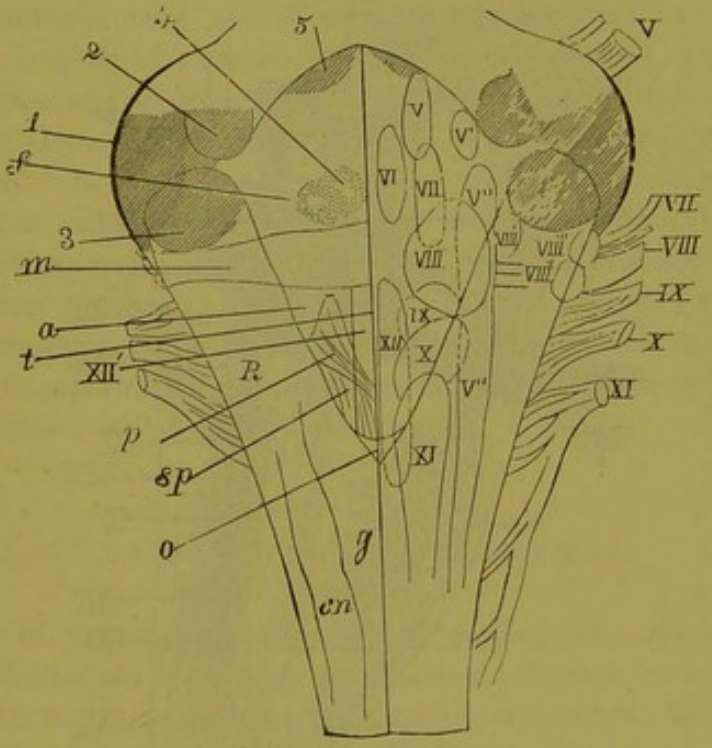
done to the ganglion itself or by partial injury of the internal capsule. Bilateral athetosis and the choreic movements which attend some of the spasmodic paralyses of infancy are caused by a parencephalus or unilateral atrophy of the motor area of the cortex of the brain. If a severe apoplectic attack, followed by a prolonged period of partial unconsciousness and death after a few weeks, is attended by hemiplegia and hemianæsthesia, rotation of one eyeball downwards and outwards and of the other upwards and outwards, dilating pupils on exposure to light, and paroxysms of Cheyne-Stokes breathing, the lesion will be found to have been a hemorrhage which has completely destroyed the whole of the pulvinar of the optic thalamus with the posterior part of the internal capsule, and to have made its way inwards so as to be only separated from the cavity of the third ventricle by a thin layer of tissue, and backwards so as to have undermined and partially destroyed the anterior tubercle of the corpora quadrigemina. Lesions which are more or less limited to the corpora quadrigemina do not probably give rise to distinctive symptoms, and the same remark applies to lesions of the corpus callosum, although cases of tumor in this situation have been recorded by Erb and Bristow.

Loss of one or more of the functions of the cranial nerves suggests the possibility of an intracranial growth at the base of the brain, or a basal meningitis. Complete loss of smell with the presence of double optic neuritis, or other symptoms of a chronic intracranial disease, indicates a growth in the anterior fossa of the skull. Complete loss of sight of one eye, with atrophy of the optic disk and paralysis of the oculo-motor nerves and of the ophthalmic division of the fifth, indicates most probably a chronic meningitis with much thickening around the optic foramen and temporo-sphenoidal fissure. If, in addition to these symptoms, there is temporal hemianopsia of the other eye, the lesion is a tumor situated over the cavernous sinus and coming as far forwards as the optic foramen. The presence of double temporal hemianopsia shows pressure on the centre of the optic commissure either by tumor of the sella Turcica or rarely by distention of the third ventricle by fluid. As the lesion increases in size complete blindness may result, but most frequently it grows to one side and gives rise to blindness of one eye, with temporal hemianopsia of the other.

When hyperæsthesia or anæsthesia in the area of distribution of the fifth nerve is accompanied by neuroparalytic ophthalmia, with or without masticatory paralysis, the lesion is likely to be situated in the middle fossa of the skull, and when both third nerves are paralyzed in association with some degree of single or double hemiplegia, and the general symptoms which indicate an intracranial growth or abscess, the lesion

is situated in the interpeduncular space. When the fifth and sixth, the seventh and eighth, or the eleventh and twelfth nerves are simultaneously paralyzed, the lesion is likely to be in the posterior fossa of the skull rather than in the substance of the pons and medulla oblongata. When a tumor is situated near the cortex of the brain the skull is often tender to percussion at the the point at which the growth comes nearest to the surface.

FIG. 183.



VIEW OF THE POSTERIOR SURFACE OF THE MEDULLA (after ERB),

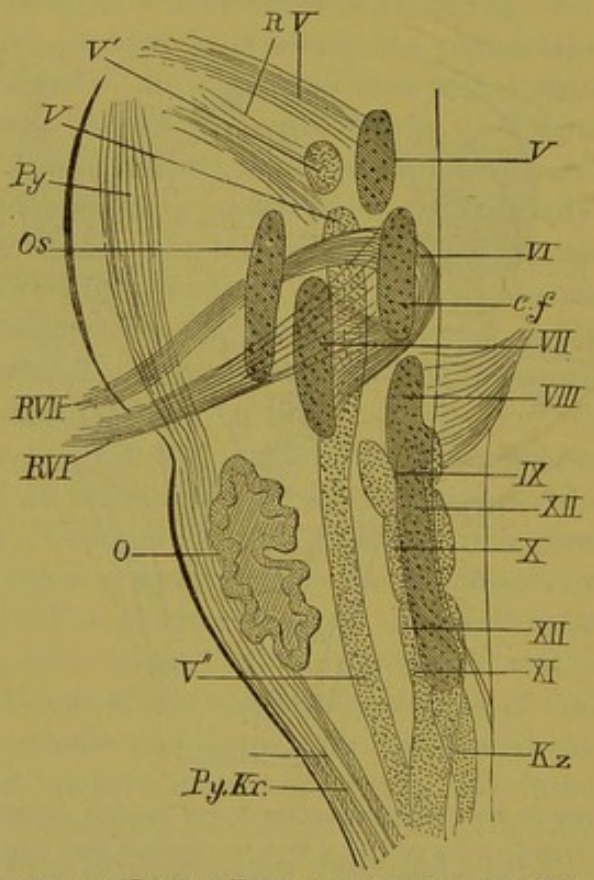
the roof of the fourth ventricle being removed to show the rhomboid sinus clearly. The left half of the figure represents: Cu, funiculus cuneatus, and *g*, funiculus gracilis; O, obex; *sp*, nucleus of the spinal accessory; *p*, nucleus of pneumogastric, *p* + *sp*, ala cinera; R, restiform body; XII′, nucleus of the hypoglossal; *t*, funiculus teres; *a*, nucleus of the acousticus; *m*, striæ medullares; 1, 2, and 3, middle, superior, and inferior cerebellar peduncles respectively; *f*, fovea anterior; 4, eminentia teres (genu nervi facialis); 5, locus cœruleus. The right half of the figure represents the nerve nuclei diagrammatically; V, motor trigeminal nucleus; V′, median, and V″, inferior sensory trigeminal nuclei; VI, nucleus of abducens; VII, facial nucleus; VIII, posterior median acoustic nucleus; VIII′ anterior median, VIII″ posterior lateral, VIII‴ anterior lateral acoustic nuclei; IX, glosso-pharyngeal nucleus; X, XI, and XII, nuclei of vagus, spinal accessory, and hypoglossal nerves respectively. The Roman numerals at the side of the figure, from V to XII, represent the corresponding nerve roots.

When the lesion is situated in the spinal cord below the crossing of the pyramidal tracts, it gives rise to a spasmodic paralysis of all muscles innervated from below the level of the disease, as well as to more or less anæsthesia of the inferior part of the body. The muscles supplied from the level of the lesion suffer from atrophic paralysis, the reflexes

of the affected segment of the cord are abolished, and a line of hyperæsthesia passing round the body generally separates the anæsthetic and sensitive areas.

In the various forms of atrophic paralysis the situation of the lesion must be inferred from the grouping of the affected muscles. In ophthalmoplegia externa the lesion is situated in the nuclei of the third

Fig 184.



Transparent Lateral View of the Medulla (after Erb),

showing the relative positions of the most important nuclei; right half of the medulla, seen from the surface of section; the parts that lie closer to this surface are deeper shaded. Diagrammatic. Py, pyramidal tract; Py, Kr, decussation of pyramids; O, olivary body; Os, superior olivary body; V, motor, V′, middle sensory; V″, inferior sensory nucleus of trigeminus; VI, nucleus of abducens; c.f, genu facialis nervi; VII, nucleus facialis; VIII, posterior median acoustic nucleus; IX, glosso-pharyngeal nucleus; X, nucleus of vagus; XI, nucleus of the accessorius; XII, hypoglossal nucleus; Kz, nucleus of the funiculus gracilis; Rv, trigeminus root; RVI, root of the abducens; RVII, root of facialis

and sixth nerves; in progressive labio-glosso-laryngeal paralysis and the acute form of the affection caused by occlusion of a branch of the basilar artery, the lesion is situated in the inferior part of the floor of the fourth ventricle (Figs. 183 and 184); and in the usual forms of acute atrophic spinal paralysis of infants and adults the lesion is situated in the anterior gray horns of the spinal cord, the exact level of the

lesion in the longitudinal extent of the cord being indicated by the particular muscles attacked as well as by the reflexes which are found abolished.

When an atrophic paralysis is restricted to the muscles supplied by a particular nerve, and when there is at the same time more or less anæsthesia in the area of distribution of the sensory branches, the lesion is situated in the peripheral course of the nerve. In atrophic paralysis from disease of nerves the muscles are affected according to the distribution of the branches of the diseased nerve, but in spinal atrophic paralysis they are affected in groups according as they are associated in their functions. If the motor disorder consists of an incoördination and not of a spasm or paralysis, the disease is likely to be either tabes dorsalis, Ménière's disease, sclerosis in patches, or a cerebellar lesion. In tabes the patellar tendon-reflexes are absent, and the gait is ataxic; in Ménière's disease the patient is deaf and suffers from whistling noises in the ears; in multiple sclerosis the patellar tendon-reactions are generally exaggerated, and the patient manifests the tremor, scanning speech, and other characteristic phenomena, while in cerebellar disease the patellar tendon-reactions are generally exaggerated, and the gait is a reel and not a true ataxia.

3. PATHOLOGICAL DIAGNOSIS.

The aim of pathological diagnosis is to determine the *nature* of the lesion no matter where it may be situated. It may be laid down as a general rule, but one requiring numerous qualifications in practice, that the diseases which develop the symptoms of depression in the course of a few minutes or a few hours, and without being set up by traumatic or toxic causes, are of *vascular* origin; that the diseases which take from a few days to a few weeks for the full development of the symptoms, and in which the symptoms of depression are preceded by those of irritation, are of inflammatory origin; and that the diseases which take from two to six months for the full development of their symptoms, and in which the primary symptoms of irritation are either absent or obscured by the more prominent symptoms of depression, are of *degenerative* origin, or are due to a slow and gradual compression of nervous tissue by the growth of a new formation.

(1) *Vascular Lesions.*—The vascular lesions which are the most frequent causes of disease of the nervous system are rupture with hemorrhage, embolism, and thrombosis. If a patient suddenly becomes profoundly comatose without premonitory symptoms, and with or with-

out evidence of the presence of hemiplegia, the lesion is more likely to be a hemorrhage rather than embolism or thrombosis. If the coma passes off in a few hours or days without leaving a trace of hemiplegia or other grave symptoms, the lesion has probably been a cerebral congestion, but if the coma has been somewhat prolonged, and if hemiplegia persists, the lesion has been a hemorrhage. Hemorrhage is to be suspected rather than embolism if an apoplectic attack occurs during the degenerative period of life or after forty-five years of age; if the arteries at the wrist and temples are hard and knotty; if the left ventricle of the heart is hypertrophied; if the second sound of the heart is much accentuated at the base; if the urine is abundant and of low specific gravity, and if it contains a small quantity of albumen; if the arterial tension is high; and if a well-marked arcus senilis and other signs of degeneration of tissue are present. Ingravescent apoplexy is almost always caused by hemorrhage, and when this form of hemorrhage occurs in comparatively young people it may be suspected that an aneurism has burst. If an apoplectiform attack, with or without hemiplegia, occur in a young or middle-aged person who is suffering from valvular disease of the heart, the lesion is likely to be embolism. This supposition is strengthened if the onset of the disease was not attended by a profound loss of consciousness, and if a more or less persistent aphasia is established. Embolism is, indeed, more probable than hemorrhage if the attack has supervened during the course of acute rheumatism, chorea, or scarlet fever, even when no signs of valvular disease can be discovered. Valvular disease may give rise to an aneurism of a cerebral vessel and to subsequent rupture, and consequently the signs of cardiac disease are only of value in determining the question of embolism or hemorrhage when taken in conjunction with other symptoms.

Thrombosis of the arteries of the brain occurs in old age from atheroma and in middle age from syphilitic endarteritis.

Occlusion of a cerebral artery by thrombosis gives rise to an apoplectic attack like that caused by an embolus, but in the former the attack is preceded by premonitory symptoms which are wanting in the latter, and the attack itself is often more gradual in its development in thrombosis than either in embolus or hemorrhage. The premonitory symptoms of thrombosis consist of headache, dizziness, loss of memory, and general confusion, together with numbness and formication of one of the limbs or of one-half the body. Syphilitic thrombosis is often preceded by severe headache with nocturnal exacerbations, while other evidences of syphilis may be obtained.

If an apoplectic attack supervenes in the puerperal state, or in the course of exhausting diseases like phthisis and cancer, and in children

after severe diarrhœa, the cause is most likely a thrombosis of one of the cerebral veins or sinuses. It is also probable that the condition known as asphyxia neonatorum, and which is followed by a spasmodic paralysis with idiocy, is caused by thrombosis of one of the cerebral veins occasioned by injury to the head during delivery.

Hemorrhage of the spinal cord occurs in comparatively young subjects. It is liable to be mistaken for acute spinal atrophic paralysis, from which it may be distinguished by the fact that the atrophic paralysis caused by the local lesion is in hemorrhage accompanied by sensory disorders and by spasmodic paralysis caused by implication of the posterior horns and the sensory and motor conducting paths.

(2) *Inflammatory Lesions.*—The inflammatory nature of the lesion must be determined by the mode of onset being more gradual than in vascular lesions; the symptoms of depression being preceded by those of irritation, and the presence during the development of the disease of elevation of temperature and other febrile symptoms. When once it is concluded that the lesion is inflammatory the further classification of the disease must be determined from the grouping of the symptoms and the localization of the lesion.

(3) *Degenerative Lesions.*—It is difficult to draw any definite line of demarcation between the secondary degenerations, which result from chronic inflammation, and those diseases which are of degenerative origin from the commencement. The development of the disease may be chronic and progressive in both, and the chief reliance in diagnosis must be placed upon the presence of symptoms of irritation and slight elevation of temperature in the former and their absence in the latter.

Multiple cerebro-spinal sclerosis is a chronic degenerative disease, probably of inflammatory origin, which, from the variability of its symptoms, is liable to be mistaken for various other diseases. It would be difficult to believe that paralysis agitans and multiple cerebro-spinal sclerosis were confounded with one another, were it not a matter of history that the two diseases were only separated from one another clinically for the first time by Charcot only a few years ago. Multiple sclerosis is a disease of youth and middle age, and paralysis agitans most commonly of advanced age. The tremor of paralysis agitans consists of fine rapid oscillations, which persist during repose; it may be temporarily arrested by voluntary effort, and seldom implicates the muscles of the head; while the tremor of multiple sclerosis has a somewhat extensive sweep, ceases during repose, is excited or aggravated by voluntary movements, and almost always implicates the muscles of the head. In paralysis agitans decided loss of motor power is not developed until long after the appearance of tremor, but in multiple sclerosis

paralysis precedes or soon follows the tremor. The nystagmus, scanning speech, and other cerebral symptoms of multiple sclerosis are wanting in paralysis agitans.

Multiple sclerosis, especially in its early stages, may be mistaken for some one or other of the system diseases of the spinal cord, such as locomotor ataxia, primary lateral sclerosis, amyotrophic lateral sclerosis, or progressive labio-glosso-laryngeal paralysis, and it is not always possible to be sure of the diagnosis until symptoms like nystagmus, scanning speech, and tremor appear in the course of the disease, which are more or less characteristic of multiple sclerosis, and never form part of the system diseases. The hereditary form of locomotor ataxia described by Friedreich is probably more allied to multiple sclerosis than to the tabes dorsalis of adults. Mercurial tremor is sometimes so like a moderately advanced case of multiple sclerosis that the former can only be recognized from the latter by the history of exposure to its cause. Hysterical tremor persists during repose, provided the patient is conscious of being observed; it may disappear for a long time and then recur, and general hysterical symptoms are usually present. The disorderly movements of chorea differ considerably from the tremor of multiple sclerosis, but the diagnosis is not always easy when, as may occasionally happen, choreiform movements complicate those proper to multiple sclerosis.

The early stage of general paralysis of the insane may very readily be mistaken for the early stage of multiple sclerosis. The hesitating and indistinct speech of general paralysis is very like the scanning speech of multiple sclerosis, and the tremors of the tongue and angle of the mouth and of the hands in the former, may be mistaken for the tremor which is so characteristic of the latter disease; while the patellar tendon-reactions may be either exaggerated or absent in both diseases. The age at which patients are attacked affords some help in diagnosis, multiple sclerosis being most common in the first three, and general paralysis in the fourth decade of life. Inequality of the pupils and loss of the light-reflex are common in general paralysis, and probably never occur, especially together, in multiple sclerosis. When the characteristic expansive delirium with grand ideas, of general paralysis is well marked, the diagnosis is easy, and even in the many cases in which it is absent the patient has a peculiar air of contentment and self-satisfaction which is wanting in multiple sclerosis. The diagnosis becomes more easy with the advance of these diseases. In general paralysis the patient manifests distinct signs of loss of memory and decided mental failure, while in multiple sclerosis the mental condition is generally one simply of emotional excitability, and only rarely of pronounced mental failure,

and in these occasional cases other characteristic symptoms, like nystagmus and a widely diffused tremor, are present to make the nature of the disease sufficiently clear.

(4) *New Formations.*—When a patient suffers from a severe and persistent headache, dizziness, and vomiting, the existence of an intracranial growth ought to be suspected, and the presence of double optic neuritis places the diagnosis almost beyond question. The localizing symptoms met with in cases of cerebral tumor are the same generally as those caused by hemorrhage and focal diseases, but these symptoms creep on gradually and progressively in tumor, and not suddenly as in the vascular diseases. Paralysis and other localizing symptoms may, however, occur in the course of a tumor from a complicating hemorrhage, or œdema of the brain, or occlusion of a vessel from compression.

When once it is concluded that a foreign body is encroaching upon the cranial cavity, the next question to be determined is the *nature* of this growth. If the symptoms of an intracranial growth are found in a young person of tubercular diathesis, and especially if the presence of cavities in the chest, of enlargement of glands, or of a chronic discharge from the ear indicate tubercular disease of other organs, then the cerebral disease is likely to be a *tubercular* tumor. This supposition is strengthened if the symptoms indicate that the tumor is situated in the pons, cerebellum, or cortex of the brain; if the progress of the case is somewhat rapid; and if there are signs of the presence of multiple lesions.

If the symptoms of an intracranial growth, indicating the same localization as that of the tubercular tumor, occur in an otherwise healthy young person who is free from syphilitic taint, the tumor is likely to be a *glioma*. This supposition will be strengthened if the development of the symptoms had been preceded by an injury to the skull, and if the progress of the disease is comparatively slow. If the chief symptom of an intracranial growth is caused by compression of the nerves at the base of the skull, the tumor is likely to be a *sarcoma*. This supposition is strengthened if the progress of the disease is slow and progressive, and the diagnosis is rendered almost certain if a sarcomatous growth is discovered in any other organ. If a patient about middle age suffering from the symptoms of an intracranial growth, has a cachectic appearance, if the disease makes rapid progress, and, above all, if there is evidence that cancer is deposited in other organs, then the growth is likely to be cancer.

If the symptoms of an intracranial growth are attended by marked psychical disturbances, and convulsions, which are at first unilateral and occasionally become general and frequently repeated near the fatal

termination, and if these symptoms occur in a butcher or pork-dealer, or in one who has suffered from tapeworm, then the tumor is likely to be *cysticercus cellulosæ*. The diagnosis of the presence of *echinococci* in the brain must be made from the general symptoms of intracranial tumor appearing and disappearing alternately, œdema of the eyelids, an opening in the cranial bones through which a fluctuating tumor projects, or exploratory puncture.

Abscess of the brain occurs as the direct consequence of an injury, such as fracture of the skull and contusion of the brain, or is associated with some other disease, such as caries of the petrous portion of the temporal bone, and ozæna, and the presence of suppuration is often indicated by rigors and fever. A cerebral abscess may remain more or less latent for a long period, and then the patient is suddenly attacked with symptoms like those of meningitis or becomes rapidly comatose. If a person beyond middle life is the subject of heart disease, extensive arterial degeneration, or syphilis, and is at the same time suffering from the symptoms of a tumor situated at the base of the brain, an aneurism of one of the arteries of the brain may be suspected, and this suspicion will be confirmed provided a murmur is audible on auscultation of the skull. It is probable that aneurisms cause more pronounced symptoms of irritation than other growths, consisting of intense cephalalgia, paroxysms of severe and intractable trigeminal neuralgia, and attacks of mania and other grave psychical disorders. If a patient who has been suffering from the symptoms of a basal tumor die suddenly from an attack of ingravescent apoplexy, it may be conjectured that the tumor has been an aneurism. If a patient suffering from a tumor in the anterior fossa of the skull die suddenly after a copious hemorrhage from the nose, it may be assumed that an aneurism of the anterior cerebral artery has perforated the cribriform plate of the ethmoid bone. If pulsation and a murmur on auscultation be observed in the orbit immediately after an injury to the skull, it is probable that a communication has been established between the internal carotid and cavernous sinus. The localizing symptoms of intracranial aneurisms are the following: aneurisms of the anterior cerebral and anterior communicating arteries cause loss of smell and probably also unilateral blindness; aneurism of the internal carotid causes intractable facial neuralgia, paralysis of the motor nerves of the eyeball, blindness of one eye and temporal hemianopsia of the other. It may also cause complete facial paralysis, with distortion of the uvula and complete deafness on the side of the lesion. Aneurism of the posterior communicating artery causes paralysis of the third nerve with or without hemianopsia; aneurism of the posterior cerebral artery or of the superior cerebellar artery causes paralysis of

the third nerve of the same side and hemiplegia of the opposite side. Aneurism of the upper part of the basilar causes paralysis of the fifth and sixth nerves with bilateral weakness of the limbs, and aneurism of the lower part of the basilar or of the vertebral artery causes symptoms of bulbar paralysis with unilateral or bilateral weakness of the limbs. Occipital pain is also a symptom of aneurism of the basilar artery.

Tumors of the vertebral canal and spinal cord, and of the peripheral nerves, have already been sufficiently considered, and we shall now make a few general remarks on syphilis of the nervous system, a subject which we have purposely reserved to the last. There is scarcely any organic disease of the nervous system but may be simulated by syphilitic lesions.

Syphilis may cause periostitis, osteitis, exostoses, and caries of bones, which will first irritate and subsequently destroy neighboring nervous structures. When the vertebræ or bones of the skull are affected, the spinal or cerebral dura mater becomes thickened, and thus a chronic pachymeningitis is constituted which damages the roots of the nerves as they pass outwards to the intervertebral foramina, or along the base of the skull. Gummata may form in the dura mater, where they become encapsulated and injure the spinal cord or brain only by pressure; or in the subarachnoid space, and then the nerves, bloodvessels, and the nervous structures themselves are involved in the growth. Gummata may also grow in the sheaths of the peripheral nerves, and the cranial nerves are often implicated at their points of origin and before they have become covered by a prolongation of the dura mater. In other cases gummatous tissue forms a diffused infiltration in the substance of the nervous tissues, instead of forming a circumscribed growth. In many cases this infiltration undergoes a partial organization, and it then gives rise to a chronic sclerosis which may affect one of the physiological tracts of the spinal cord, or a localized portion of the brain, or it is disseminated in patches throughout the brain and spinal cord. When the syphilitic infiltration occurs in the membranes it renders the tissues, on being partially organized, dense, inelastic, and opaque, and causes adhesions between the dura and pia mater, and between the latter membrane and the brain. In such cases the neighboring nervous tissues are imperfectly nourished owing to the retraction of the lumen of the bloodvessels. The arteries of the body, as a whole, are liable to undergo chronic changes in syphilis, caused by an endarteritis, which may ultimately lead to the formation of aneurisms in various parts and occlusion of arteries by thrombosis, or even by embolism, or to rupture and hemorrhage. Syphilitic diseases of the bones and gummata may act like foreign bodies and set up a more or less acute inflammation of neighboring nerve structures, and thus extensive inflammatory affec-

tions which are not at all syphilitic in their own nature, may be originated by this poison, and when once a sclerosis of nervous tissue is set up by a syphilitic lesion, it is probable that the process will pursue a progressive course independently of the syphilitic poison. Syphilis appears also to favor the appearance of acute diseases of the nervous system without the presence of any lesion which is distinctively syphilitic. Acute ascending paralysis, for example, has been observed with preponderating frequency in syphilitic subjects, but an examination of the nervous tissues has not led to the discovery of any characteristic syphilitic lesion.

In the peripheral nerves syphilis gives rise occasionally to acute, more frequently to chronic neuritis, and to compression of both spinal and cranial nerves by the formation of gummata. In the spinal cord it tends to produce acute ascending spinal paralysis and probably acute spinal meningitis and acute myelitis, and it likewise causes, with variable frequency, chronic spinal meningitis, chronic myelitis, lateral spinal sclerosis, progressive locomotor ataxia, progressive muscular atrophy, labio-glosso-laryngeal paralysis, and ophthalmoplegia externa. In the brain it may be the cause of acute and chronic meningitis, hemorrhage, thrombosis, and even embolism of the arteries; thrombosis of one of the sinuses in cases of chronic pachymeningitis; a defined gummatous growth; and a chronic degeneration in which the symptoms are very similar to those of general paralysis of the insane, while it may also be the direct or indirect cause of functional cerebral lesions like epilepsy, hysteria, and chorea.

Inherited syphilis, besides giving rise to various local diseases in the peripheral nerves, spinal cord, and brain, or their membranes, is often the cause of imbecility in childhood, which progresses, if unchecked, until the mental faculties are destroyed. The disease appears to begin as a chronic meningitis of the convexity, and it leads to a progressive destruction of the cortex of the brain. When the change spreads over the motor area of the cortex it gives rise to a bilateral sclerosis of the pyramidal tracts, and corresponding to this change there is, during life, a spasmodic paralysis of the legs first, and ultimately of all the extremities. The growth of the brain may also be arrested by thickening of the cranial bones from syphilitic osteitis, or from narrowing of the arteries from syphilitic endarteritis. It asserted by Virchow that syphilis is sometimes a cause of congenital myelitis and encephalitis, and hydrocephalus appears to be frequently met with in the children of syphilitic parents.

Nervous affections belong, as a rule, to the later manifestations of syphilis, and make their appearance long after the more prominent phe-

nomena of constitutional syphilis have ceased to exist. Search must then be made for cicatrices on the genitals or on the groins; circular pigmented spots on the skin; depressed and irregular cicatrices over the forehead and front of the legs, with the integument adhering to the subjacent bones; radiated cicatrices on the mucous membranes, especially of the mouth; circular depressions on the arches of the palate or tonsils, which look as if a piece of tissue had been punched out; irregular protuberances on the surfaces of the bones; a moderate degree of hard swelling of the occipital, cervical, or cubital lymphatic glands; enlargement and knobby induration or atrophy of one testicle; cicatrix on the penis; and the existence of iritic adhesions. An inquiry into the history of a case may throw great light on its nature. If the patient be a man, it may be asked whether he has ever suffered from syphilitic infection. In the case of a married woman, valuable information may be obtained by ascertaining whether or not she has had miscarriages, if some of her children were stillborn or died soon after birth, or if those living manifest any of the characteristic symptoms of congenital syphilis. It is probable that nearly one-half of the cases of paralysis of the third, fourth, fifth, and sixth nerves, when one nerve only is affected, are of syphilitic origin, and curable by antisyphilitic treatment. The paralysis of the ocular nerves which occurs in the course of locomotor ataxia, is likewise, in the large majority of cases, an indirect result of syphilis. When a gummatous deposit takes place into the spinal pia mater, or into the substance of the spinal cord, the clinical features of the case often present more or less of the form of a hemiparaplegia, but the symptoms are never strictly unilateral, and there is an absence of the definite phenomena which are observed on a level with the upper limit of the lesion in other forms of spinal tumor. An outburst of cerebral syphilis is often preceded for two, three, or more weeks, by a deep-seated headache, with intense nocturnal exacerbations and sleeplessness. If a person after suffering from a headache of this kind is attacked with recurring unilateral convulsions, each of which is followed by a slight degree of paresis of the muscles affected by spasm, the case is one of local syphilitic cerebral meningitis of the convexity, either primary or secondary to disease in the bones, or a gummatous growth in or near the cortex. The presence of double optic neuritis in such a case points to a gumma rather than meningitis. An apoplectic attack occurring before forty-five years of age in a person who is free from cardiac or renal disease, points strongly to syphilis, thrombosis if the attack be slight, or hemorrhage from the bursting of an aneurism if it assume the form of ingravescent apoplexy.

The manner in which the symptoms are associated may help us in recognizing the presence of syphilis even in the absence of other evidence. Syphilitic lesions are, as Dr. Broadbent expresses it, often "multiple, seldom symmetrical." Peripheral paralysis of one of the ocular motor nerves of one side is probably never associated with a similar paralysis of the corresponding nerve of the opposite side, but is frequently accompanied by a unilateral epilepsy, or by a hemiplegia on one or other side, from syphilitic thrombosis. Syphilitic affections of the fifth nerve are also probably never bilateral, and if both of these nerves are paralyzed by a tumor at the base of the brain, the growth is likely to be cancer. The succession of the symptoms may also afford important aid in diagnosis. The lesions of syphilis being multiple, appear at different times and in widely different parts of the nervous system, and thus a group of symptoms, such as that caused by paralysis of a cranial nerve, is apt to be followed after a time by a convulsion from a deposit of gummatous tissue on the surface of the brain, or by hemiplegia from thrombosis of a vessel. In nervous affections supposed to result from inherited syphilis, it is important to observe the general conformation and physiognomy of the patient, and to examine the long bones for evidence of periostitis, and the fundus of the eye for *choroiditis disseminata*, besides making minute inquiries into the family history.

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