Diseases of the nervous system : for the general practitioner and student / by Alfred Gordon.

DISEASES
OF THE
NERVOUS SYSTEM

GORDON

DISEASES OF THE NERVOUS SYSTEM

FOR THE GENERAL PRACTITIONER AND STUDENT

BY

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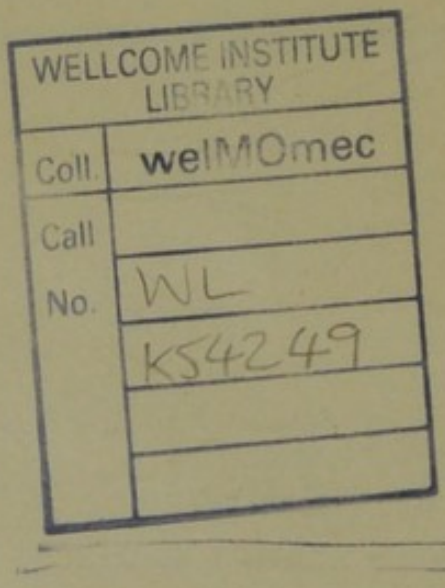

PREFACE TO THE SECOND EDITION

In presenting the second edition of this work I had again in view principally the general practitioner. The new facts which have been developed since the first edition I have endeavored to describe in a concise but at the same time complete manner. Each chapter almost without exception has been enlarged and among a number of additions the following important articles may be mentioned: (1) Fracture of the Skull; (2) Concussion of the Brain; (3) Lumbar Puncture; (4) Cerebro-spinal Fluid; (5) Wasserman Reaction; (6) Radiculitis; (7) Psychoanalysis.

Treatment has received special attention in accordance with the new data accumulated, such as administration of antimeningococcus serum, of salvarsan, surgical procedures, etc.

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PREFACE TO THE FIRST EDITION

In preparing this book for the medical public I had in view chiefly the general practitioner and the student. To both Neurology has always appeared to be a difficult and an insurmountable subject and many a student hesitates to take up its study in earnest. The fault lies partly in the text-books and treatises in which neurological subjects are discussed from the standpoint of the expert neurologist. In my daily association with students and general practitioners I have invariably heard this continuous complaint, viz., that they cannot get hold of a book on Neurology which could give them a **plain** and **practical** account of diseases of the nervous system. It is the want of such a work that I have endeavored to meet in the modest volume I am offering.

The modern physician is not satisfied with a mere enumeration of facts. In reading a description of any malady he wishes also to know the reason of the disturbed functions and the anatomical substratum of the morbid phenomena. Otherwise speaking he feels that he must know the relation of a certain manifestation to the normal and morbid physiology of an affected tissue or organ. The knowledge of **pathology** is therefore a *sine qua non* to every thinking man. This chapter must therefore precede any other in giving an account of a certain disease.

As I am aiming almost exclusively to present Nervous Diseases from a practical standpoint, I naturally avoided too technical and debatable points of pathology, but on the contrary endeavored to present the most essential changes necessary for a thorough understanding of various clinical manifestations.

In discussing the *symptomatology* I point out, whenever it is possible, the direct relation between certain phenomena and the pathological changes so as to give the reader an intelligent idea of the morbid symptoms.

Each form of functional or organic nervous disease is also discussed from the standpoint of **differential diagnosis.** All possible affections which may simulate a given disease have been taken up *seriatim* and differences emphasized.

The **course** of the diseases, their mode of **termination,** their **prognosis** and the **etiology** have been given full consideration. In describing the latter, the most well known and well established factors have been pointed out first. The reader may be surprised to find Etiology placed in some chapters before Symptomatology and in others immediately before Treat-

ment. This was arranged according to the importance Etiology plays in certain diseases or according to the amount of knowledge we possess of the causative factors in various diseases.

Considerable space has been devoted to **Treatment.** Only the most useful and the best known devices, appliances, operations and drugs are described. Medications that are uncertain as to their therapeutic value are omitted or else only mentioned.

True to my original aim I have endeavored to present to the reader only the most essential points, whether it was in Pathology, Symptomatology, Pathogenesis, Etiology or Treatment. The latest views, ideas and thoughts have been presented as far as it was possible. Intentionally I avoided details on disputable questions and omitted them altogether whenever I could without sacrificing the clearness of the subject.

A Chapter on the **Method for examination** of patients precedes the description of diseases of then ervous system. In it are indicated what phenomena are considered normal or abnormal. I have described here the motor, sensory and trophic phenomena, also the reflexes, the state of sphincters and electrical contractility of muscles or nerves.

Finally a chapter on the Normal Anatomy of Brain and Cord, also Malformations of the Nervous System, has been added. Detailed descriptions have been omitted whenever it was possible and instead clear illustrations are given.

On the whole I feel that I am presenting a practical book to the average physician, but if also the neurologist, the teacher, the advanced student may find in it some ready references which they may peruse in their scientific studies, my labors will be more than compensated.

A word of thanks is due to the publishers. They have facilitated my task by allowing a large number of illustrations, without which no modern scientific work can be satisfactory.

ALFRED GORDON.

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DISEASES OF THE NERVOUS SYSTEM

CHAPTER I

ANATOMY AND PHYSIOLOGY OF THE CENTRAL NERVOUS SYSTEM

SPINAL CORD

THE cord covered by three membranes is placed in the vertebral canal and extends from the upper border of the atlas down to the upper border of the second lumbar vertebra. It occupies therefore only two-thirds of the vertebral canal, viz. its cervical and thoracic portions. It is approximately a cylindrical body presenting two enlargements and a conical termination. Its length is about 45 cm. (18 inches) in the male and 41 cm. (16 inches) in the female.

The cord is continuous above with the medulla and below it forms a thread-like termination (**filum terminale**) which extends to the coccyx to which it is attached (Fig. 1).

The spinal cord is divided into the following segments: **cervical, thoracic, lumbar** and **sacral** or Conus Medullaris. The Cervical and Lumbar segments are the thickest parts of the cord, viz. the enlargements mentioned above.

The segments correspond to the following vertebræ. The **Cervical enlargement,** which supplies nerves to the upper extremities also gives origin to the phrenic nerve, extends from the third cervical to the second thoracic vertebra and has its maximum of development at the level of the sixth cervical vertebra. The portion of the cord above the enlargement corresponds to the first two cervical vertebræ. The **Thoracic segment** extends from the second to the ninth thoracic vertebra. The **Lumbar enlargement,** which supplies nerves to the lower extremities, commences at the level of the ninth thoracic and terminates at the lower border of the first lumbar vertebra. Its maximum corresponds to the twelfth thoracic vertebra.

The **Conus Medullaris** or sacral segment is the very small conical portion extending from the first to the second lumbar vertebra.

Exterior of the Cord.—The cord is divided into two halves by an

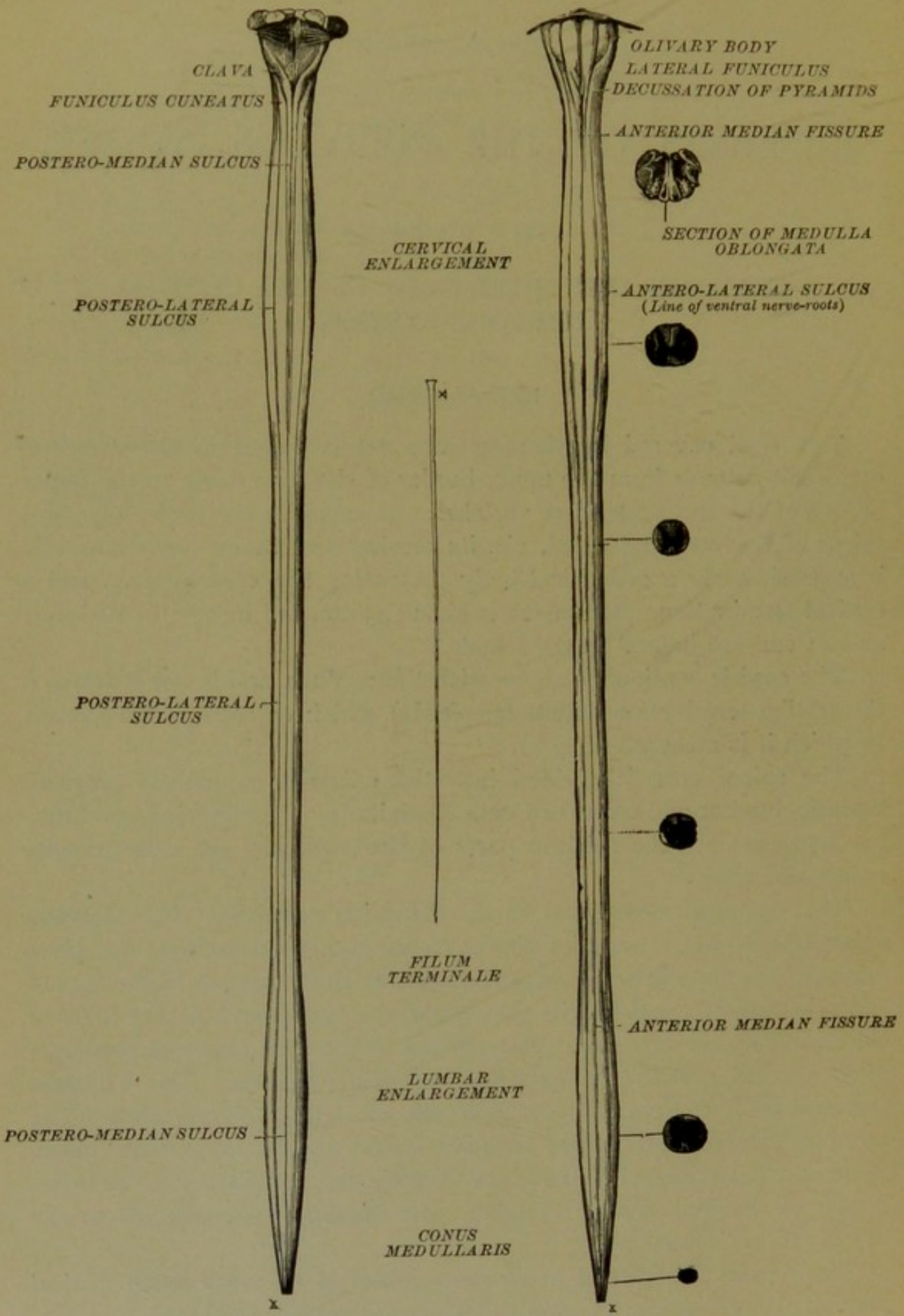


FIG. 1.—POSTERIOR AND ANTERIOR VIEWS OF THE SPINAL CORD. (*Morris, modified from Quain.*)

anterior and **posterior** median fissure. The anterior is a deep and broad fissure and contains a duplication of the pia-mater with its important blood vessels. The posterior median fissure is simply a sulcus, a **septum.** Each half of the cord is divided by two sulci into three portions. They are: the **postero-lateral** sulcus, which receives the posterior sensory roots, and the **antero-lateral** sulcus, which is the place of exit of the anterior roots.

The portions of the cord between the sulci present in each half longitudinal columns, viz. **posterior, lateral** and **anterior.**

Interior of the Cord.—A transverse section shows that the spinal

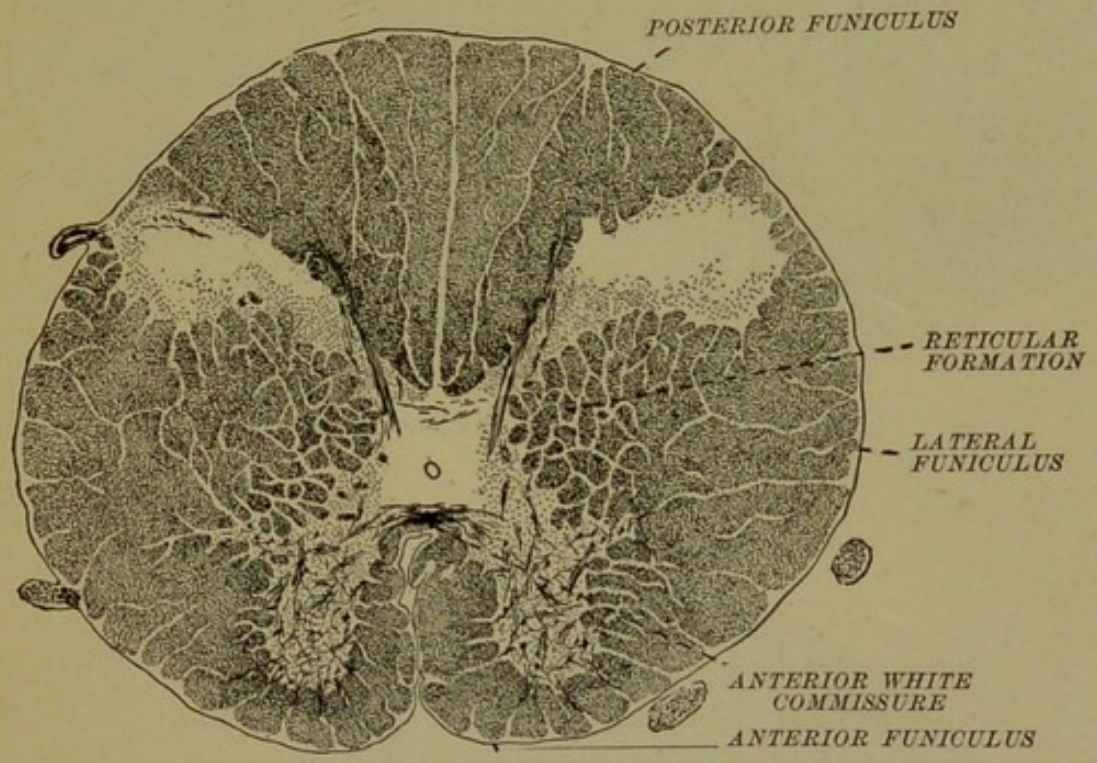


FIG. 2.—CERVICAL II. (*After Morris' Anatomy.*)

cord consists of a central **gray** and a peripheral **white** substance (Figs. 2, 3, 4, 5, 6 and 7).

Gray Substance.—It presents two symmetrical halves united in the middle line by a gray commissure in the center of which is the **central canal.** The two halves with the intermediate commissure give the impression of the letter H. The central canal extends through the entire length of the cord.

Each half of H-shaped gray mass has an anterior and posterior portion, called "cornua." The **anterior cornu** is distinguished by its larger, wider and thicker appearance than the posterior. Besides, it is separated from the periphery of the cord by the white substance. Between the anterior portions of the two anterior cornua lies a bundle of fibers crossing the middle line, which constitutes the **anterior white commissure.**

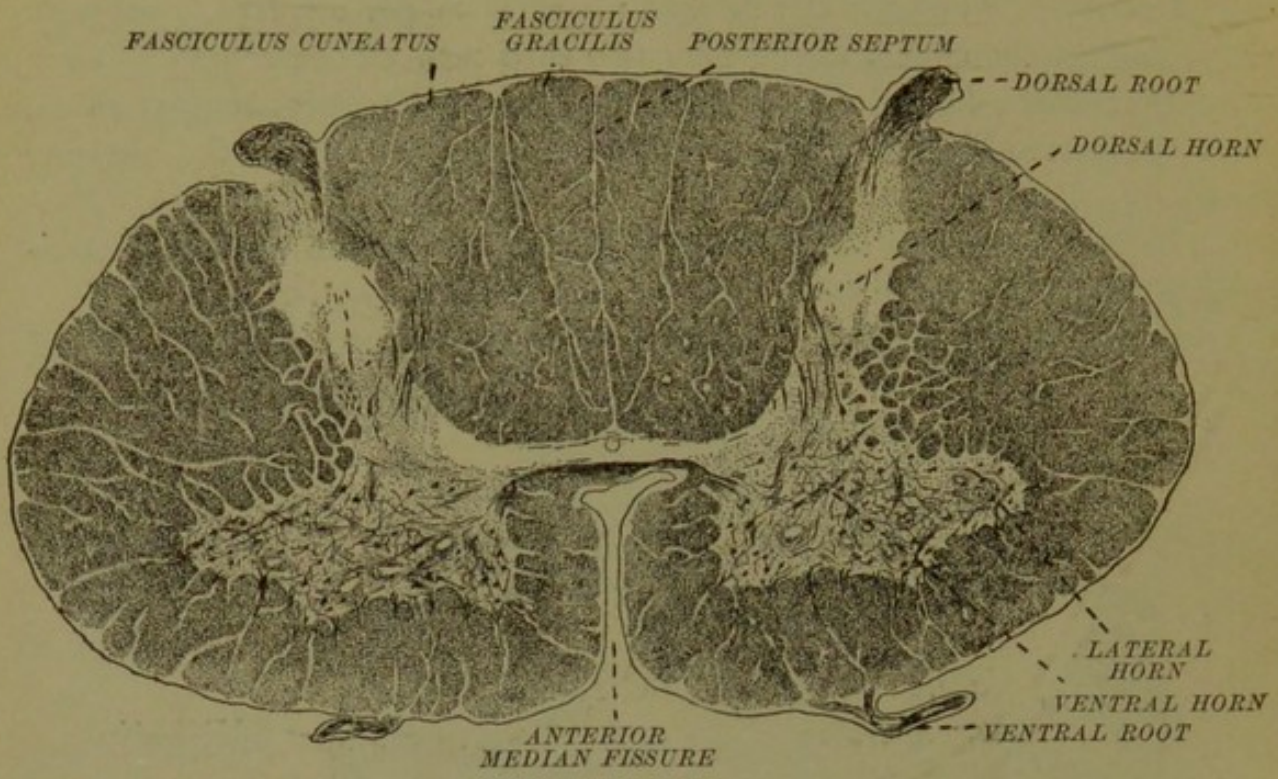


FIG. 3.—CERVICAL VI. (*After Morris' Anatomy.*)

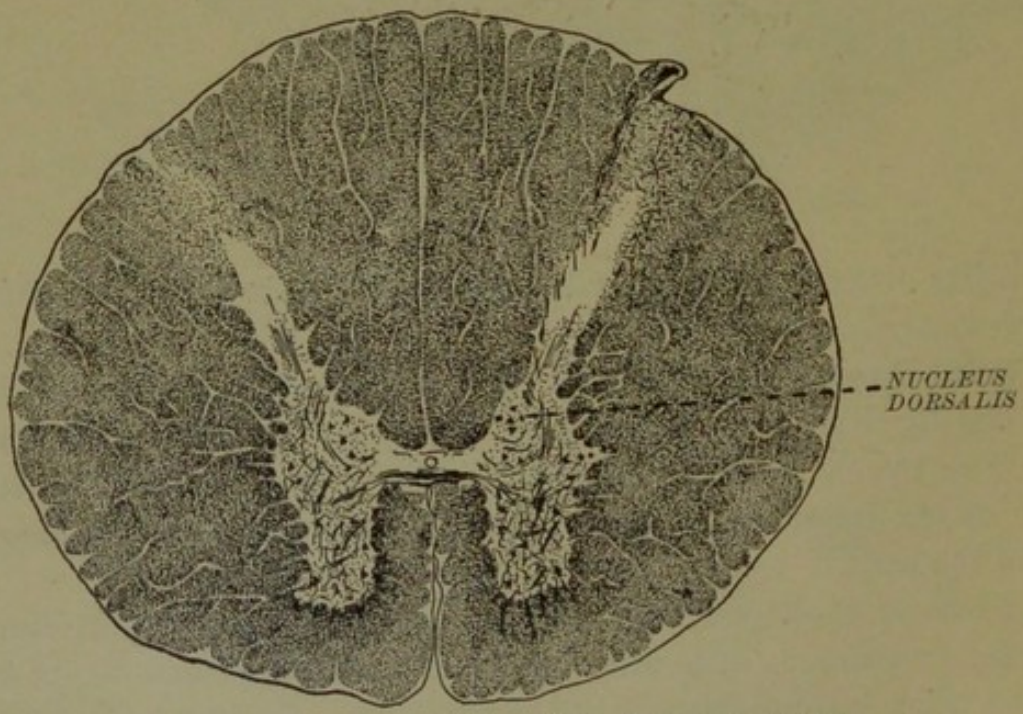


FIG. 4.—THORACIC VIII. (*After Morris' Anatomy.*)

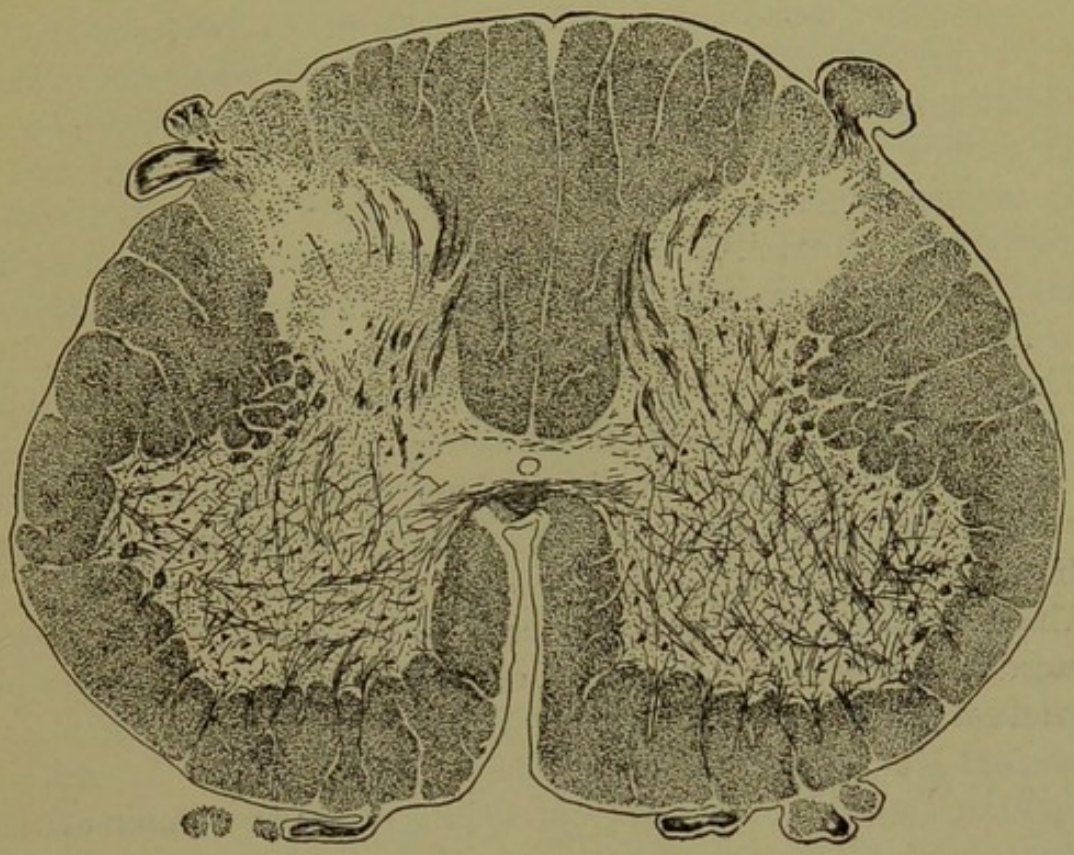


FIG. 5.—LUMBAR III. (*After Morris' Anatomy.*)

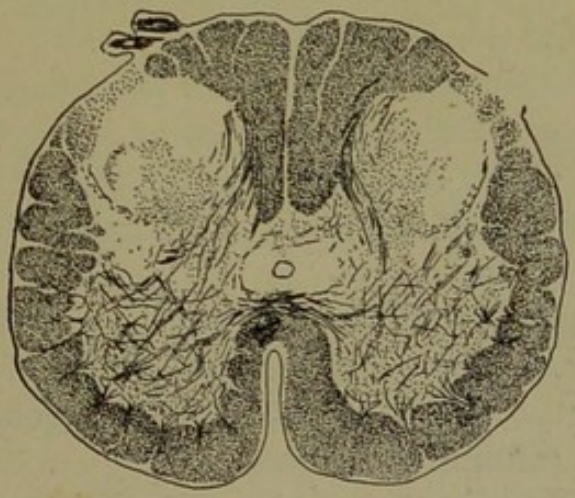


FIG. 6.—SACRAL IV. (*After Morris' Anatomy.*)

FIG. 7.—COCCYGEAL. (*After Morris' Anatomy.*)

FIGS. 2 TO 7.—TRANSVERSE SECTIONS FROM DIFFERENT SEGMENTS OF THE SPINAL CORD, SHOWING SHAPE AND RELATIVE PROPORTIONS OF GRAY AND WHITE SUBSTANCE IN THE DIFFERENT SEGMENTS REPRESENTED. (*Morris' Anatomy.*)

The **posterior cornu** is thin, long and extends to the periphery of the cord. It presents a head, neck and base. The head is surrounded by the **Gelatinous substance of Rolando.** The latter is particularly marked in the cervical region.

In the **upper thoracic** segment of the cord there is a lateral prominence of gray matter situated between the bases of the anterior and posterior cornua. This is the so-called **lateral cornu.** It is not found in the cervical or lumbar region.

The anterior and posterior cornua retain their relative size through the entire cord, but the amount of gray matter in them varies according to the level of the cord. Thus in the lumbar and cervical enlargements they reach the maximum. In the midthoracic region they are at the minimum.

The gray matter is composed essentially of **cells.** The latter are arranged in **groups** in each cornu. In the anterior cornu there are mainly: an **external** group which is the principal origin of the anterior roots and an **internal** group which sends out fibers for formation of the white commissure (see above). A special group of cells (**vesicular column of Clarke**) is situated in the internal portion of the base of the posterior cornu. It extends from the eighth cervical to the third lumbar segment of the cord. The majority of these cells are the origin of the direct cerebellar tract of the same side (see below).

The gelatinous substance of Rolando is rich in cells.

White Substance.—The fibers composing it originate from the cells of the gray matter of the cord and from the cells of the brain and cerebellum. They form systems of fibers or **columns** with different physiological functions (FIG. 8).

I. **Posterior Columns.**—Uniform in the lower half of the cord, they are divided into two distinct columns at the level of the upper thoracic and cervical segments. The median bundle is the **column of Goll** and the outer bundle is the **column of Burdach.** They are composed essentially of fibers of ramification of the posterior roots.

The fibers of the posterior roots emanate mostly from the cells of the spinal ganglia. The axone leaving its cell undergoes a division in the shape of letter T. One branch goes to the periphery and the other enters the posterior root. The fibers of the latter ascend and subdivide in **ascending** and **descending** branches. The descending ones are mostly short fibers, give off collaterals and enter the cells of the gray matter. The ascending branches are long and short. The first extend to the medulla where they terminate in the nuclei of Goll and Burdach. The latter end in the cells of the gray matter.

The root fibers, upon their entrance into the cord, are divided into two groups: an external, which constitutes **Lissauer's tract** or **marginal zone of Lissauer** (in formation of which participate also axones from the posterior horns) and an internal which constitutes the posterior columns properly.

Both branches of each root-fiber give off **collaterals.** They are **short** and **long.** The **short** ones end around the cells of the posterior cornua

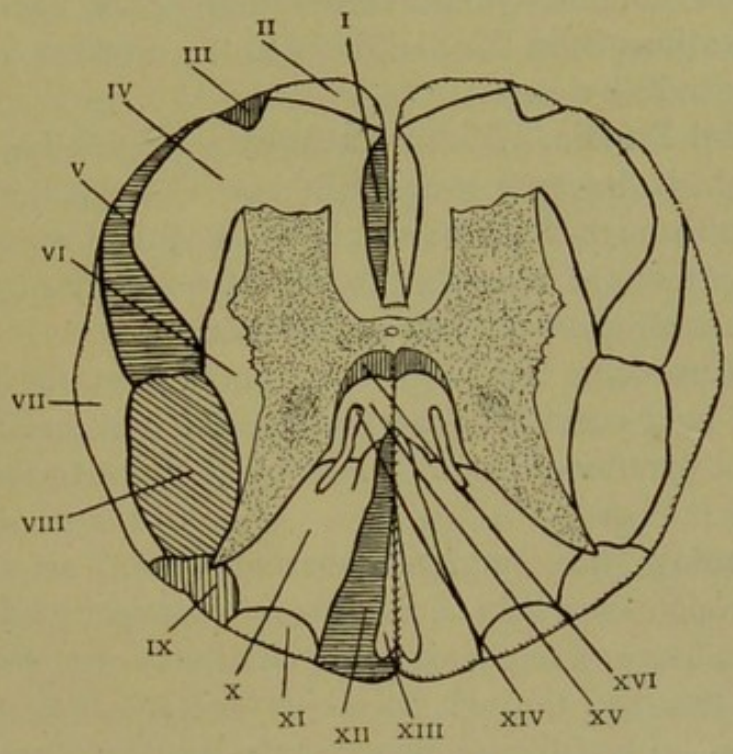


FIG. 8.—SCHEMATIC REPRESENTATION OF THE SITUATION OF THE VARIOUS TRACTS OF FIBERS IN THE SPINAL CORD. (*Gordinier.*)

I. Direct pyramidal tract. *II.* Descending tract of Marchi and Lowenthal. *III.* Olivary or triangular tract. *IV.* Antero-lateral ground bundles of fibers. *V.* Antero-lateral ascending tract of Gowers. *VI.* Lateral limiting layer. *VII.* Direct cerebellar tract. *VIII.* Crossed pyramidal tract. *IX.* Lissauer's tract. *X.* Middle root zone. *XI.* Posterior root zone. *XII.* Postero-internal or column of Goll. *XIII.* Septo-marginal tract. *XIV.* Comma tract of Schultze. *XV.* Anterior root zone. *XVI.* Cornu-commissural tract.

and of the cells of Clarke; they also cross the gray commissure and end in the cells of the posterior cornua of the opposite side.

The **long** collaterals end around the cells of the anterior cornua. They therefore carry to the motor cells of the anterior cornua peripheral sensory impulses, received from the periphery through the spinal ganglia. They therefore form a part of the **arc reflex.**

In the posterior columns there are also fibers originating in the cord itself (**endogenous**). In the very anterior portion behind the gray commissure there is the **Cornu-commissural tract.** Its fibers originate in the cells of the posterior cornu. They have an ascending direction.

In the **cervical region** there is a small bundle situated in the middle of the posterior columns and called **comma of Shultze.** A small bundle situated in the thoracic region at the periphery (**Hoche's bundle**), in the lumbar region on each side of the median septum and in the middle (Flechsig's oval bundle) and in the sacral region on the posterior and internal portion (Gombault's and Philippe's triangle) are probably the same system of fibers. All these separate fasciculi have a descending course.

The posterior columns proper are composed of **sensory neurones** conducting sensations from the periphery to the cerebral cortex and have therefore an ascending course.

II. **Pyramidal Bundle. Motor Pathway.**—Originating in the motor area of the brain it descends toward the base through the internal capsule lower down through the pons and in the medulla forms the **pyramids.** In the lower part of the medulla the largest majority of the pyramidal fibers decussate with those of the opposite side.

The **non-decussating** fibers (Türck's bundle) descend in the anterior columns of the cord occupying the portion near the median fissure. It is called **"direct pyramidal tract."** It extends down to the middle thoracic segment of the cord.

The **decussating** fibers (**crossed pyramidal tract**) after passing from the medulla through the anterior cornua on the opposite side of the cervical cord (first and second segments) are placed very posteriorly in the lateral portion of the cord through its entire length. It is separated from the periphery of the cord by the direct cerebellar tract, except in the lumbar region where the latter does not exist.

The pyramidal fibers with their collaterals terminate around the cells of the anterior cornua.

They are motor and centrifugal (descending); they transmit to the motor cells of the anterior cornua voluntary impulses from the cortical motor centers.

III. **Direct Cerebellar Tract** (Flechsig).—The fibers originate in the cells of Clarke's columns and commence at the level of the first lumbar segment. They occupy the periphery of the postero-lateral portion of the cord.

They are **sensory** and **centripetal,** have an ascending course and at the level of the medulla enter the restiform bodies (inferior cerebellar peduncle) to terminate in the cerebellum. They carry to the cortical cells of the latter impressions received by Clarke's cells from the posterior roots.

IV. **Gowers' Tract** (Antero-lateral fasciculus).—The majority of its fibers originate from cells of the anterior cornua of the opposite side, although the exact source is not known. The bundle is situated antero-

laterally, in front of the crossed pyramidal and in front of and internally to the direct cerebellar tract. It commences at the level of the dorso-lumbar region.

Its fibers are **sensory** and have an **ascending course.** Some of its fibers go to the cerebellum through the superior cerebellar peduncle and terminate in the cortex of the superior vermis. Others enter the restiform body. A few reach the anterior corpora quadrigemina.

V. **Antero-lateral Ground Bundle** surrounds immediately the antero-lateral portion of the gray matter. It reaches the periphery only in front of the anterior cornu. Its fibers originate partly in the cells of the gray matter and serve for associating various levels of the cord. It contains also fibers descending from the cerebellum, red nucleus, Deiter's nucleus, corpora quadrigemina and optic thalamus. **Loewenthal's bundle** or **anterior marginal fasciculus** is a narrow band occupying the border of the cord between the anterior end of Gower's tract and the anterior median fissure. It originates in the roof nucleus of the cerebellum (nucleus fastigii) and terminates about the cells of the anterior horns. Its fibers are therefore **descending.**

Roots of the Spinal Nerves.—The axones emanating from the cells of the anterior cornua, reach the periphery of the cord and form the **anterior roots.** After piercing the membranes of the cord they advance to the intervertebral foramina and beyond the spinal ganglia situated in those foramina they join the posterior roots to form a spinal nerve.

The function of the anterior roots is **motor** (and trophic).

The **posterior** roots are formed of **sensory** fasciculi coming from the periphery, penetrate the intervertebral foramina where they meet the spinal ganglia. From there, covered by the three membranes of the cord, they reach the postero-lateral sulcus and enter the cord.

The spinal nerves formed of the junction of the anterior and posterior roots are 31 pairs in number.

The roots of the first cervical nerve are horizontal. Beginning with the thoracic nerve the direction of the roots is very oblique, so that the last roots extend through a distance of several vertebræ.

MENINGES OF THE SPINAL CORD

The cord is surrounded by three membranes: **dura-mater, arachnoid** and **pia-mater.** (FIG. 9.)

Dura consists of one layer. It is a resistant membrane and contrary to the dura of the brain, does not constitute the internal periosteum of the spinal canal. It is separated from the bony walls of the spinal canal by a loose areolar tissue with a plexus of veins.

It commences at the foramen magnum and terminates at the level of the third piece of the sacrum, while the spinal cord ends only at the level of the second lumbar vertebra. Below the cord the cavity of the dural sac is occupied by a bundle of nerves—cauda equina—in the midst of which is seen the filum terminale. Below the third sacral vertebra the dura becomes only a filament extending to the coccyx (coccygeal ligament). It is attached to the canal by ligaments and prolongations which together with the pia and arachnoid accompany and surround the spinal roots on their way to the intervertebral foramina.

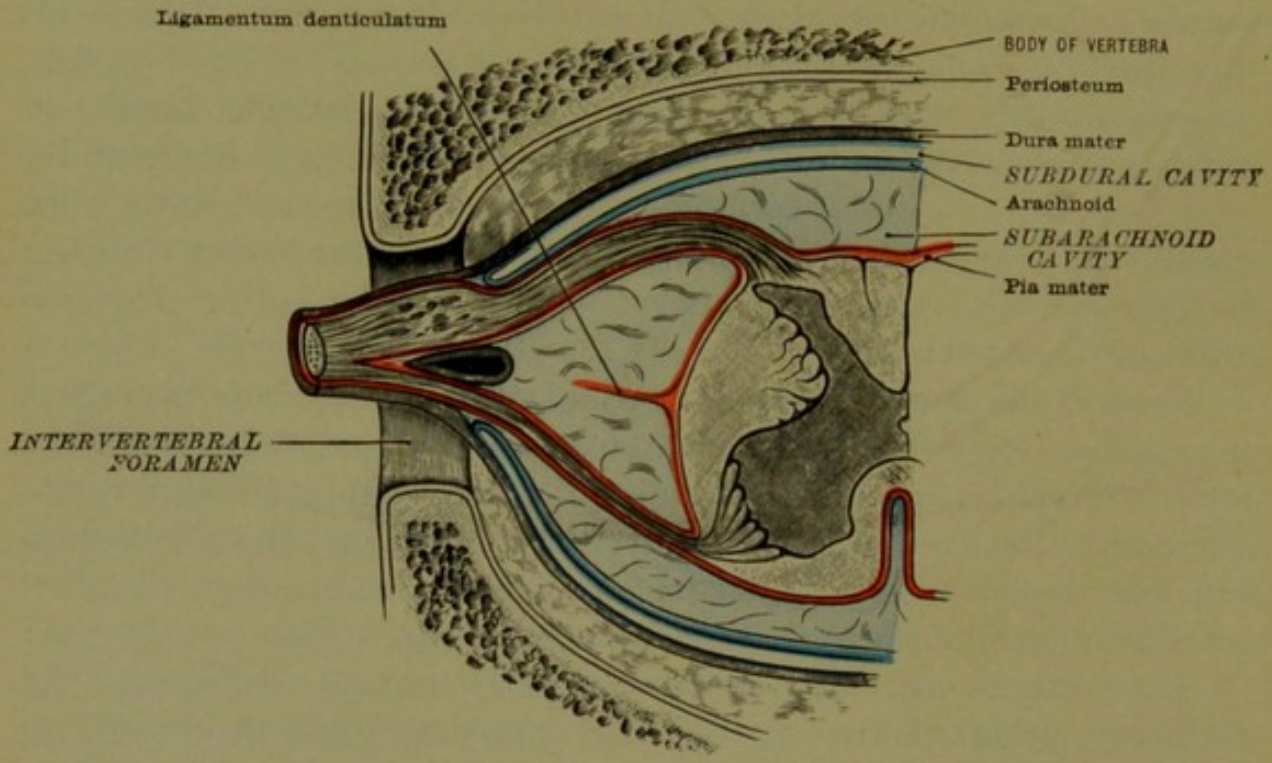


FIG. 9.—DIAGRAM SHOWING RELATIONS OF MENINGES TO SPINAL NERVE-ROOTS. (*Morris' Anatomy.*)

The inner surface of the dura is attached to the pia-mater by **dentate ligaments** through the arachnoid. Between the dura and the arachnoid there is the so-called **subdural cavity** which contains cerebro-spinal fluid.

Arachnoid.—It covers the cord and the cauda equina and is continuous with the arachnoid of the brain. Between it and the pia there is the so-called sub-arachnoid space containing a reticulum the meshes of which are occupied by the cerebro-spinal fluid. The above mentioned **dentate ligaments** and the **septum posticum** keep it attached to the pia and dura. It gives off prolongations to the spinal roots.

Pia.—It is the most internal of the three meninges. It is an extremely vascular membrane closely adherent to the cord. It is thicker in the spinal cord than in the brain. It is composed of two layers. It forms a

fold in the anterior fissure of the cord. It sends off prolongations to the spinal roots. At the level of the filum terminale it behaves like the dura (see above). **Ligamentum dentate** mentioned above is a fold of the pia and presents processes which are attached on the inner surface of the dura between the roots of the spinal nerves.

BLOOD SUPPLY OF THE SPINAL CORD

The main **arteries** of the spinal cord are three in number, viz. one **anterior spinal artery** along the anterior spinal fissure, two **posterior spinal arteries,** one on each side behind the line of entrance of the posterior nerve roots. These arteries give off branches which enter the cord to be distributed more in the gray than in the white matter.

The spinal arteries originate from the vertebral arteries which pass through the inter-vertebral foramina together with the spinal roots.

The **veins** are situated along the anterior and posterior fissures of the cord and laterally one on each side along the roots. They then pass through the foramina and open into the vertebral veins.

Lymph spaces are found around the nerve cells and blood vessels.

MEDULLA OBLONGATA

Pons and Fourth Ventricle

(Rhombencephalon)

The medulla or bulb is the upward continuation of the spinal cord. It occupies the basilar groove of the occipital bone with its lower extremity in the foramen magnum. Its upper extremity marks the beginning of the pons.

Its length is 25 mm. (one inch), thickness 14 mm. (one-half inch) and width at the lower end one-half inch, at the upper end three-fourths of an inch. Its direction is almost vertical and only slightly inclined forward.

Exterior of the Medulla.—It has an anterior, a posterior and two lateral surfaces, also an upper and lower extremities. The anterior median fissure of the spinal cord is continued on the anterior surface of the medulla, but obliterated in its lower portion on account of **decussation of the pyramids.** In the upper portion of the **anterior surface** are seen the **Pyramids.** They are two large bodies thicker at the upper ends than at the lower, bounded laterally by a sulcus from which emerge the twelfth nerves (hypoglossi). The sulcus corresponds to a similar antero-lateral sulcus

of the spinal cord from which emerge the anterior roots. The sulcus separates the pyramids from oval bodies called **Olives.** Laterally to each olive lies the Restiform body and in the sulcus between them emerge the roots of the ninth (glosso-pharyngeal), tenth (vagus) and eleventh (spinal accessory) nerves. At the upper border of the pyramids

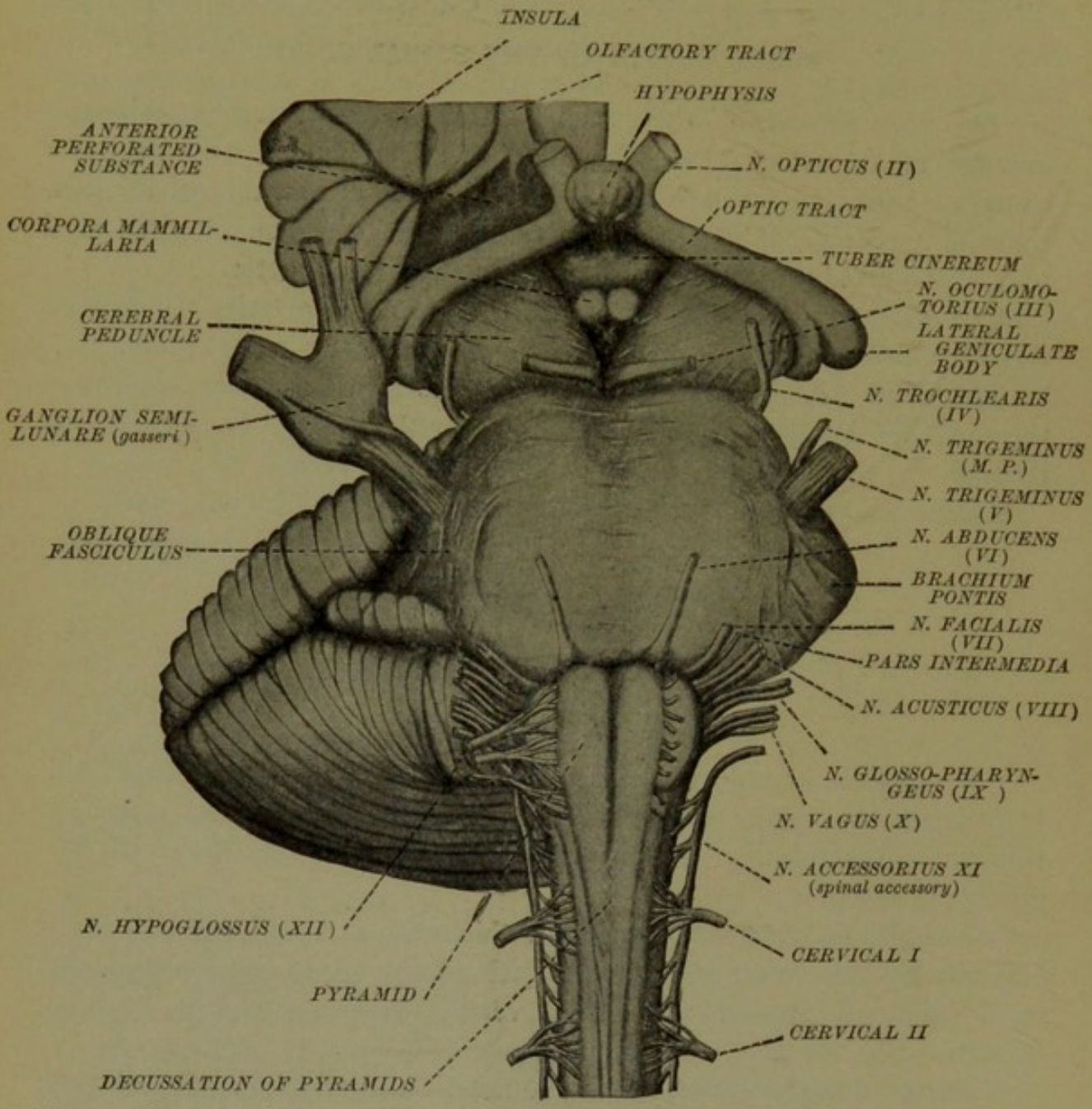


FIG. 10.—VENTRAL ASPECT OF BRAIN-STEM INCLUDING MAMMILLARY AND OPTIC PORTIONS OF THE HYPOTHALAMUS. (*Morris' Anatomy.*)

and olives, viz. at the lower border of the pons emerge the roots of sixth (abducens) and seventh (facial) nerves. On the **Posterior surface** of the medulla are seen the following elements. The lower portion is the closed part of the medulla, the upper portion, in which the two halves become separated, forms an open triangle as a part of the fourth ventricle.

There is a **median sulcus,** continuation of the posterior sulcus of the cord. The columns of Goll end in an elevation (**clava**) which contains a nucleus,

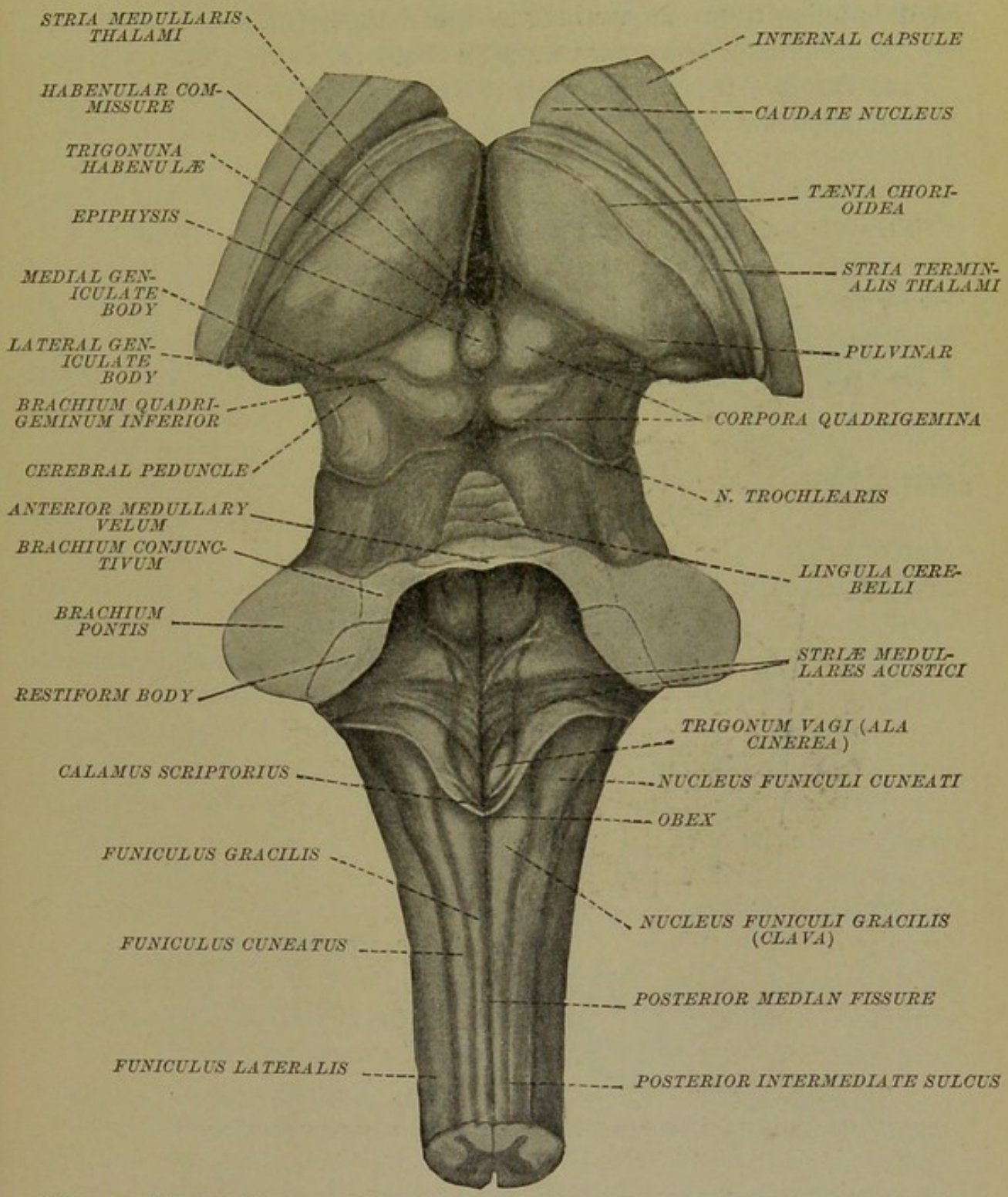


FIG. 11.—DORSAL ASPECT OF MEDULLA OBLONGATA AND MESENCEPHALON, SHOWING THE FLOOR OF THE FOURTH VENTRICLE (RHOMBOID FOSSA). (*Morris, Modified from Spalteholz.*)

called nucleus of Goll (**nucleus gracilis**). Laterally and anteriorly there is another elevation containing the nucleus in which the fibers of Bur-

dach's columns terminate; it is called nucleus of Burdach (**nucleus cuneatus**).

Laterally are located the **Restiform bodies,** which contain ascending and descending fibers connecting the spinal cord with the cerebellum; they are the inferior cerebellar peduncles (Figs. 10, 11).

Pons Varolii.—It is a large mass of white substance situated between the medulla below and cerebral peduncles (crura) above.

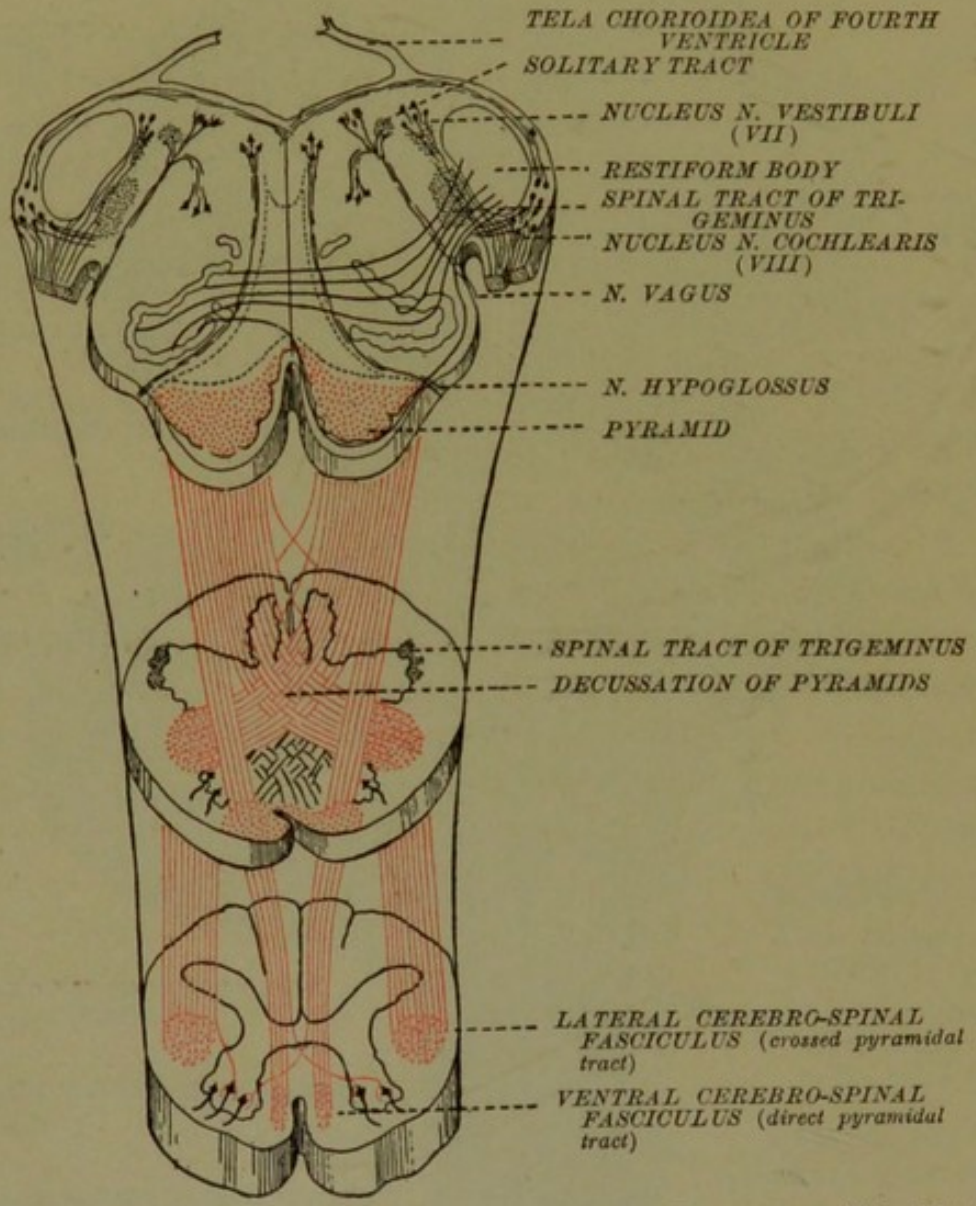


FIG. 12.—DIAGRAM SHOWING THE DECUSSATION OF THE PYRAMIDS. (*Morris' Anatomy.*) The uppermost level represented is near the inferior border of the pons.

Its **anterior surface,** containing the basilar artery, is situated on the basilar process of the occipital bone. It is continuous laterally with the middle cerebellar peduncles. The roots of the fifth nerve (trigominus) emerge on both sides of this surface. The **posterior surface** is continuous with the posterior surface of the medulla and forms with the latter the

floor of the fourth ventricle, covered by the cerebellum. The **upper border** separates the pons from the cerebral peduncles. It corresponds to sella turcica of the sphenoid bone. From it emerge the third (oculomotor) nerve and the fourth (pathetic) nerve. The pons consist of superficial and deep transverse fibers between which pass the fibers of the cerebral peduncles from above to constitute the pyramids below.

Fourth Ventricle.—It is a rhomboidal cavity situated between the medulla, pons and cerebellum. It is continuous below with the central canal of the spinal cord and above with the aqueduct of Sylvius.

The Floor.—It is lined with the epithelium which is continuous with the ependyma of the central canal. Its superior half belongs to the pons, the inferior half to the medulla. It contains eminences and depressions. On each side of the median sulcus lies a longitudinal band (**funiculus teres**) which commences at the lower end (**calamus scriptorius**) as a grayish mass (**ala cinerea**).

Striæ Acusticæ, bundles of fibers arising in the nuclei of termination of the cochlear division of the eighth nerve, cross the floor of the fourth ventricle and divide it into two halves: **upper** and **lower.**

Upper Half.—It contains the upper portion of the funiculus teres (see above), on each side of which lies the **acustic tubercle** (one of the origins of the eighth nerve). Between them there is a depression (**fovea anterior,** or **fovea trigemini**) which overlies the larger portion of the nucleus of the fifth nerve. Above this fovea lies a grayish mass (locus cœruleus), which is also a portion of the nucleus of the fifth nerve.

Lower Half.—It contains below at the origin of the funiculus teres **ala cinerea** (see above), which corresponds to the nuclei of the ninth and tenth nerves. Mesial and above the ala cinerea is an eminence (**trigonum hypoglossi**) which corresponds to the origin of the twelfth nerve. Externally to the latter there is another eminence (**trigonum acusticum**) corresponding to the eighth nerve.

Roof of the Fourth Ventricle.—The anterior portion is formed by the cerebellum and superior cerebellar peduncles. It is covered by a lamina of white matter, the **anterior medullary velum,** the inferior portion of which is continuous with the white substance of the cerebellum.

The middle portion is covered by the **posterior medullary velum,** which is also continuous with the white substance of the cerebellum.

The inferior portion is covered by an epithelial membrane (**tela chorioidea**), a double fold of the pia containing vascular processes, viz. **choroid plexuses.** The thickened lateral portions of the tela are called **ligulæ** and the thickened portion of it at the calamus is called **obex.**

The **Margins** of the fourth ventricle.

The **superior** borders are formed by the superior and middle cerebellar peduncles, the **inferior** by the restiform bodies and the terminations of the posterior columns of the cord.

Interior of the Medulla and Pons.—Sections beginning from the lower level of the medulla upward show the gradual formation of the

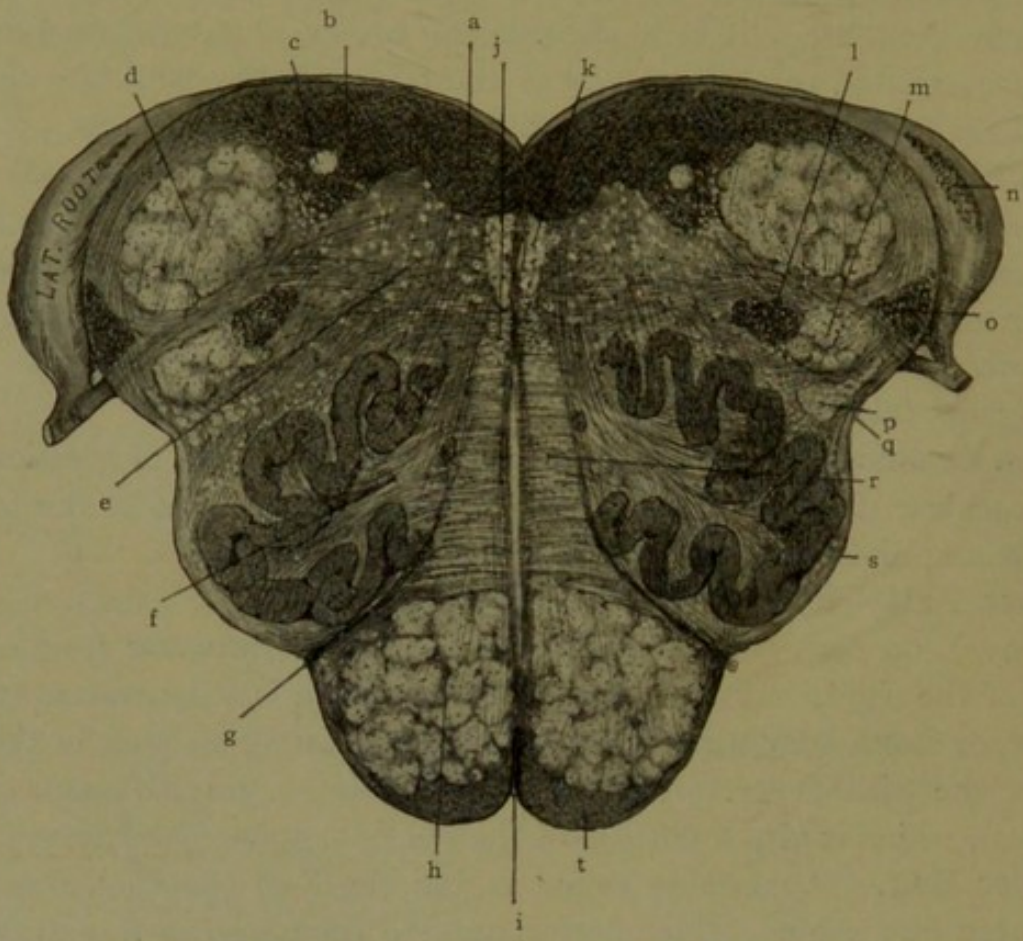


FIG. 13.—SECTION OF MEDULLA OBLONGATA NEAR THE PONS. (*Santee.*)

a. Hypoglossal nucleus. *b*. Vestibular nucleus. *c*. Tractus solitarius. *d*. Restiform body. *e*. Substantia reticularis. *f*. Hilus of olivary nucleus containing cerebello-olivary fibers. *g*. Anterior lateral sulcus. *h*. Pyramid. *i*. Anterior median fissure. *j*. Anterior longitudinal bundle. *k*. Medial longitudinal bundle *l*. Nuc. tractus spinalis n. trigemini. *m*. Tractus spinalis n. trigemini. *n*. Lateral cochlear nucleus. *o*. Ventral cochlear nucleus. *p*. Ascending anterior cerebello-spinal, spino-thalamic and rubro-spinal tracts. *q*. Posterior lateral sulcus. *r*. Medial fillet, interolivary stratum. *s*. Anterior external arcuate fibers. *t*. Arcuate nucleus.

Pyramids. They are formed, properly speaking, of the pyramidal fibers descending from the cerebral peduncles through the deep and superficial fibers of the pons. At the lower level of the medulla they begin to decussate to go down in the cord (see Spinal Cord, Fig. 12). Immediately behind the pyramids lies the median **fillet** or **lemniscus.** This bundle of fibers originates in the nuclei of Goll and Burdach (see above). As we have seen above, the sensory neurones of Goll's and Burdach's

columns of the spinal cord terminate in the lowest parts of the medulla in two nuclei (gracilis or Goll and cuneatus or Burdach). The function, however, of these neurones is continued: new fibers originate in the cells of these nuclei and ascend. They begin to decussate in the median line (raphe) with their fellows of the opposite side, immediately above the decussation of the pyramids and form a large sensory bundle, called **median fillet** (**lemniscus**). At the level of the pons the median fillet spreads laterally on both sides; the lateral portions are called **lateral**

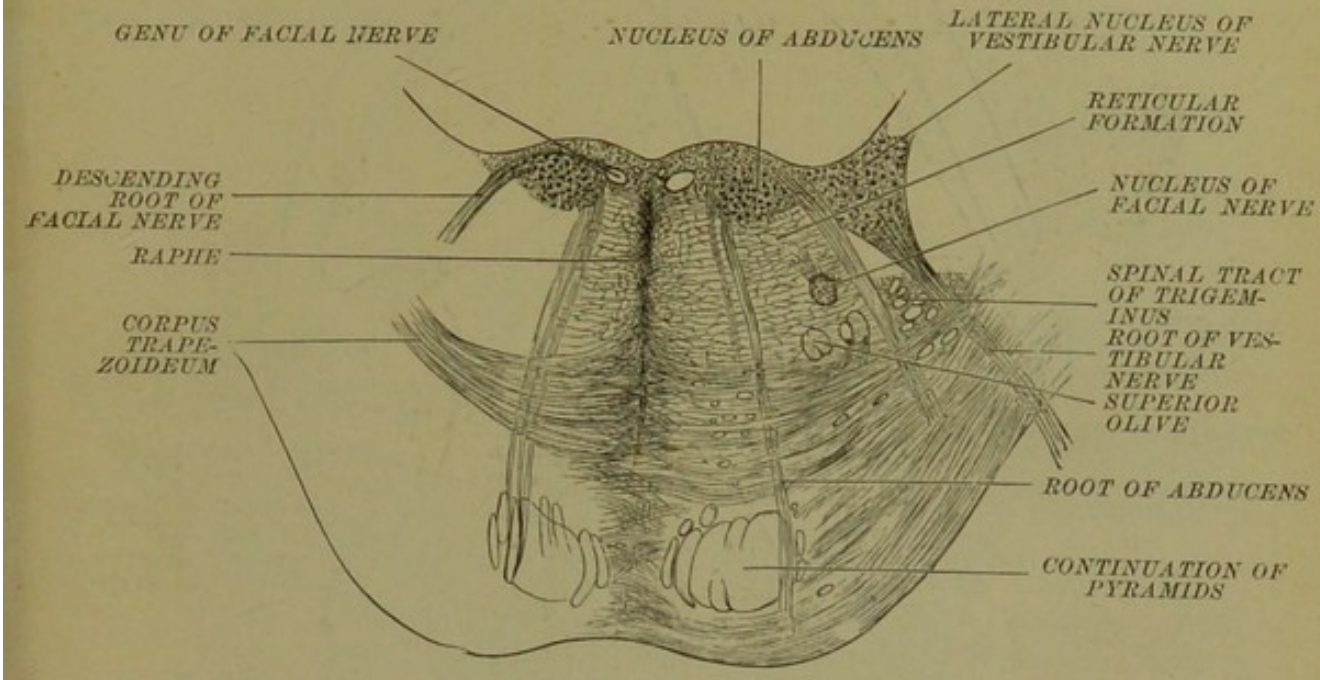


FIG. 14.—DIAGRAM OF TRANSVERSE SECTION OF INFERIOR PART OF PONS. (*Morris, after Schwalbe.*)

The restiform body, not included, occupies the curved space lateral to the nucleus of vestibular nerve.

lemnisci. Posteriorly lies a third bundle, called **"posterior longitudinal fasciculus,"** the function of which is to associate the nuclei of the cranial nerves (Figs. 13, 14).

In addition to these three fasciculi there are a number of fibers running various courses. Such are the **internal arcuate** fibers, some of which connect one restiform body (inferior cerebellar peduncle) with the olivary body and the restiform body of the opposite side; others are destined for both cerebellum and cerebrum.

The **Olives** are isolated masses of gray substance containing a dense mass of fibers. Between them and the pyramids pass the twelfth cranial nerves. There are also accessory olives situated about the main olivary bodies.

The **gray matter** of the medulla and fourth ventricle contains chiefly **nuclei of the last ten cranial nerves.** The nuclei of the third

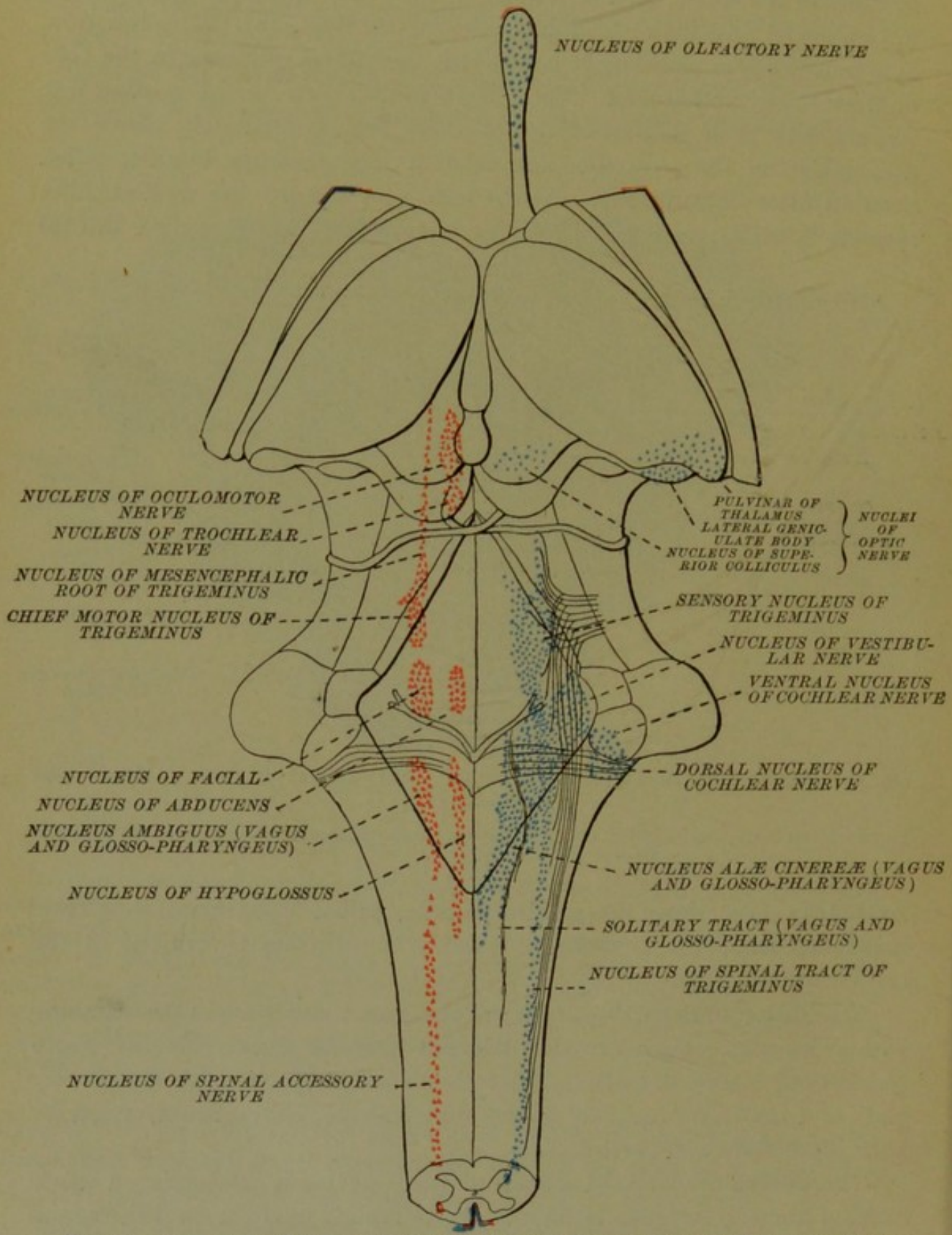


FIG. 15.—SCHEME SHOWING THE RELATIVE SIZE AND POSITION OF THE NUCLEI OF ORIGIN (RED) OF THE MOTOR AND THE NUCLEI OF TERMINATION (BLUE) OF THE SENSORY CRANIAL NERVES. (*Morris' Anatomy.*)

and fourth nerves are situated in the uppermost portion, while the twelfth, tenth and eleventh in the lowest portions. The accompanying illustration shows sufficiently their anatomical seats without entering into a detailed description (Fig. 15).

Sections of the medulla also show the seat of the three pairs of **cerebellar peduncles** and their relation to the **restiform bodies,** which are the continuation of the postero-lateral tracts of the cord and to the **pons,** which can be considered as a continuation of the middle cerebellar peduncles.

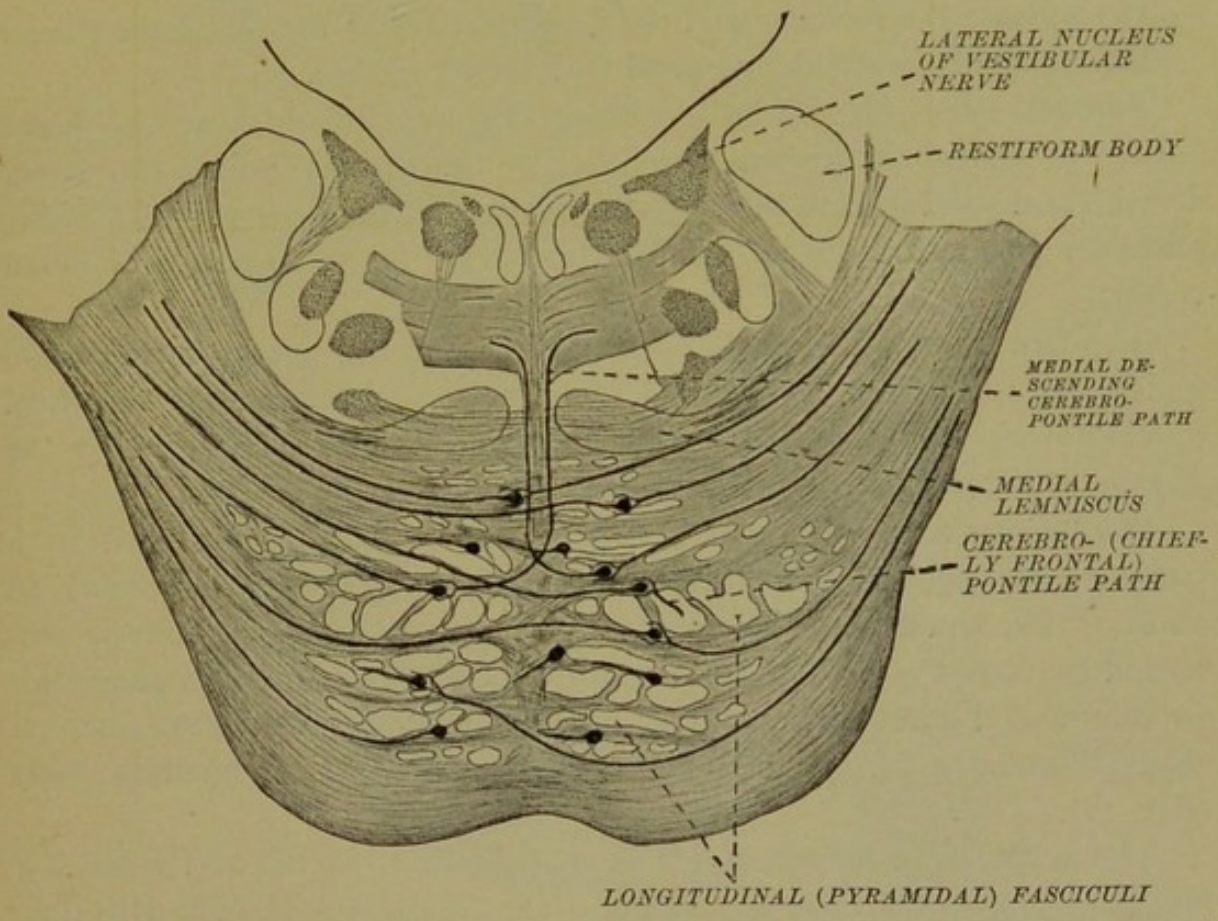


FIG. 16.—DIAGRAM SHOWING CONNECTIONS OF THE FIBERS OF THE PONS. (*Morris' Anatomy.*) The plane of the section is obliquely transverse or parallel with the direction of the brachia pontis.

Sections of the **Pons** show the passage between its deep and superficial fibers of the **longitudinal pyramidal fasciculi,** which are the continuation of the cerebral peduncles (**crura**) and which at the lower border of the pons emerge as pyramids proper. These fibers are situated in the **ventral portion** of the pons. In its dorsal portion (**tegmentum**) the structures are continuous with those of the medulla below (Fig. 16).

The pons also contains separate aggregations of gray matter, called **Nuclei pontis.** They are dispersed between the pontine and pyramidal

fibers. They receive a large portion of these fibers. They therefore form the following connections.

(*a*) Fibers of **cerebellar** hemispheres end in the nuclei pontis of the opposite side.

(*b*) Fibers of **cerebral** hemispheres end in the nuclei pontis, from which new fibers emerge and go to the opposite cerebellar hemispheres.

AREA OF CRURA AND CORPORA QUADRIGEMINA (MESENCEPHALON) (MIDDLE BRAIN)

It connects the medulla and the pons below with the forebrain above.

Exterior.—It presents a **ventral** and **dorsal** surface.

Ventral.—It is formed by the cerebral peduncles which at the upper border of the pons present two thick bundles. Ascending the latter diverge and produce the **interpeduncular space,** the floor of which is the **posterior perforated space.** The latter serves for the passage of blood vessels.

The **Crura** spread in their upward direction and penetrate the brain under the optic tracts. Between their inner surfaces emerge the third (oculomotor) nerves. Their external surface, which is covered by the Temporal lobes, is surrounded by the fourth (pathetic) nerves.

Dorsal Surface.—The roof of the middle brain is constituted by a lamina surmounted by **quadrigeminal bodies.** The latter present an anterior pair and a posterior pair. Externally each anterior body is connected by means of a white bundle (**anterior brachium**) with a ganglionic swelling called **external geniculate body;** a **posterior brachium** connects each posterior quadrigeminal body with an **internal geniculate body** (Fig. 17).

The **anterior quadrigeminal bodies** with their brachia and geniculate bodies belong to the **optic apparatus,** while the posterior bodies with their attachments form a part of the **auditory apparatus.**

Posteriorly to the quadrigeminal lamina are seen the following elements: (1) a thick-white tract, viz. frenulum of the anterior medullary velum, situated between the anterior quadrigeminal bodies; (2) from each side of this tract emerge the fibers of the fourth (pathetic) nerve; (3) the termination of the superior cerebellar peduncles which disappear under the posterior quadrigeminal bodies.

Between the quadrigeminal bodies dorsally and the crura ventrally lies a funnel-shaped cavity, which connects the fourth ventricle below and the third ventricle in front, viz. aqueduct of Sylvius (Fig. 18).

Interior of the Mid-brain.—A transverse section shows a **ventral**

and **dorsal portion.** In the ventral are seen the longitudinal pyramidal fasciculi covered by a pigmented stratum of gray matter (**locus niger**). The dorsal portion (**tegmentum**) consists of **red nuclei,** in which apparently terminate the superior cerebellar peduncles, the gray matter surrounding the aqueduct of Sylvius, the lamina supporting the quadrigeminal bodies and the continuation of other formations of the medulla.

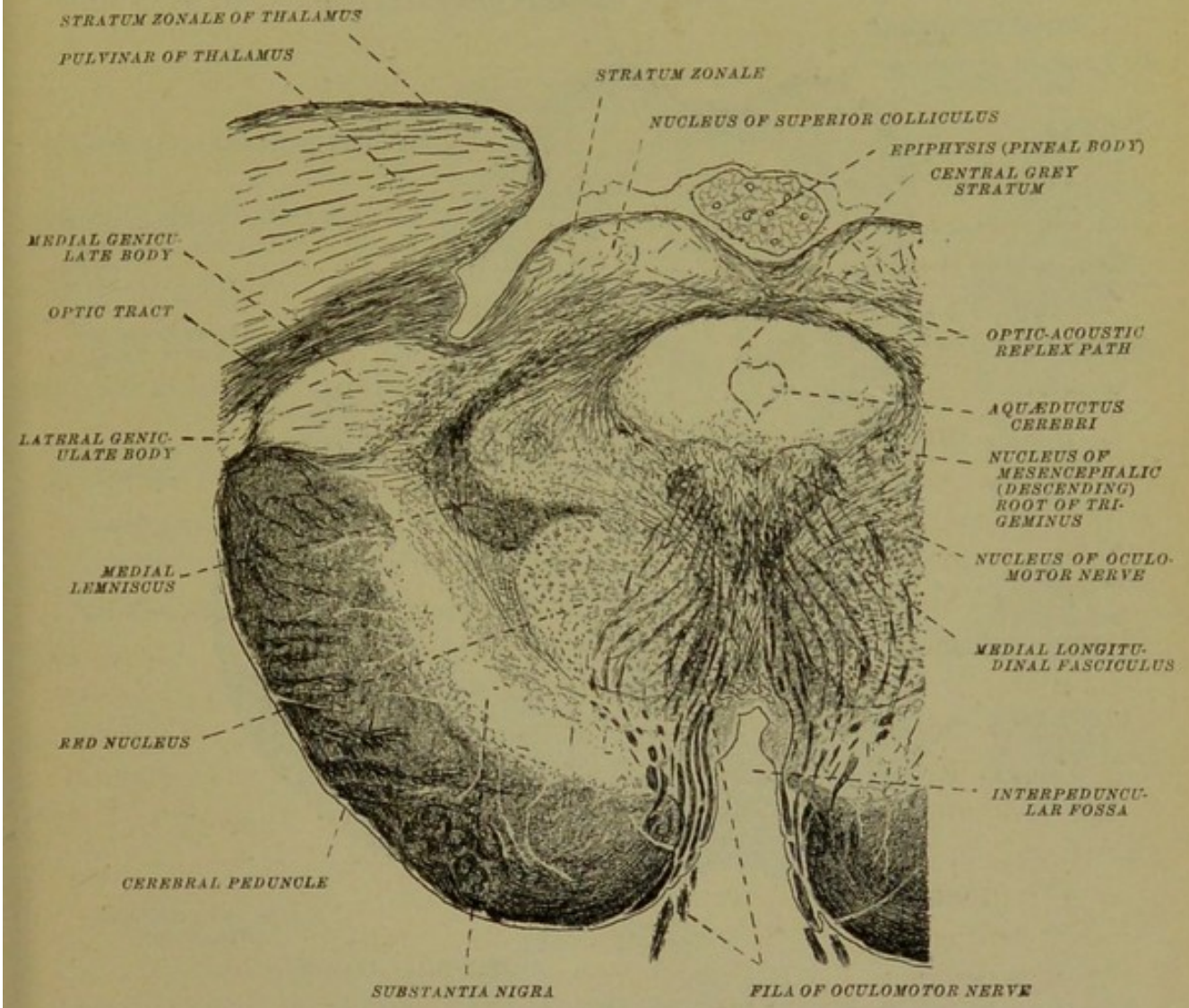


FIG. 17.—TRANSVERSE SECTION THROUGH LEVEL OF SUPERIOR QUADRIGEMINAL BODIES. (*Morris' Anatomy.*)

The **median** and **lateral** lemniscus of the medulla are here fully developed. The largest part of the latter is connected with the nucleus of the cochlear nerve and terminate in the posterior longitudinal bodies; they have therefore an **auditory function.** The median lemniscus (see Medulla) in its upward passage, after having received sensory fibers from the nuclei of the cranial nerves, in reaching the mid-brain sends some fibers to the anterior quadrigeminal bodies and terminates in the Thala-

mus opticus and Hypothalamic nucleus, or body of Luys, which is situated immediately below the lateral and anterior nuclei of the Thalamus. From the latter fibers emerge and go through the internal capsule to the sensory area of the cortex.

The **posterior longitudinal bundles** of the medulla are found here in

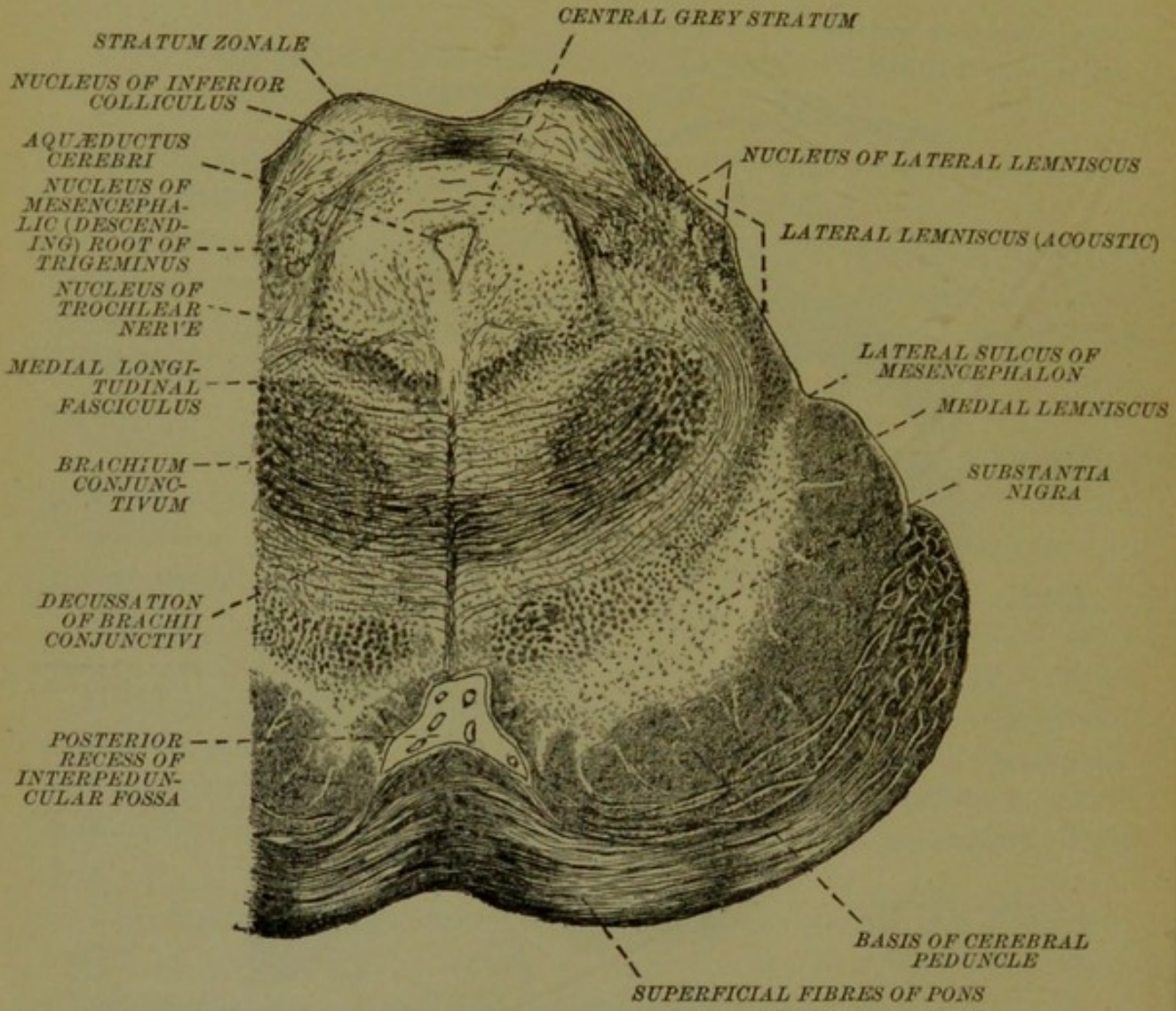


FIG. 18.—TRANSVERSE SECTION THROUGH THE INFERIOR QUADRIGEMINATE BODIES. (*Morris' Anatomy.*)

the most intimate connection with various nuclei of nerves supplying the eye muscles and other cranial nerves.

The **gray matter** is the continuation of the same matter of the cord and medulla. Three cranial nerves are in connection with the mid-brain, viz. third, fourth and fifth nerves. The nuclei of the third and fourth form a continuous column of nerve-cells situated in the gray matter surrounding the aqueduct of Sylvius. A section through the posterior

quadrigeminal bodies shows the origin of the fourth nerves and through the anterior bodies the origin of the third nerves (Fig. 19).

The nucleus of the third nerve is connected with the optic tract by means of neurones originating in the anterior quadrigeminal bodies, with the fourth, sixth and seventh nerves through the posterior longitudinal bundle (see Medulla) and with the eighth nerve through the same bundle and lemniscus.

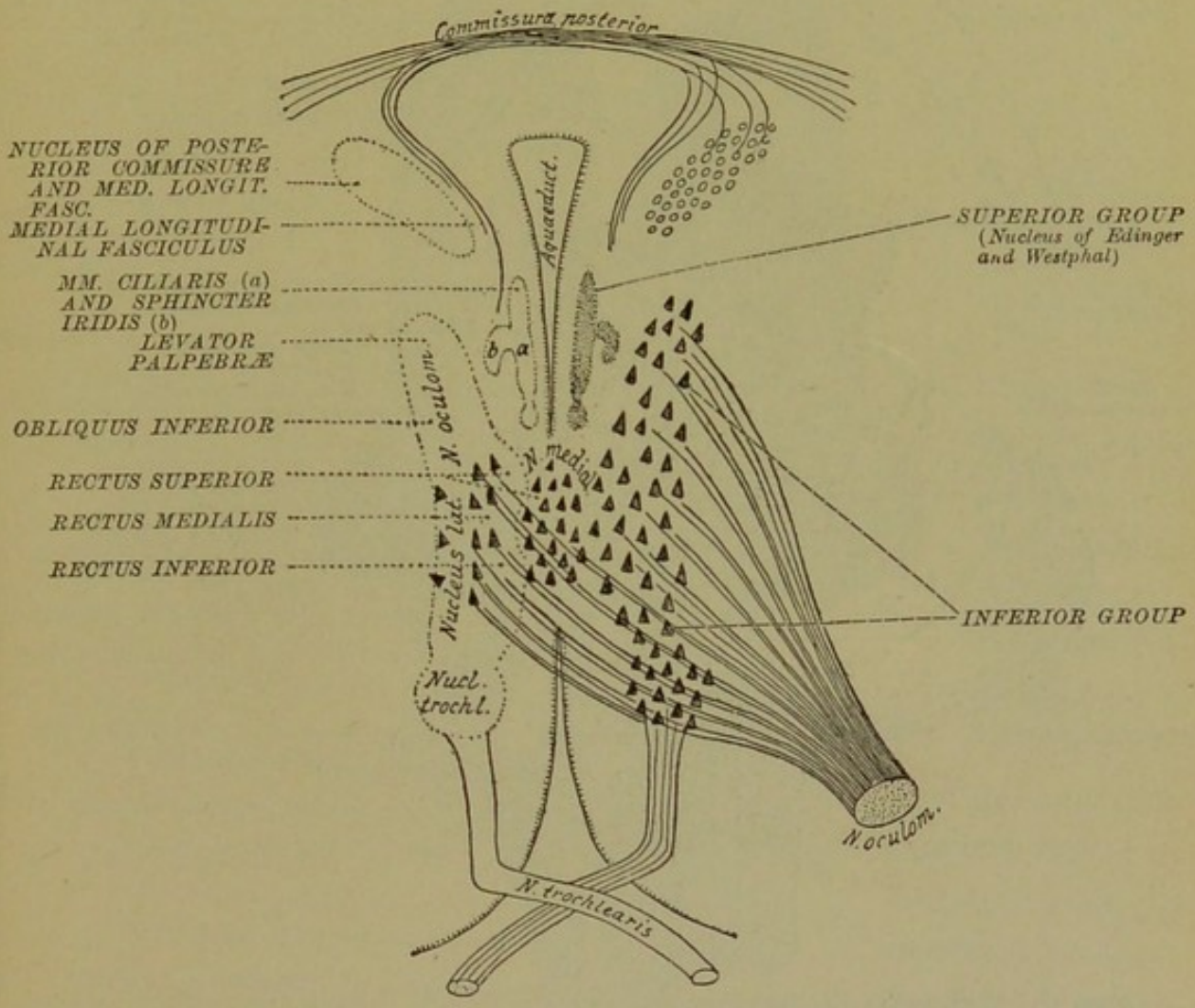


FIG. 19.—DIAGRAM OF LONGITUDINAL SECTION OF NUCLEUS OF OCULOMOTOR NERVE. (*Morris' Anatomy, after Edinger.*)

As to the nucleus of the fifth (trigeminus) nerve, its motor portion with the descending motor fibers is found in the mid-brain. The **Red Nuclei** are two round pigmented masses of gray matter, situated in the tegmentum under the anterior quadrigeminal bodies. They receive fibers from the cerebral cortex and corpus striatum, also send out fibers to the thalamus and spinal cord (Fig. 20).

AREA OF OPTIC THALAMI, THIRD VENTRICLE. DIENCEPHALON. INTERBRAIN

The **Optic Thalami** are two voluminous ovoid masses of gray substance situated in front of and laterally to the quadrigeminal bodies (see preceding chapter) and on both sides of the third ventricle. Close to each other in front they diverge posteriorly.

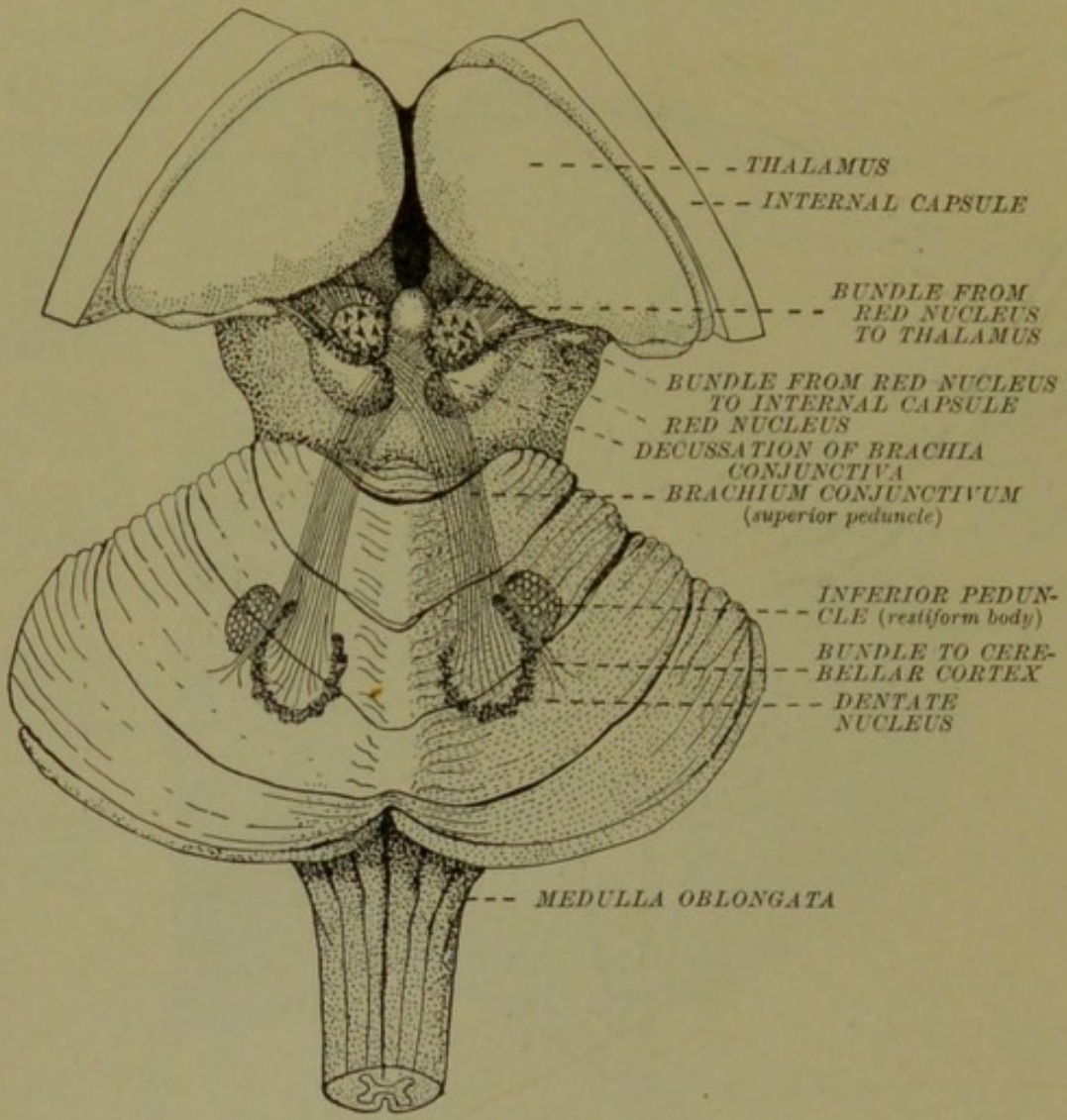


FIG. 20.—TRANSPARENCY DRAWING SHOWING THE ORIGIN, COURSE AND CONNECTIONS OF THE SUPERIOR CEREBELLAR PEDUNCLES (BRACHIA CONJUNCTIVA) IN THE FORMATION OF "STILLING'S SCISSORS." (*Morris' Anatomy.*)

The **upper surface** (stratum zonale) is white. On its anterior portion it presents an elevation, called the **anterior tubercle** or nucleus. The posterior elevated portion is called **pulvinar.** There is also a **medial nucleus.** On the postero-internal portion of the same surface there is the **trigonum habenulæ.** The external margin is separated from the adjacent caudate nucleus by a linear white substance (**tænia semicircularis**).

The **lower surface is** adherent to the **cerebral peduncles.**

The **outer surface** is in connection with the internal capsule and caudate nucleus.

The **inner surface** forms the walls of the third ventricle; in its anterior half it is united with that of the opposite side by a **gray commissure.**

The **Geniculate bodies** lie close to the Thalami. The external gen-

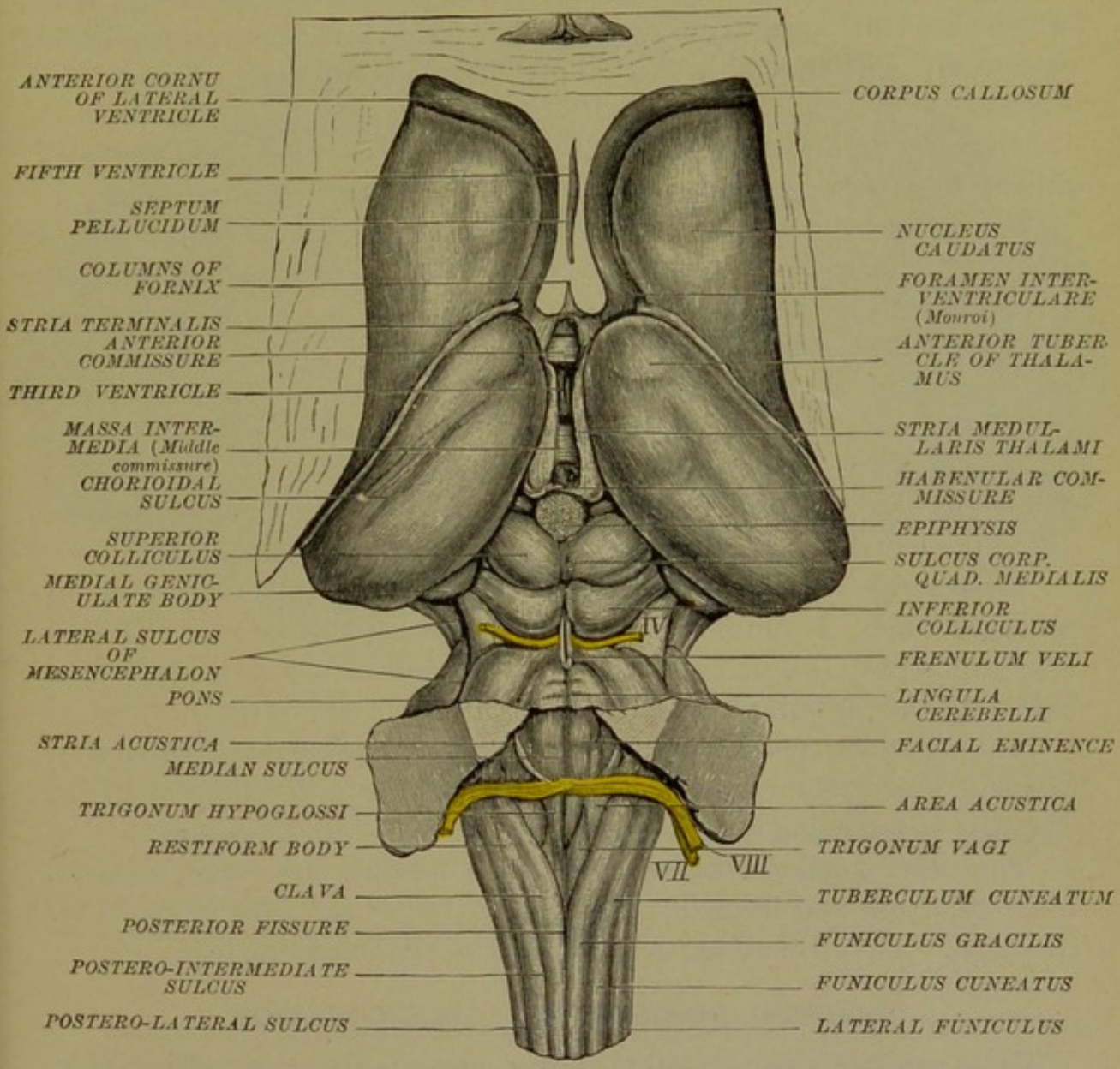


FIG. 21.—DORSAL SURFACE OF DIENCEPHALON WITH ADJACENT STRUCTURES (*Morris, after Obersteiner.*)

iculate body is closely attached to the posterior extremity or base of the Thalamus (pulvinar) and through the anterior brachium is connected with the anterior quadrigeminal body (Fig. 21).

Third Ventricle.—It is a single cavity situated between the optic thalami, beneath the fornix. The **floor** is represented from the anterior

angle backward by the posterior perforated space, mammillary bodies, tuber cinereum, optic chiasma.

The **lateral** surfaces are formed by the thalami.

The **anterior border** extends from the foramen of Monro down to the optic chiasma and contains the two anterior pillars of the fornix between which lies the anterior white commissure.

The **roof** is formed of the tela chorioidea covered by the fornix, upon which lies the corpus callosum. The third ventricle communicates posteriorly with the aqueduct of Sylvius and anteriorly through two openings (foramina of Monro) with the lateral ventricles.

In connection with the thalamic area must be mentioned the **Epithalamus** or **Epiphysis (pineal body)** and the **Hypophysis (pituitary gland).**

Epithalamus or Epiphysis (pineal body).—It develops on the roof of the third ventricle. It presents an ovoid body of 10 mm. long, situated posteriorly at the entrance of the third ventricle in the groove between the anterior quadrigeminal bodies. It is fixed by its adherence to the pia-mater and by its continuity with the walls of the third ventricle. From its base appear two bands (striæ pinealis) which extend anteriorly upon the upper border of the third ventricle, one on each side. Just below and lateral to the epiphysis there is a small group of nerve cells, called the habenular nucleus.

Hypophysis or Pituitary Body.—It lies in the sella turcica of the sphenoid bone. It consists of **two portions:** a large anterior or glandular lobe and a posterior or cerebral lobe. The infundibulum is continuous only with the posterior lobe.

The glandular portion originates from a diverticulum of the buccal cavity. The cerebral portion comes from the floor of the third ventricle. The first is ascending and the second is descending in development.

The other portions belonging to the interbrain are: optic chiasma, tuber cinereum and infundibulum. The latter is the apex of the tuber cinereum. They are all parts of the floor of the third ventricle.

CEREBRAL HEMISPHERES. TELENCEPHALON

They are two symmetrical masses of nervous tissue, ovoid in shape. They occupy the cranial cavity. Their surface is gray (cortex). The average length is 16 cm., width 13 cm., height 12 cm. They present a **Convexity** and **Base.**

A **longitudinal fissure** separates the two hemispheres. From the cerebellum, over which lie the occipital lobes, and from the mesencephalon (see above) they are separated by a transverse fissure. Each hemisphere presents an **external, mesial** and **inferior surface.**

A number of fissures or sulci divide each hemisphere into lobes, lobules, convolutions. The adjoining illustrations are sufficient to give a correct idea of the division and subdivision of the cerebral cortex by the fissures, also of the Base of the Brain with the cranial nerves (Figs. 22, 23, 24, 25, 26, 27).

As to the function of the various areas, see the chapter on Localizations.

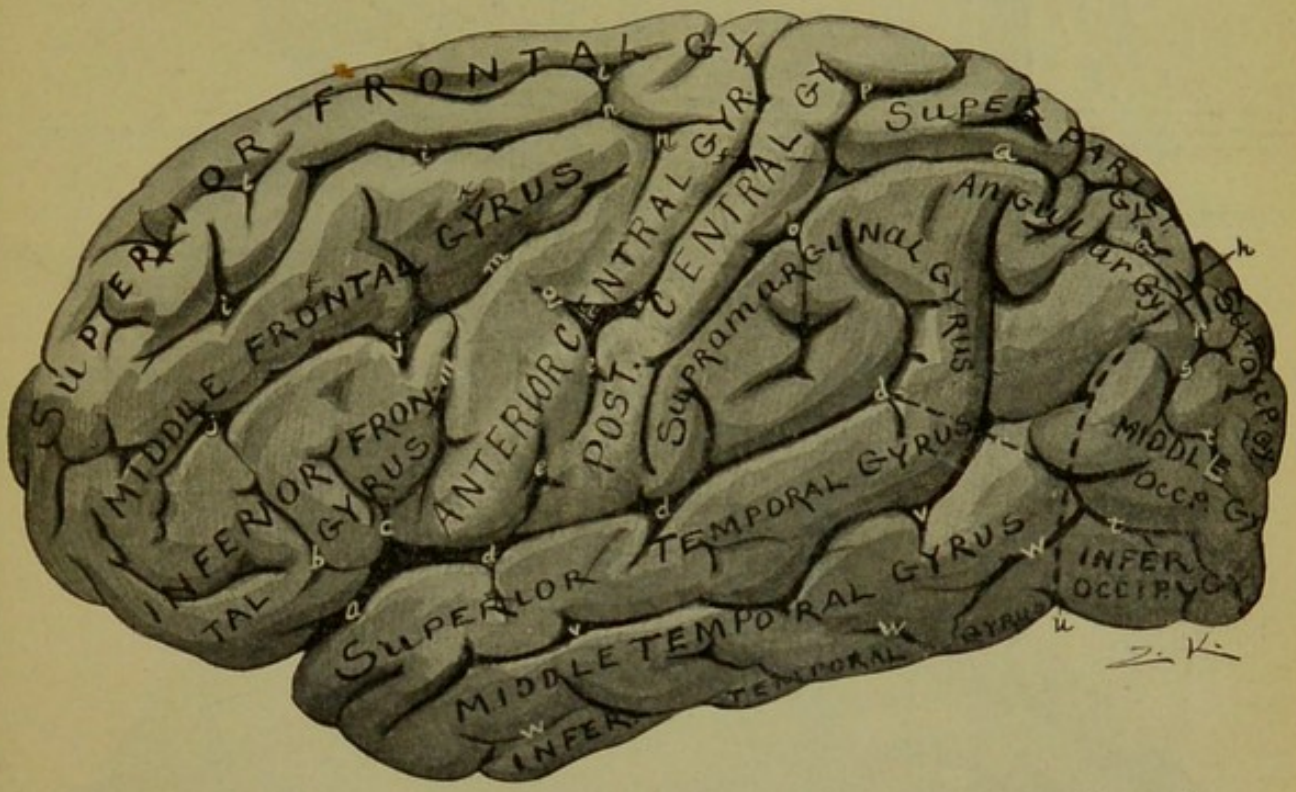


FIG. 22.—GYRI OF CONVEX SURFACE OF LEFT CEREBRAL HEMISPHERE. (*Santee.*)

Fissura lateralis cerebri: *a*. Stem. *b*. Horizontal anterior ramus. *c*. Ascending anterior ramus. *d*. Posterior ramus. *e, e*. Sulcus centralis (Rolandi). *f*. Genu superius. *g*. Genu inferius. *h*. Sul. occipito-parietalis. *i, i, i*. Sul. frontalis superior. *j, j*. Sul. frontalis inferior. *k, k*. Sul. frontalis medius. *l, l*. Sul. paramedialis. *m, m*. Sul. præcentralis inferior. *n, n* Sul. præcentralis superior. *o*. Sul. post-centralis inferior. *p*. Sul. post-centralis superior. *q*. Ramus horizontalis and *r*, ramus occipitalis of interparietal sulcus. *s*. Sul. transversus. *t*. Sulci superior and lateralis. *u*. Incisura præoccipitalis. *v*. Sul. temporalis superior. *w*. Sul. temporalis medius.

Interior of the Brain.—A horizontal section through both hemispheres reveals the presence of **gray** and **white** matter, also of **lateral ventricles.**

Gray Substance.—Besides the cortical gray matter there are also isolated masses called **basal ganglia.** The latter are three in number, viz. **Thalamus, Caudate** and **Lenticular** nuclei. The two last ones are also known under the name of **Corpora striata.** The thalamus was discussed in the preceding chapter (Fig. 28).

Caudate Nucleus belongs to the wall of the lateral ventricle. It forms the floor in the upper portion and the roof in the lower portion of

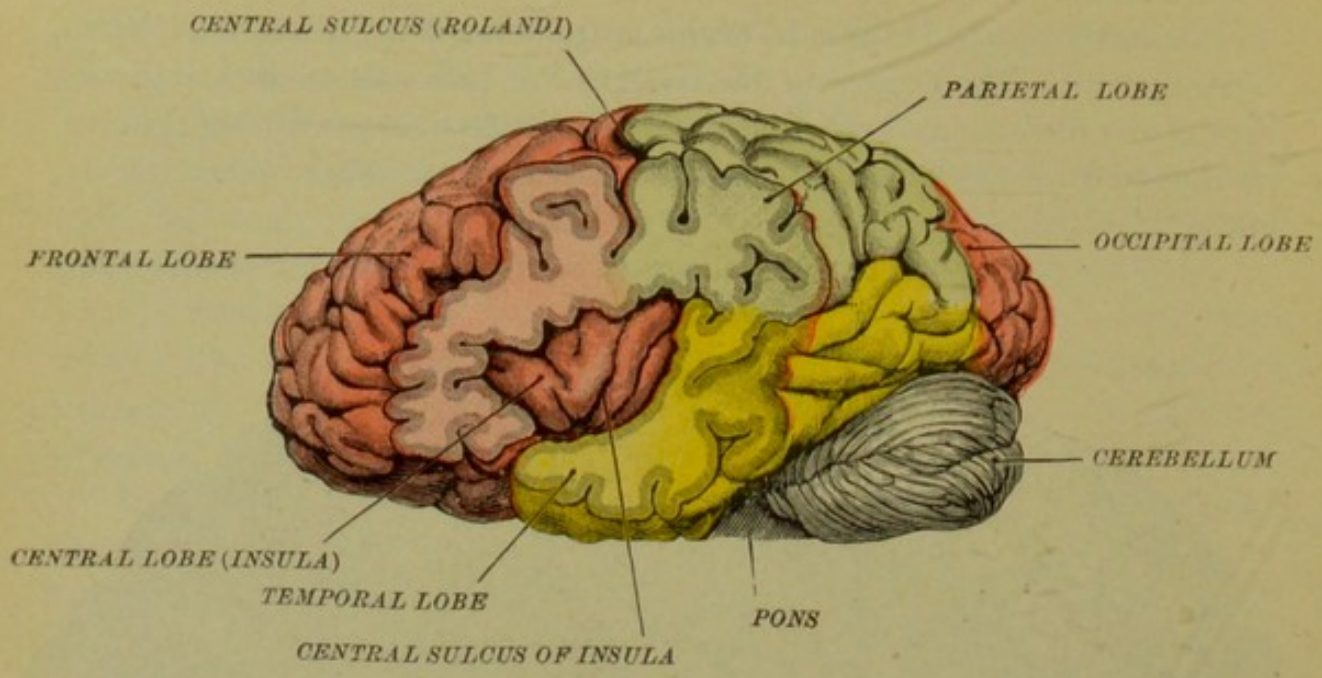


FIG. 23.—DIAGRAM OF THE CONVEX SURFACE OF THE LEFT CEREBRAL HEMISPHERE SHOWING THE FIVE PRINCIPAL LOBES OF THE PALLIUM. (*Morris' Anatomy.*)

The opercular regions of the frontal, pacietal, and temporal lobes are removed to show the central lobe or island of Reil.

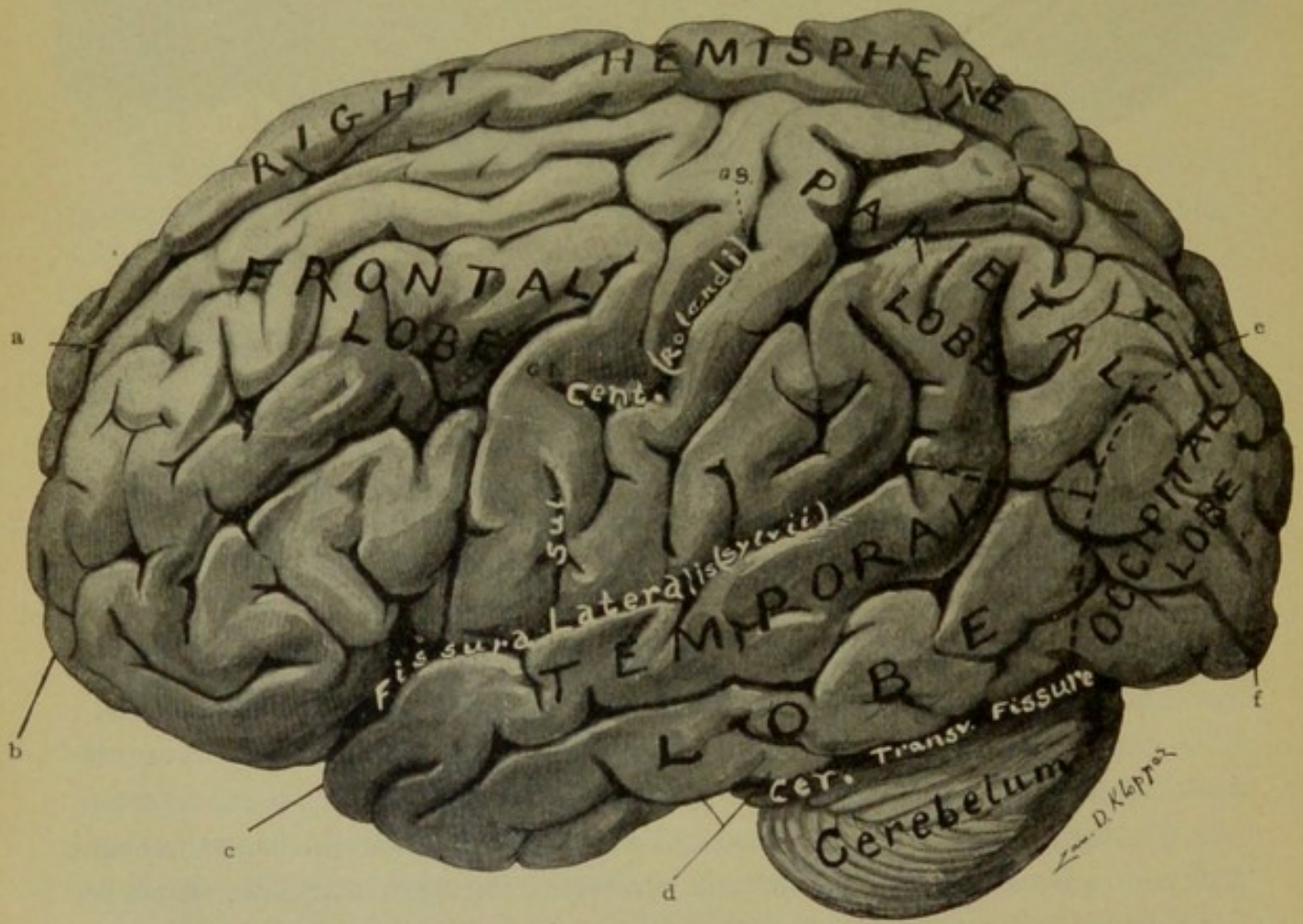


FIG. 24.—LATERO-SUPERIOR VIEW OF BRAIN, SHOWING FISSURES AND LOBES. (*Santee.*)

a. Longitudinal fissure. *b*. Frontal pole. *c*. Temporal pole. *d*. Impressio petrosa. *e*. Occipito-parietal sulcus. *f*. Occipital pole.

the lateral ventricle. The anterior portion (head) is closely connected with the internal capsule, the body is applied to the optic thalamus, the posterior portion passes around the posterior border of the capsules to

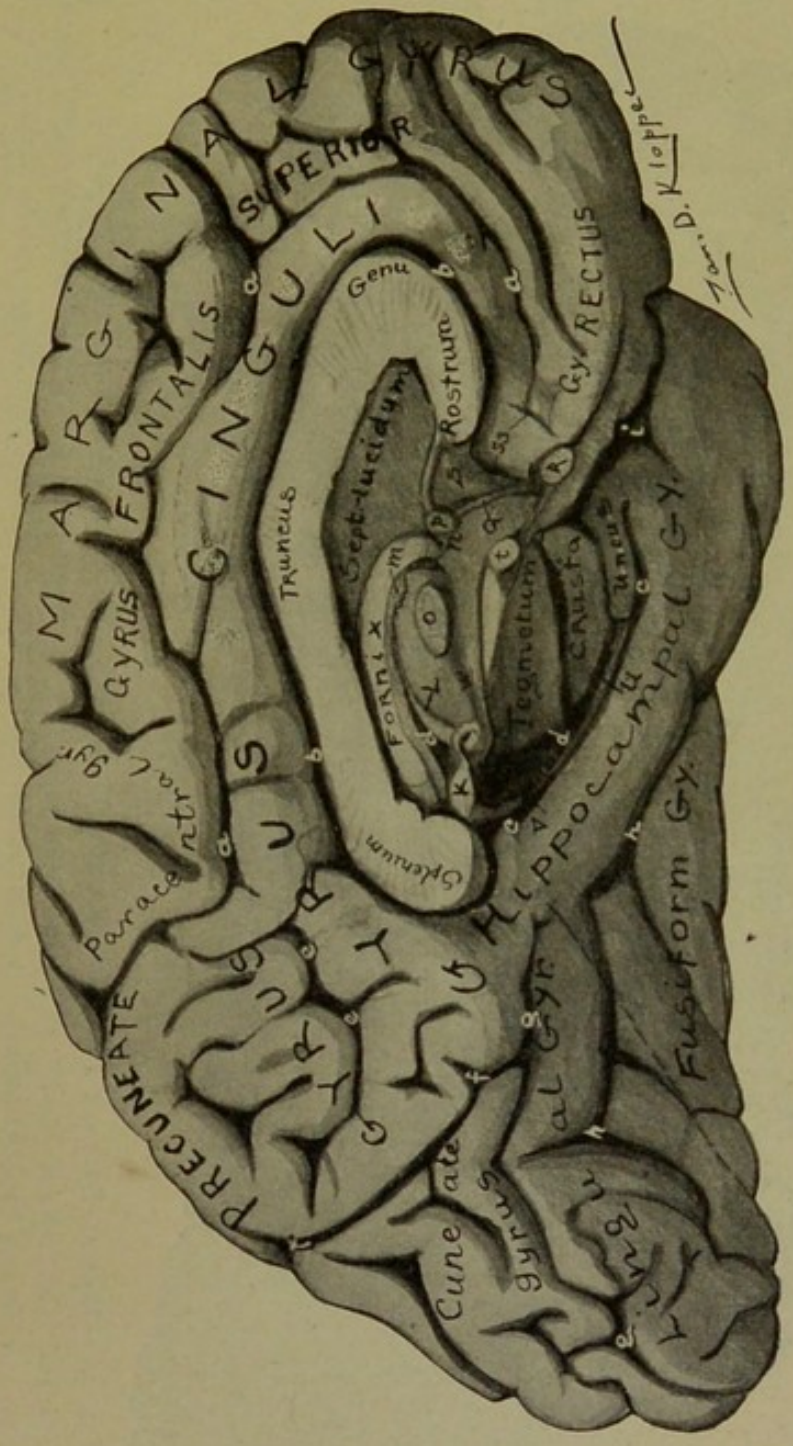


FIG. 25.—GYRI ON MEDIAL SURFACE OF HEMISPHERE. (*Santee.*)

aaa. Sulcus cinguli. *bb*. Callosal sulcus. *cc*. Hippocampal fissure. *dd*. Chorioidal fissure. *ee*. Subparietal sulcus. *ff*. Occipito-parietal sulcus. *gg*. Anterior and posterior calcarine fissure. *hh*. Collateral fissure. *i*. Ectorhinal sulcus. *k*. Pineal body. *l*. Stria medullaris. *m*. Chorioid tela of third ventricle. *n*. Interventricular foramen. *o*. Massa intermedia. *p*. Anterior commissure. *q*. Lamina terminalis. *r*. Optic chiasma. *ss*. Sulci parolfactorii. *t*. Corpus mammillare. *u*. Crus fornicis. *v*. Posterior commissure.

go down into the inferior cornu of the lateral ventricle and then forward to the apex of the temporal lobe.

The **Lenticular Nucleus** is an extra-ventricular body. It is situated between the thalamus and insula, externally and beneath the caudate nucleus. It is separated externally from the insula by a layer of white

substance, called **External Capsule** and the intervening claustrum. The internal surface is in relation with the thalamus, caudate nucleus and internal capsule. The lenticular nucleus is divided by two vertically

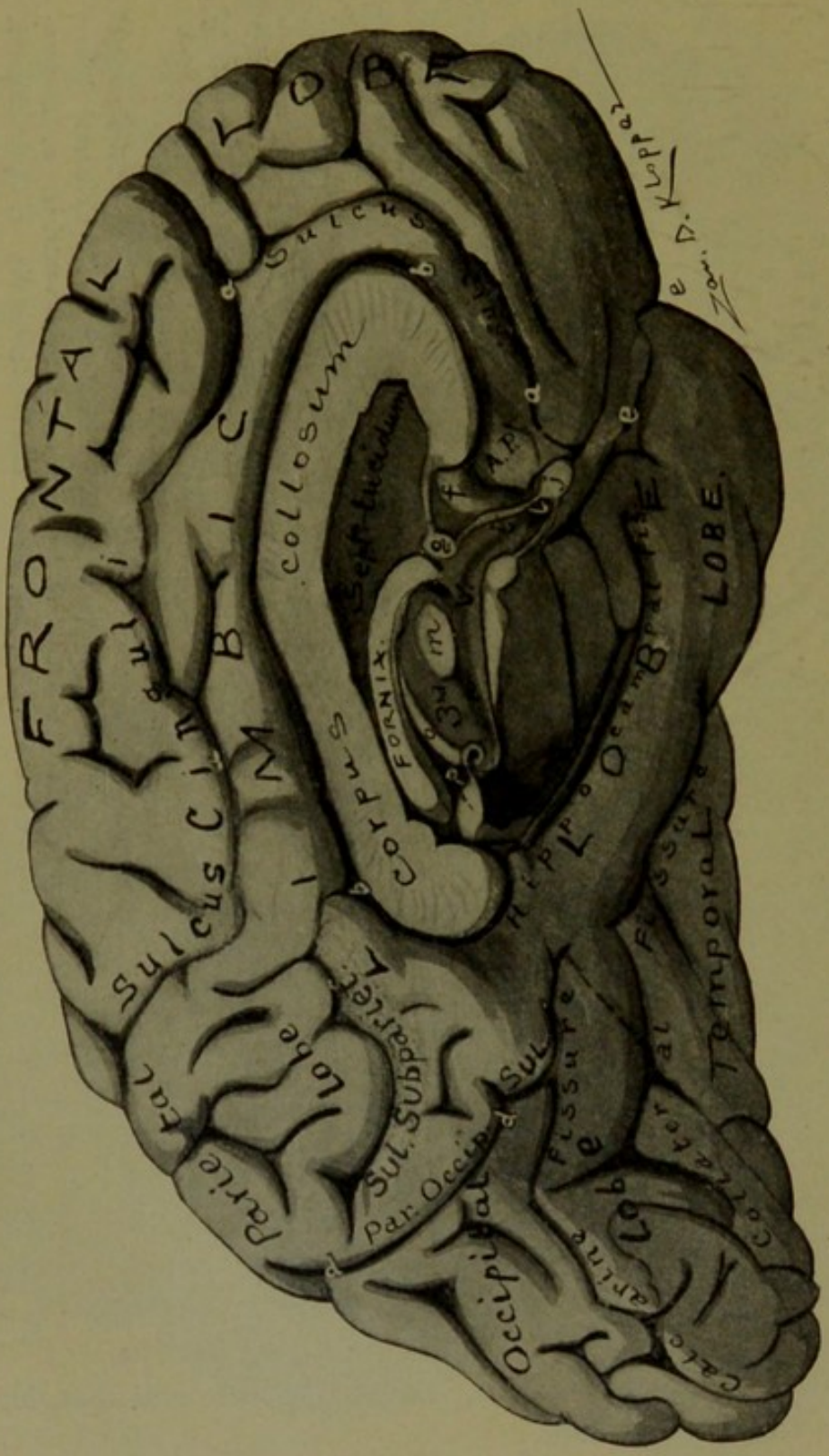


FIG. 26.—MEDIAL SURFACE OF LEFT CEREBRAL HEMISPHERE, SHOWING LOBES AND SULCI. (*Santee.*)
aaa. Sulcus cinguli. *bb*. Callosal sulcus. *cc*. Fissura lateralis cerebri. *f*. Gyrus subcalolsus *i*. Lamina terminalis. *l*. Optic recess. *n*. Sulcus hypothalamicus. *o*. Stria medullaris. *p*. Pineal body. *A*, *P*. Area parolfactoria.

curving laminæ into three portions, viz. **globus pallidus** for the two inner zones and **putamen** for the outer zone.

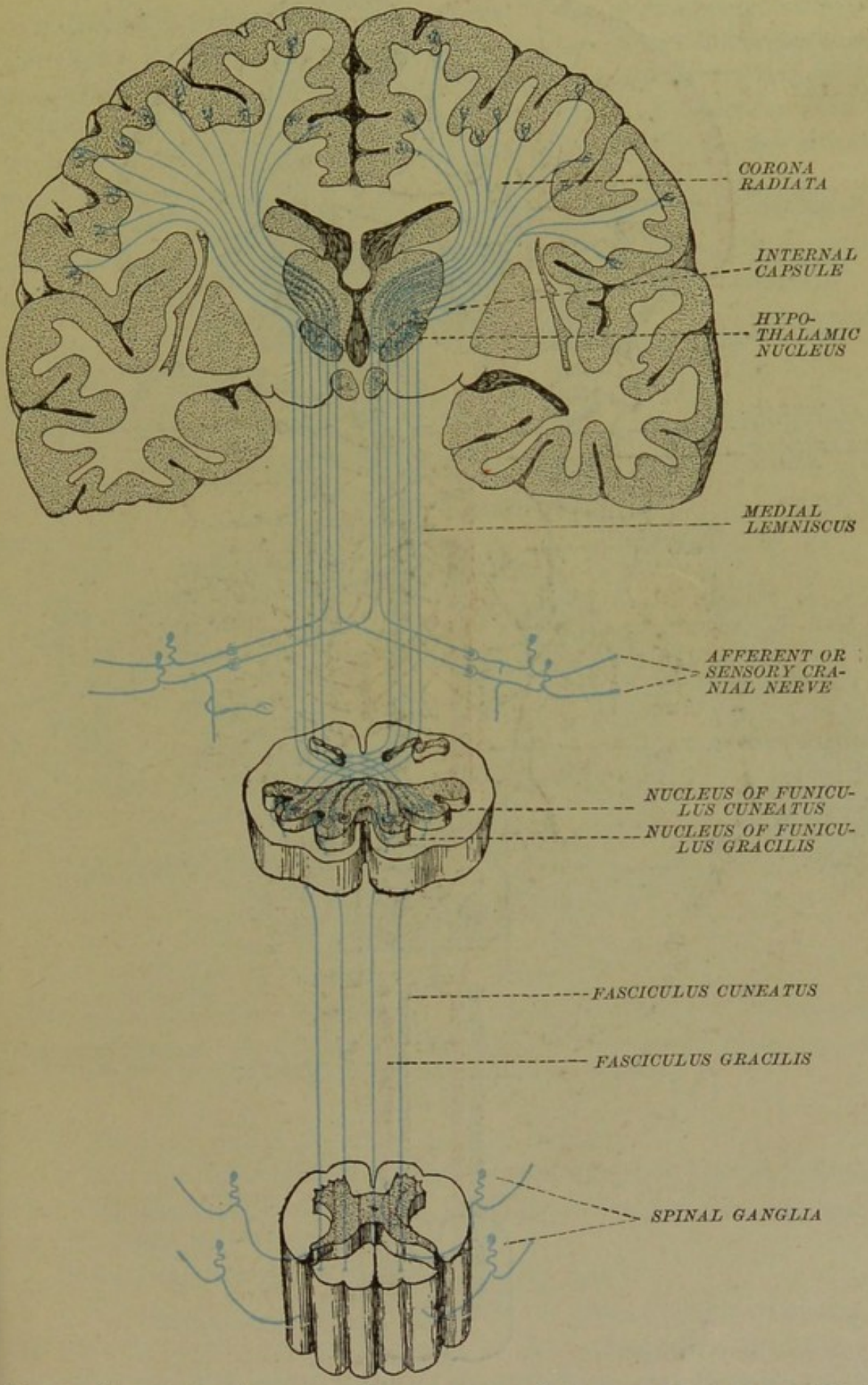


FIG. 29.—SCHEME OF ASCENDING CEREBRO-SPINAL CONDUCTION PATHWAYS. (*Morris' Anatomy.*)

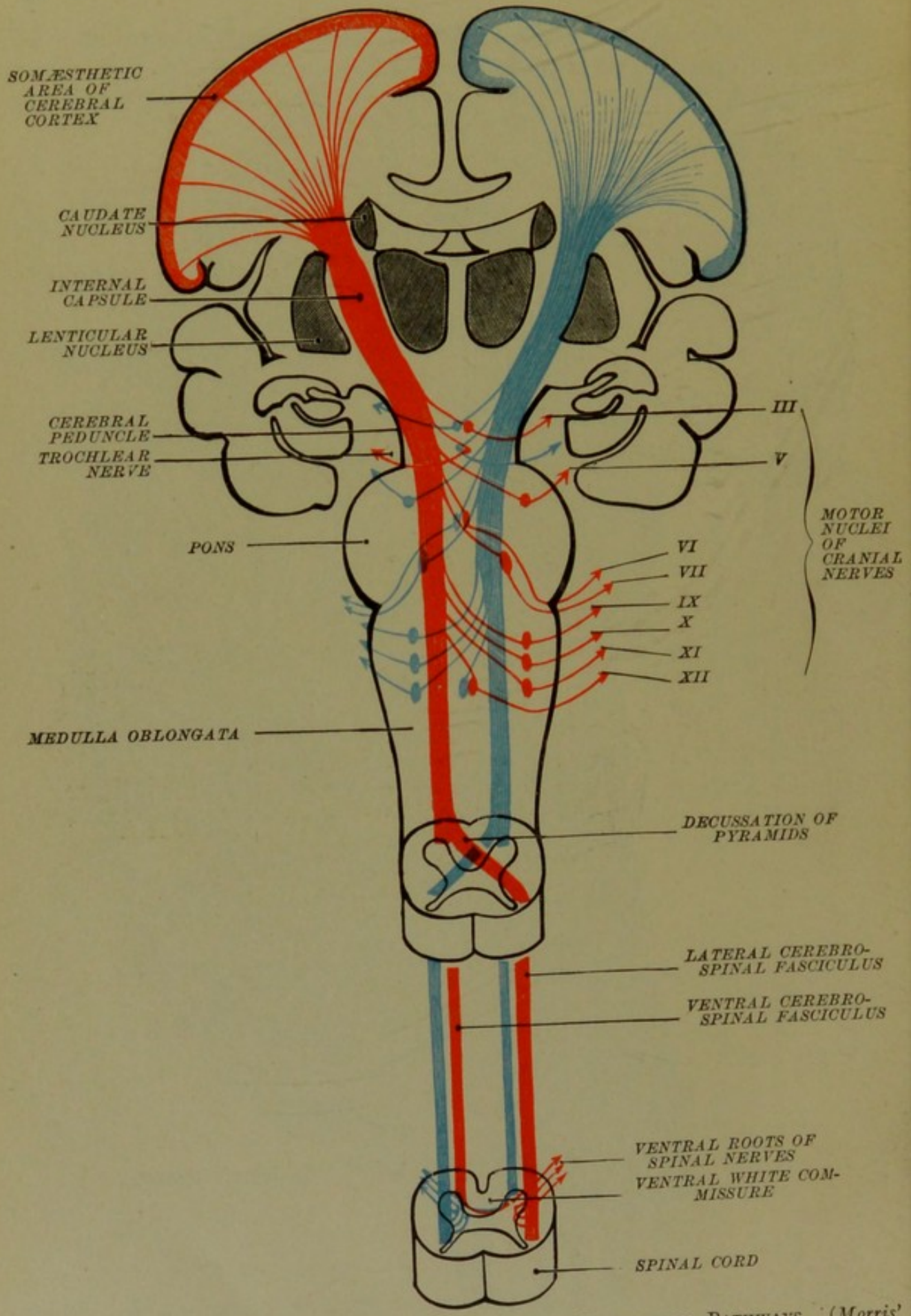


FIG. 30.—SCHEME OF DESCENDING CEREBRO-SPINAL CONDUCTION PATHWAYS. (*Morris' Anatomy.*)

1. **Sensory,** which are the continuation of the median lemniscus (see Medulla). They terminate in the thalamus and subthalamic nucleus, from which new fibers emerge and go through the posterior portion of the posterior limb of the internal capsule to terminate in the sensory area of the cortex. The median lemniscus is joined by fibers from the sensory nuclei of the cranial nerves.

2. The cochlear branch of the eighth nerve also sends projection fibers through the lateral lemniscus to the posterior limb of the internal capsule, from which fibers go to the cortex of the temporal lobe.

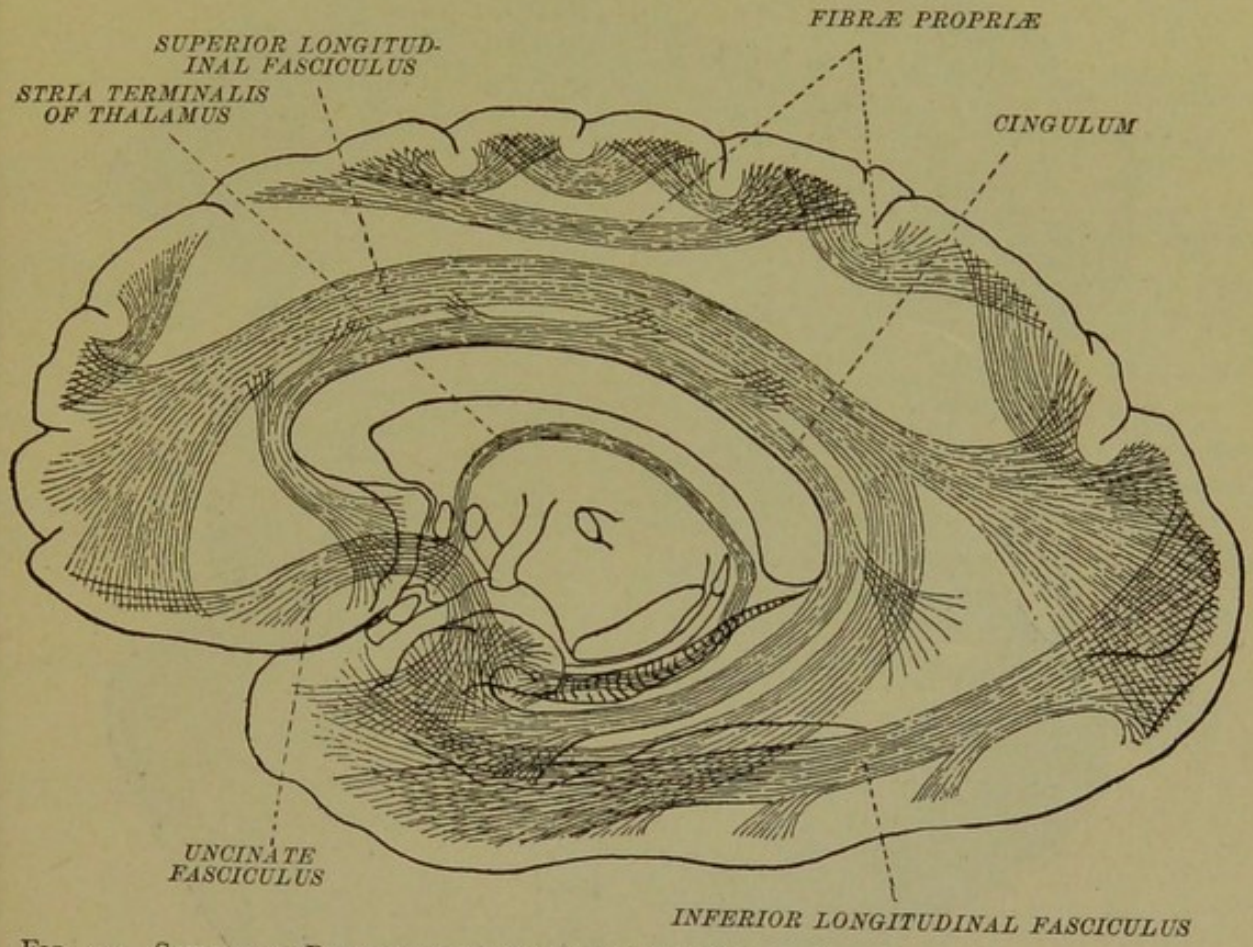


FIG. 31.—SCHEMATIC REPRESENTATION OF CERTAIN OF THE ASSOCIATION PATHWAYS OF THE CEREBRAL HEMISPHERE. (*Morris' Anatomy.*)

3. The optic radiations are mentioned above.

4. The superior cerebellar peduncles terminate mostly in the red nuclei (see Mesencephalon and Thalami), from which new fibers ascend and go to the sensory area of the cortex.

Descending Fibers are the following (Fig. 30).

1. Pyramidal fibers, which originate in the cells of the motor area, pass through the largest part of the posterior limb adjacent to the knee and through the latter, down to the crura, pyramids, and after decussating

in the medulla descend in the cord and terminate around the cells of the anterior cornua.

2. **Temporal Bundle** originates in the cortex of the first two temporal gyri and passing through the posterior limb of the internal capsule descend into the lateral portion of the crura and thence to the nuclei of the pons.

3. **Frontal Bundle** originates in the cortex of the frontal lobe and

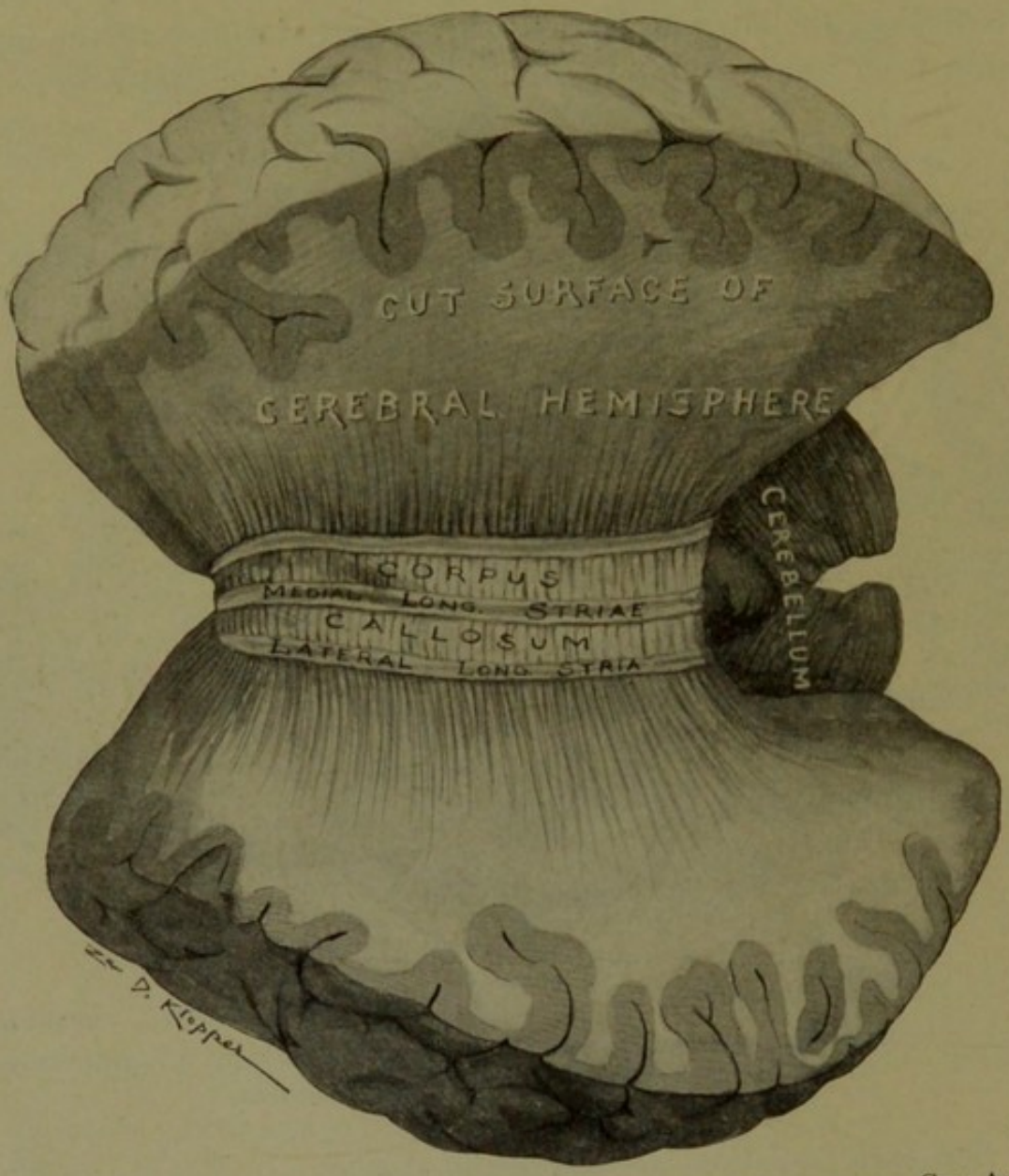


FIG. 32.—DORSAL SURFACE OF CORPUS CALLOSUM, CEREBRAL HEMISPHERE CUT AWAY TO EXPOSE IT. (*Santee.*)

passing through the anterior limb of the internal capsule ends in the nuclei of the pons.

4. **Occipital Bundle** (visual) originates in the cortex of the cuneus and calcarine fissure and passing through the most posterior portion of the internal capsule ends in the anterior quadrigeminal body.

B. **Association Fibers.**—They connect different parts of the same hemisphere. They are the following (Fig. 31).

1. **Superior Longitudinal Bundle** which connects the frontal, temporal and occipital lobes.

2. **Inferior Longitudinal Bundle** which connects the occipital and temporal lobes.

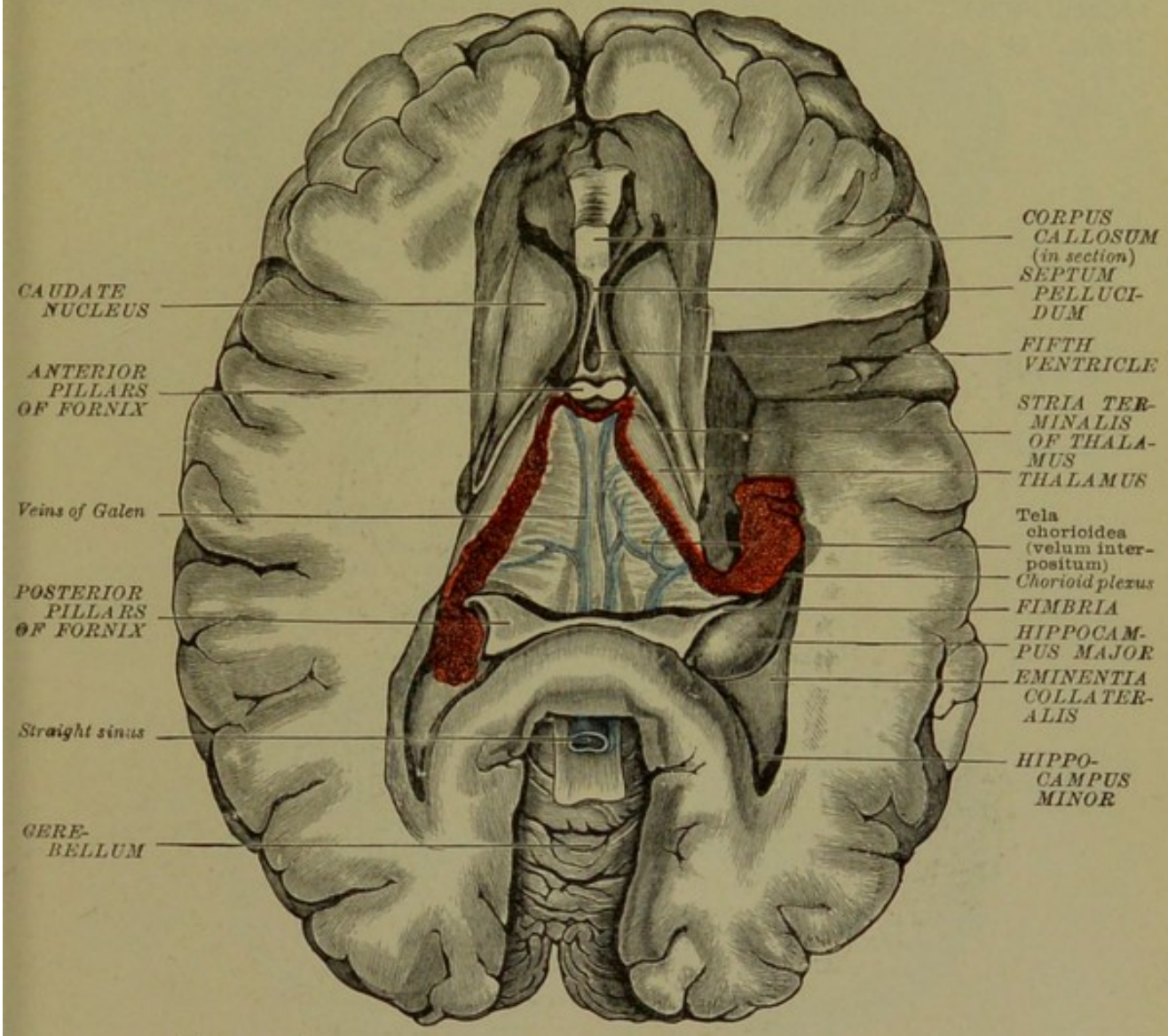


FIG. 33.—HORIZONTAL DISSECTION OF THE CEREBRAL HEMISPHERES.
The fornix has been removed to show the relation of the tela chorioidea of the third ventricle to the chorioid plexus of the lateral ventricles. (From a mounted specimen in the Anatomical Department of Trinity College, Dublin.)

3. **Occipito-frontal Bundle.**

4. Fibræ propriæ connecting contiguous gyri with each other.

5. **Uncinate Bundle** connects the uncus with the frontal lobe.

6. The **Cingulum** (see illustration).

C. **Commissural Fibers.**—They connect one hemisphere with the other. They consist of

1. **Corpus Callosum** (see above, Fig. 32).

2. **Anterior commissure,** the largest part of which connects one temporal lobe with another.

3. **Hippocampal commissure** connects the two gyri of the same name.

Lateral Ventricles.—Each of these cavities commences within the frontal lobe, is directed backward, turns around the optic thalamus to go again forward and downward and terminate in the apex of the temporal lobe. It presents an **anterior, posterior** and **inferior cornu** and a central

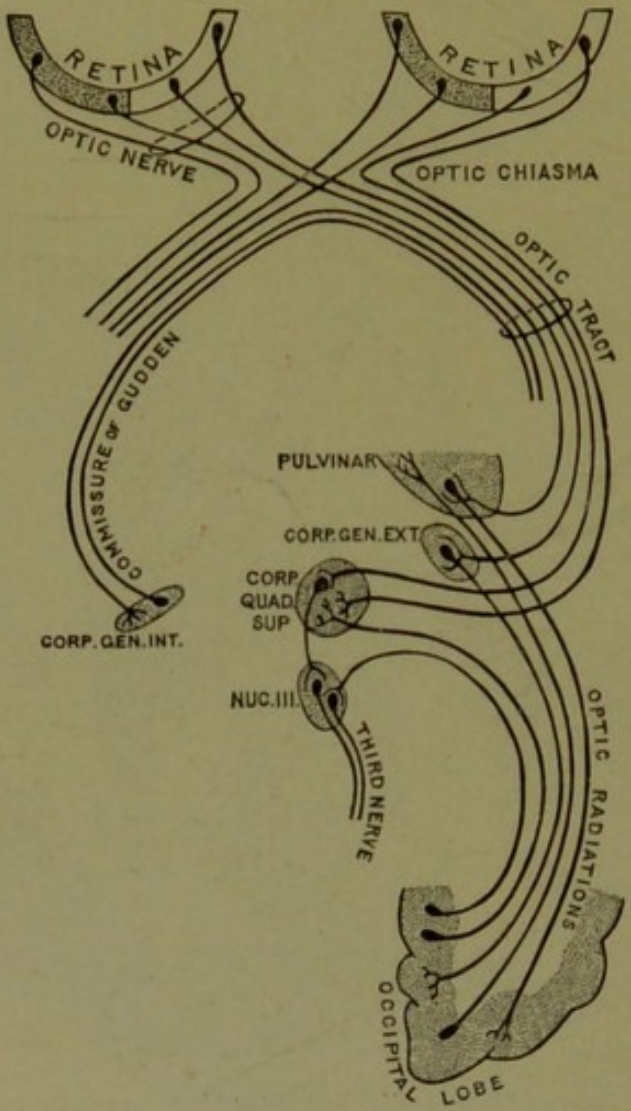


FIG. 34.—DIAGRAM OF PRINCIPAL PATHWAYS OF OPTIC APPARATUS. (*Morris, after Cunningham.*)

portion or **body.** Its relation to various portions of the brain has been already mentioned. It communicates with the third ventricle by means of the foramen of Monro situated in the frontal portion of the latter. The **tela choroidea** of the third ventricle is continuous into the lateral ventricles and the varicose mass composed of blood vessels found in the lateral ventricles is known under the name of choroid plexus. The anterior portions

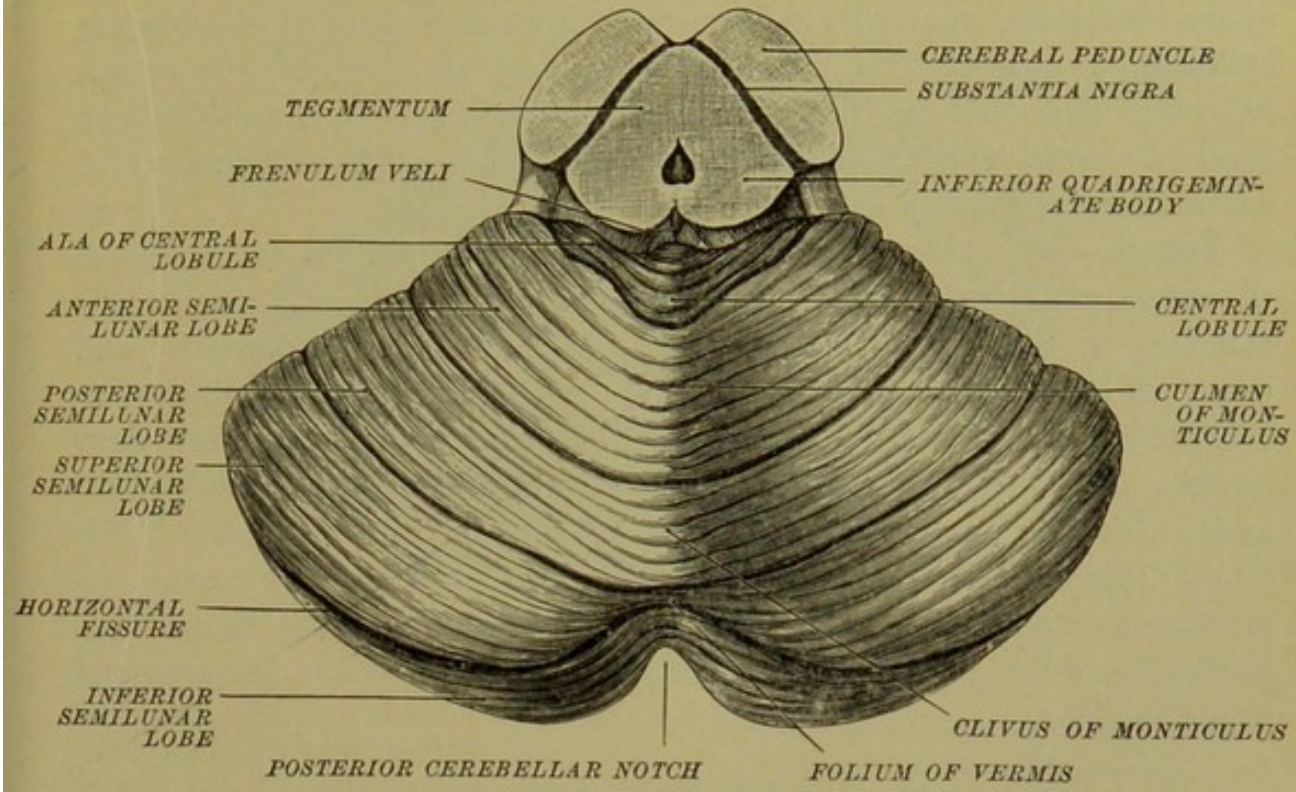


FIG. 35.—DIAGRAM OF THE DORSAL SURFACE OF THE CEREBELLUM. (*Morris' Anatomy.*)

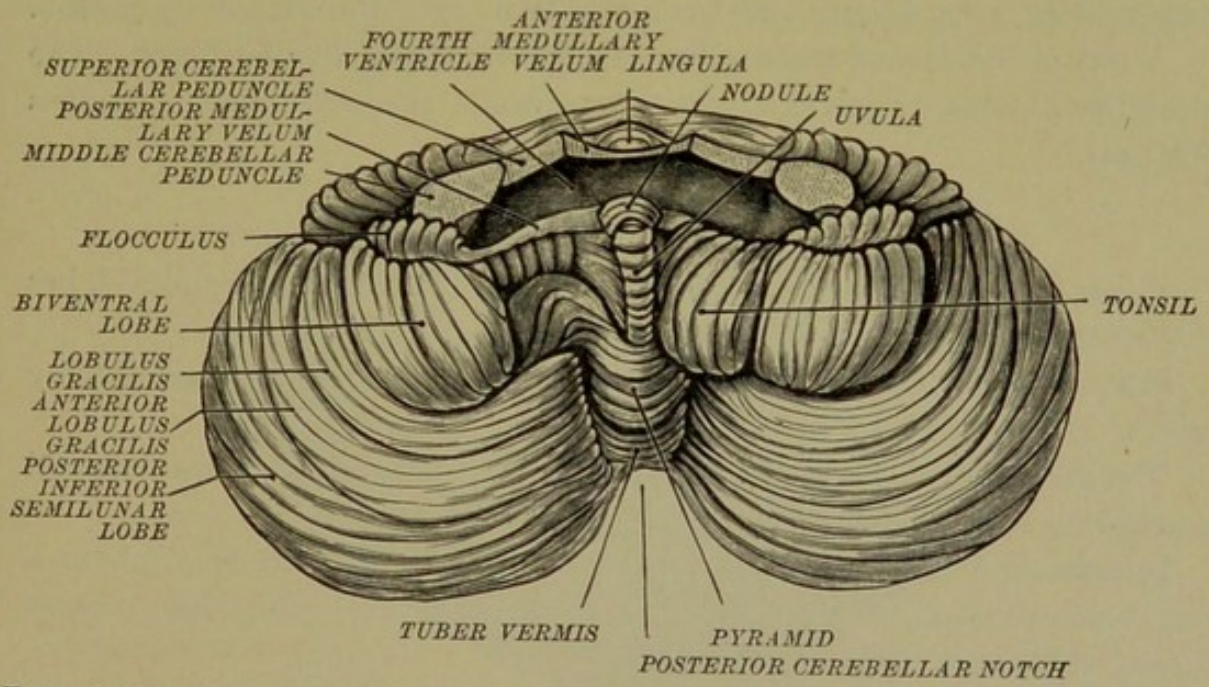


FIG. 36.—DIAGRAM OF THE VENTRAL AND INFERIOR SURFACE OF THE CEREBELLUM AFTER THE REMOVAL OF THE MEDULLA OBLONGATA, PONS, AND MESENCEPHALON. (*Morris' Anatomy.*)

The tonsil of the right side is omitted in order to display the connection of the pyramid with the biventral lobe, the furrowed band of the uvula, and more fully the posterior medullary velum. The anterior notch is less evident than in the actual specimen.

of the two lateral ventricles are separated from each other by the **Septum lucidum,** a thin vertical membrane attached in front to the corpus callosum and the anterior pillars of the fornix. It consists of two layers, between which there is a closed cavity called **fifth ventricle,** which has no communication with other ventricles (Fig. 33).

Fornix.—It is placed beneath the corpus callosum. It extends from the splenium to the anterior portion of the corpus callosum, to which it is attached by the septum lucidum. It presents a body and anterior and posterior pairs of bands (columns or pillars). The **anterior** pillars run forward and downward, and appear at the base of the brain as **mammillary** bodies. The **posterior** pillars curve backward, downward and forward and end in the uncus. The fornix is an **association tract** of the limbic lobe. The posterior pillars connect the hippocampal gyri by means of a lamina situated between them. Fibers emanating from the mammillary bodies (anterior pillars) go to the thalamus and crura of the same and opposite side.

OPTIC APPARATUS

In discussing the structure of the brain various portions of white and gray matter were mentioned in connection with the **visual apparatus.**

A recapitulation of those elements will be useful in this place. The adjoining illustration gives a satisfactory idea of the entire optic apparatus (Fig. 34).

CEREBELLUM

It lies in the posterior fossæ of the cranium, under the occipital lobes of the cerebrum, behind the medulla oblongata.

Exterior.—Similarly to the cerebrum, the cerebellum is divided by fissures and sulci into lobes and lobules (Figs. 35 and 36).

The adjoining illustrations give a sufficiently clear idea of the external appearance of the cerebellum.

Interior.—A section of the cerebellum reveals in each hemisphere a gray cortical mass, central white matter and several ganglionic masses of gray substance (Figs. 37 and 38).

Gray Matter.—It is presented by the **Dentate** nucleus and **accessory nuclei.**

The **Dentate nucleus,** situated in the center of the white substance, presents an ovoid and folded lamina with an opening (hilus). It resembles the olivary bodies of the medulla.

Three **accessory** small nuclei are situated near the dentate nucleus.

White Matter.—In addition to the central white mass, there are also

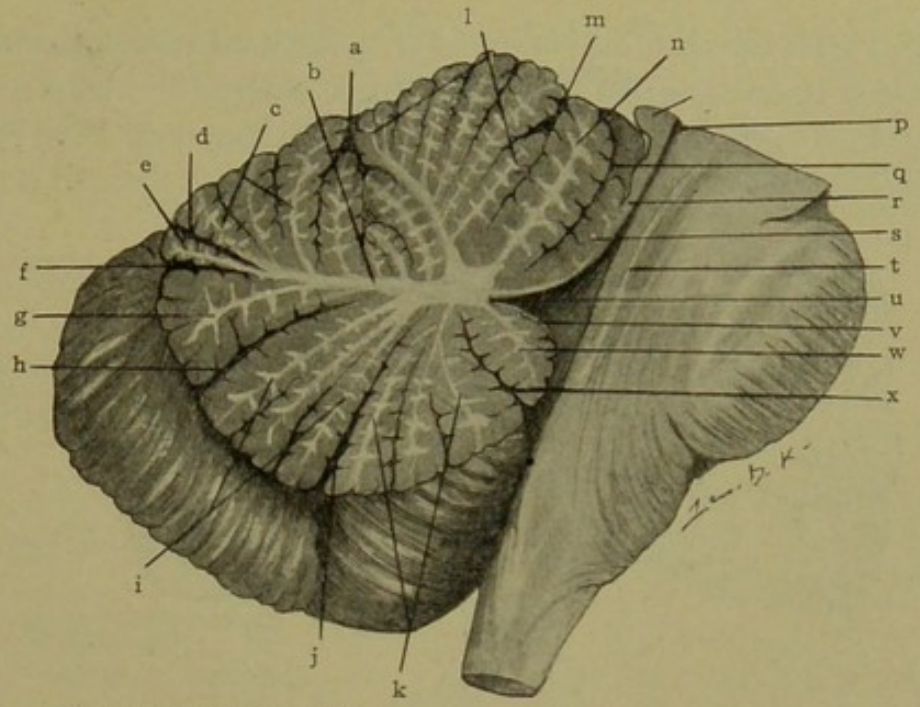


FIG. 37.—MEDIAN SECTION OF CEREBELLUM, PONS AND MEDULLA. (*Santee.*)
a. Predeclivil sulcus. *b*. Arbor vitæ. *c*. Declive monticuli. *d*. Postdeclivil sulcus. *e*. Folium vermis. *f*. Horizontal sulcus. *g*. Tuber vermis. *h*. Postpyramidal sulcus. *i*. Pyramid. *j*. Prepyramidal sulcus. *k*. Uvula. *l*. Culmen monticuli. *m*. Postcentral sulcus. *n*. Central lobule. *o*. Inferior colliculus of corp. quad. *p*. Cerebral aqueduct. *q*. Precentral sulcus. *r*. Superior medullary velum. *s*. Lingula. *t*. Medial longitudinal bundle. *u*. Fastigium. *v*. Inferior medullary velum. *w*. Nodule. *x*. Postnodular sulcus.

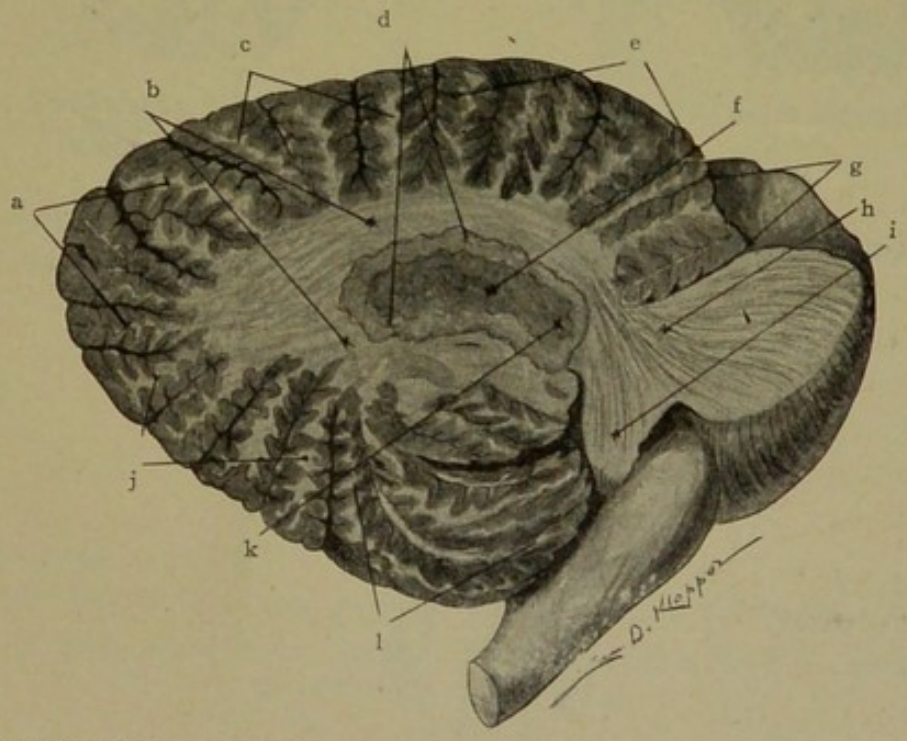


FIG. 38.—SAGITTAL SECTION OF CEREBELLUM, CUTTING NUCLEUS DENTATUS. (*Santee.*)
a. Sup. semilunar lobule. *b*. Corpus medullare. *c*. Post. part quadrangular lobule. *d*. Nucleus dentatus. *e*. Ant. part of quadrangular lobule. *f*. Interior of dentate nuc. *g*. Central sulci. *h*. Brachium pontis. *i*. Restiform body. *j*. Inf. semilunar and slender lobules. *k*. Hilus of nuc. dent. *l*. Biventral lobule.

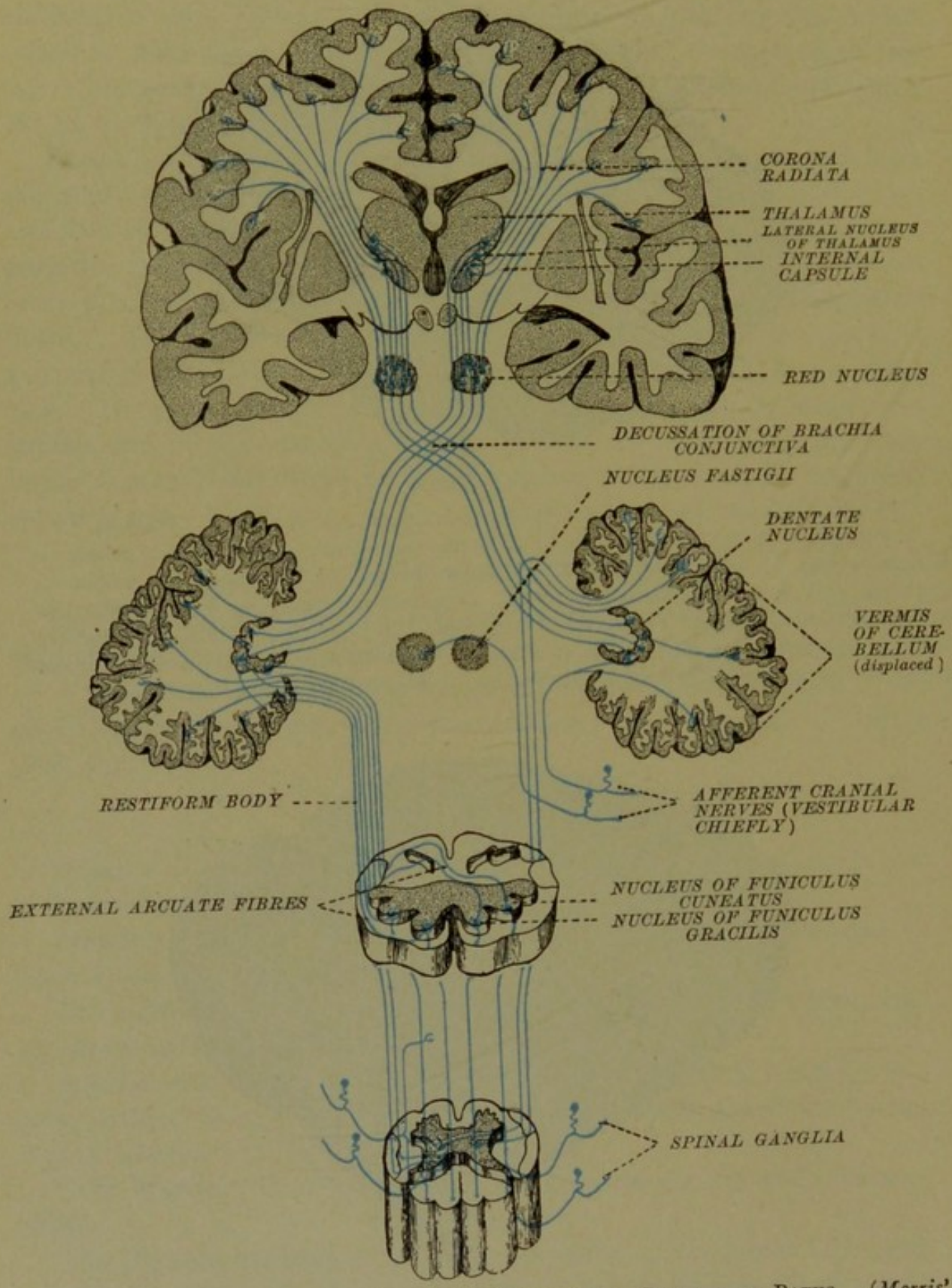


FIG. 39.—SCHEME OF PRINCIPAL ASCENDING CEREBELLAR CONDUCTION PATHS. (*Morris' Anatomy.*)

three pairs of peduncles uniting the cerebellum with the brain, mid-brain and spinal cord. The situation, course and termination of the cerebellar peduncles have been sufficiently discussed in the study of each portion of the central nervous system (see also illustrations, Figs. 39 and 40).

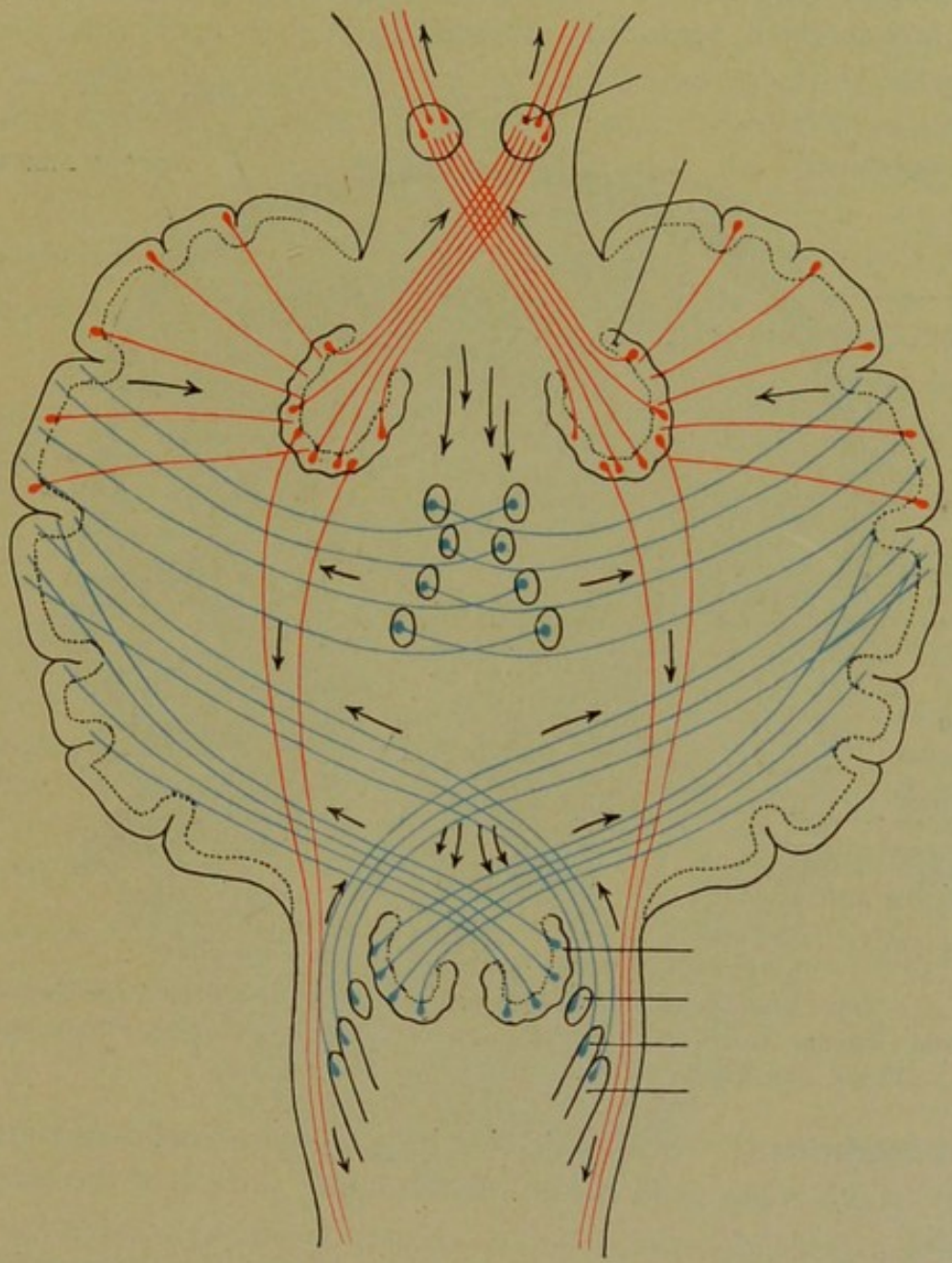


FIG. 40.—SCHEME OF CEREBELLAR CONNECTIONS. (*From Poirier and Charpy.*)
Efferent or centrifugal fibers in red. Afferent or centripetal fibers in blue. Small circles in the center are pontine nuclei.

MENINGES OF THE BRAIN

The brain is surrounded by three membranes, viz. **dura-mater, arachnoid** and **pia-mater.** (FIG. 41).

Dura.—It consists of two layers: an outer which serves as endosteum

of the cranium, and an inner layer which sends processes between subdivisions of the brain.

The **outer** layer adheres to the cranium, especially along the sutures and at the base.

The **inner** layer gives off prolongations, **three** in number, viz. falx cerebri, falx cerebelli, tentorium cerebelli.

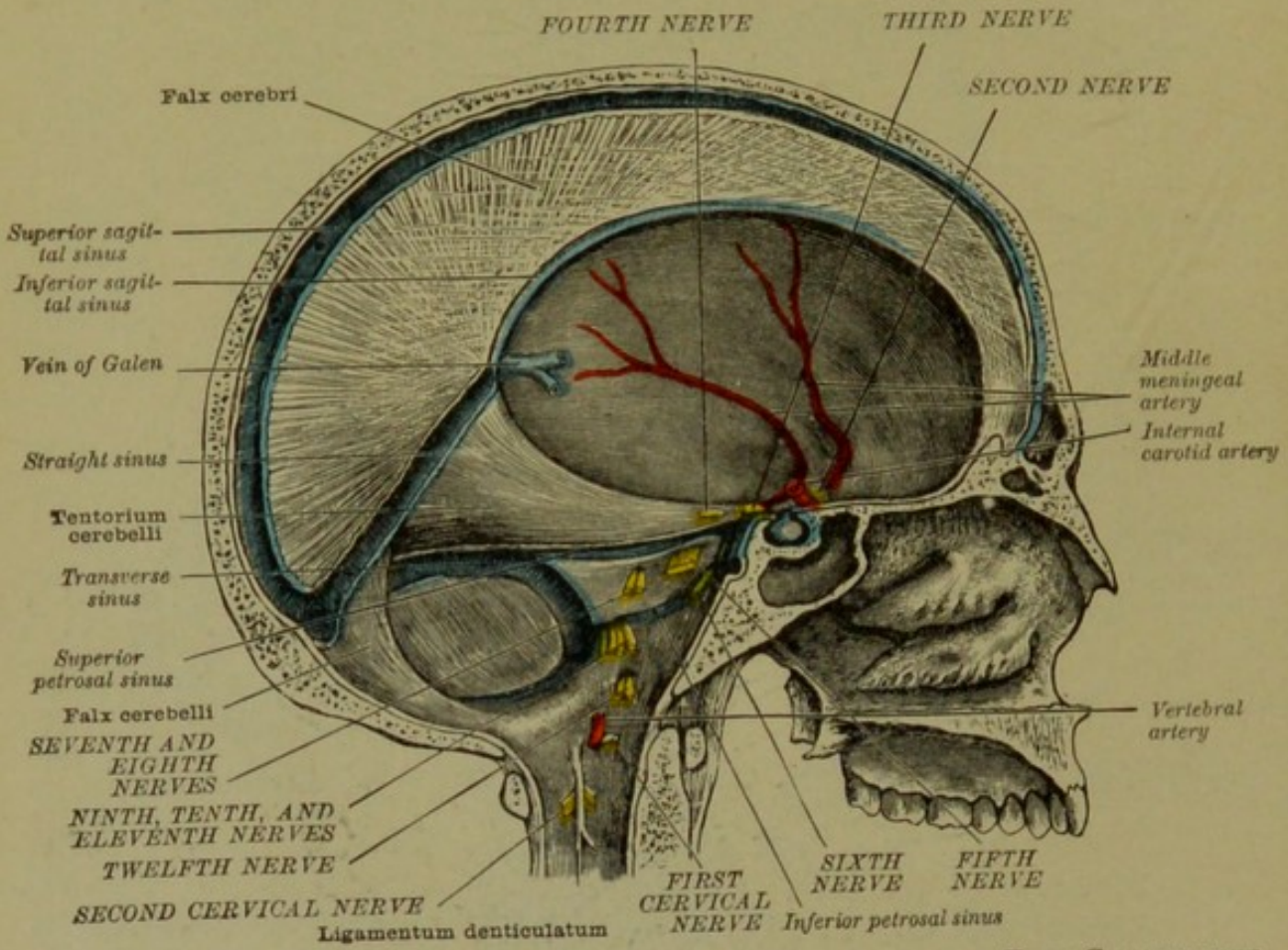


FIG. 41.—THE CRANIUM WITH ENCEPHALON REMOVED TO SHOW THE FALX CEREBRI, THE TENTORIUM CEREBELLI, AND THE PLACES WHERE THE CRANIAL NERVES PIERCE THE DURA MATER. (*Morris, after Sappey.*)

Falx cerebri is a vertical projection in the longitudinal fissure attached in front to the crista galli, behind to the upper surface of the tentorium cerebelli.

Falx cerebelli is a smaller process of the dura than the preceding one, which is inserted between the hemispheres of the cerebellum. It is attached behind to the internal occipital crest and above to the tentorium cerebelli.

Tentorium cerebelli is an arched tent-like process between the cerebrum and cerebellum.

The two layers of the dura become separated in certain places to form **venous sinuses.** They are fifteen in number—five paired and five single.

The paired ones are two lateral, two superior petrosals and two inferior petrosals, two cavernous and two occipitals. The single sinuses are the superior longitudinal, inferior longitudinal, the straight, the circular and the transverse.

The accompanying illustrations give a satisfactory idea of their respective seats (Figs. 41, 42 and 43).

Arachnoid.—It is situated between the dura and pia. It envelops the brain but does not penetrate the fissures. At the level of the fissures into

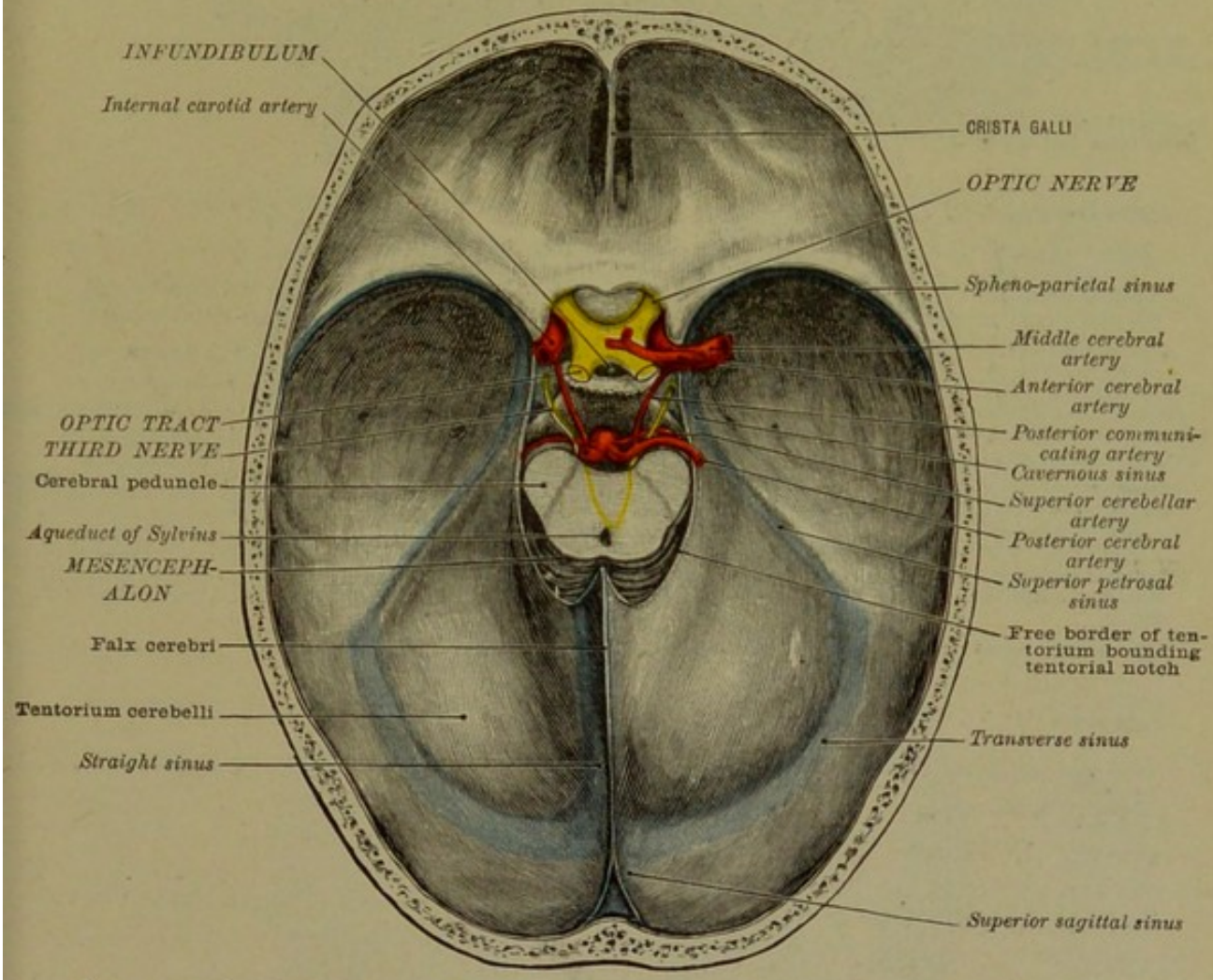


FIG. 42.—SHOWING THE UPPER SURFACES OF THE TENTORIUM CEREBELLI AND THE TENTORIAL NOTCH THROUGH WHICH THE MID-BRAIN AND POSTERIOR CEREBRAL ARTERIES ENTER THE MIDDLE FOSSA OF THE CRANIUM. (*Morris' Anatomy.*)

which the pia dips the two membranes are widely separated. This is the **sub-arachnoid** space containing cerebro-spinal fluid.

On the vertex of the brain, along the longitudinal fissure, there are small nodules, outgrowths of the arachnoid, called **Pacchionian** bodies.

Pia-mater.—It is an extremely thin and vascular membrane closely

applied to the cortex and continuing in the fissures. Together with the other two membranes it forms the sheaths of the cranial nerves.

Special processes are sent off in the spaces between certain portions of the brain. In the cavities of the third and fourth ventricles they are known as **Tela choroidea,** the margins of which are wrinkled, forming a vascular fringe known as **Choroid plexus.** The latter is also found in the lateral ventricles.

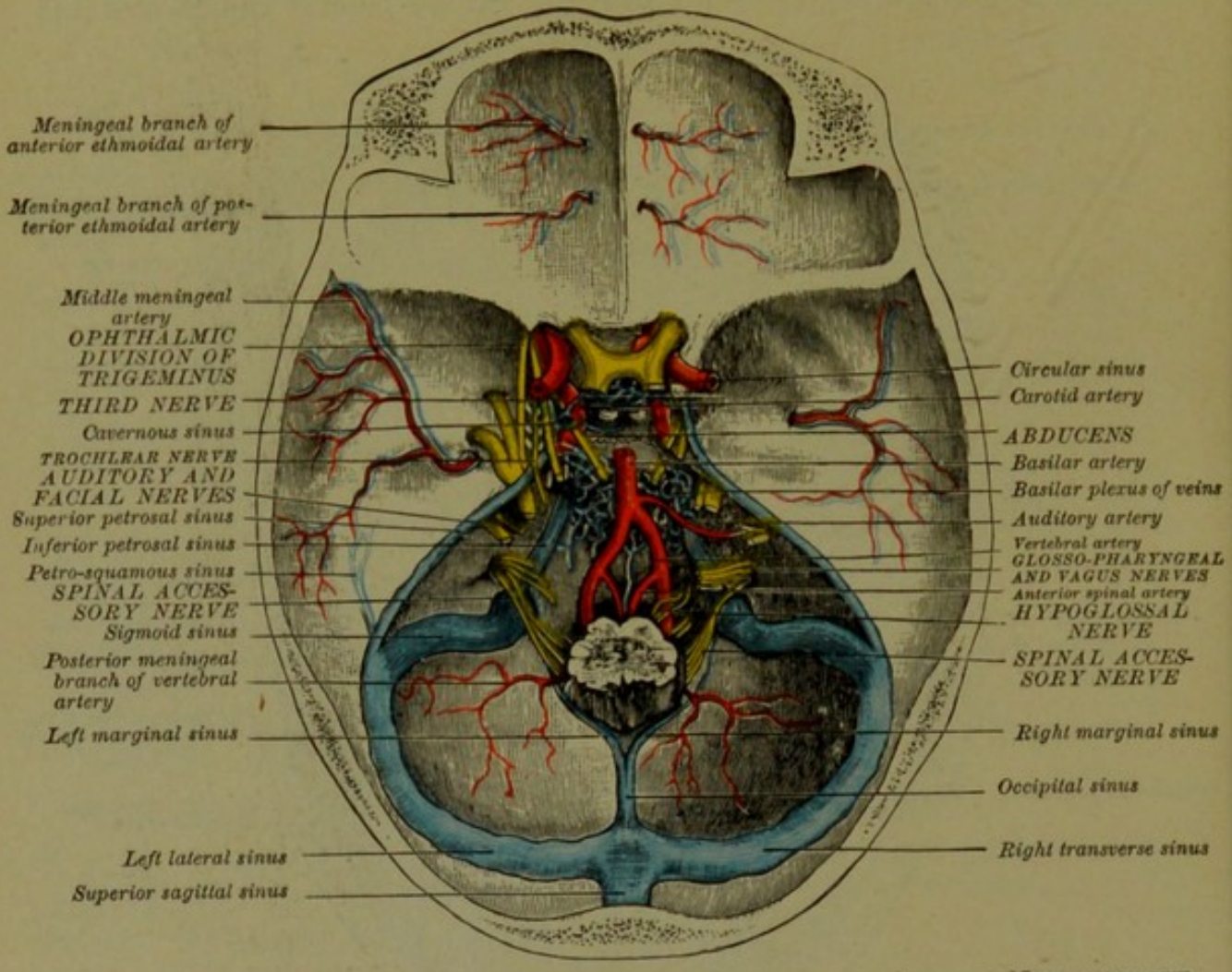


FIG. 43.—SHOWING BLOOD VESSELS OF CRANIAL DURA MATER AND CRANIAL NERVES IN THE BASE OF THE SKULL. (*Morris' Anatomy.*)

(On the left side the dura mater has been removed in the middle fossa.)

BLOOD SUPPLY OF THE BRAIN

The **arteries** of the brain are supplied by the two **internal carotids** and the **vertebrals.**

A. The internal carotid arteries divide into **anterior** and **middle cerebral** arteries and **posterior communicating.**

The anterior cerebral arteries supply the frontal and olfactory lobes, the optic nerves, corpus callosum and anterior perforated space.

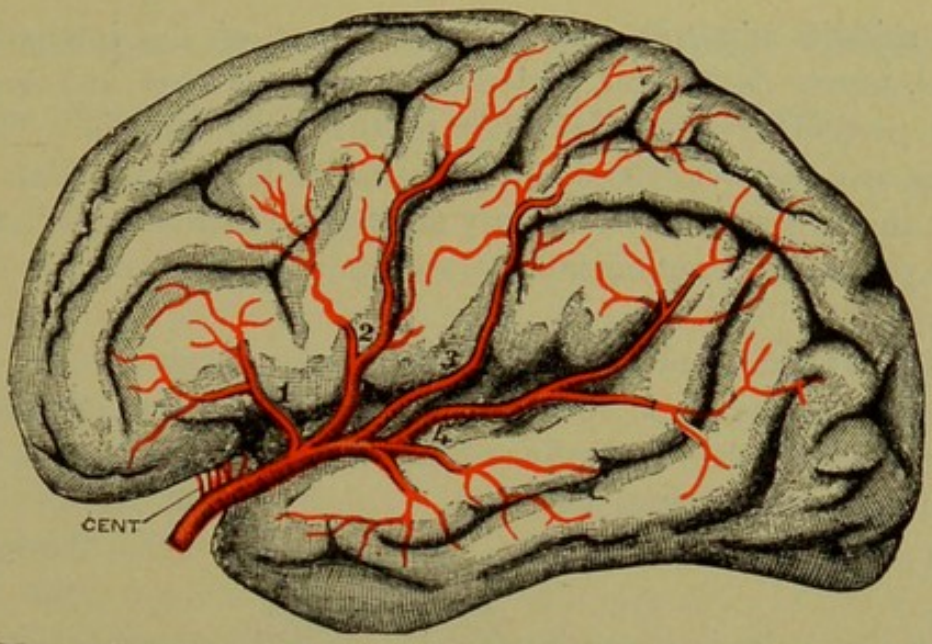


FIG. 44.—MIDDLE CEREBRAL ARTERY AND BRANCHES. (*Gordinier, after Quain and Charcot.*)

Cent. Antero-lateral group of ganglionic arteries. 1. Inferior external frontal artery. 2. Ascending frontal artery. 3. Ascending parietal artery. 4. Parieto-temporal artery.

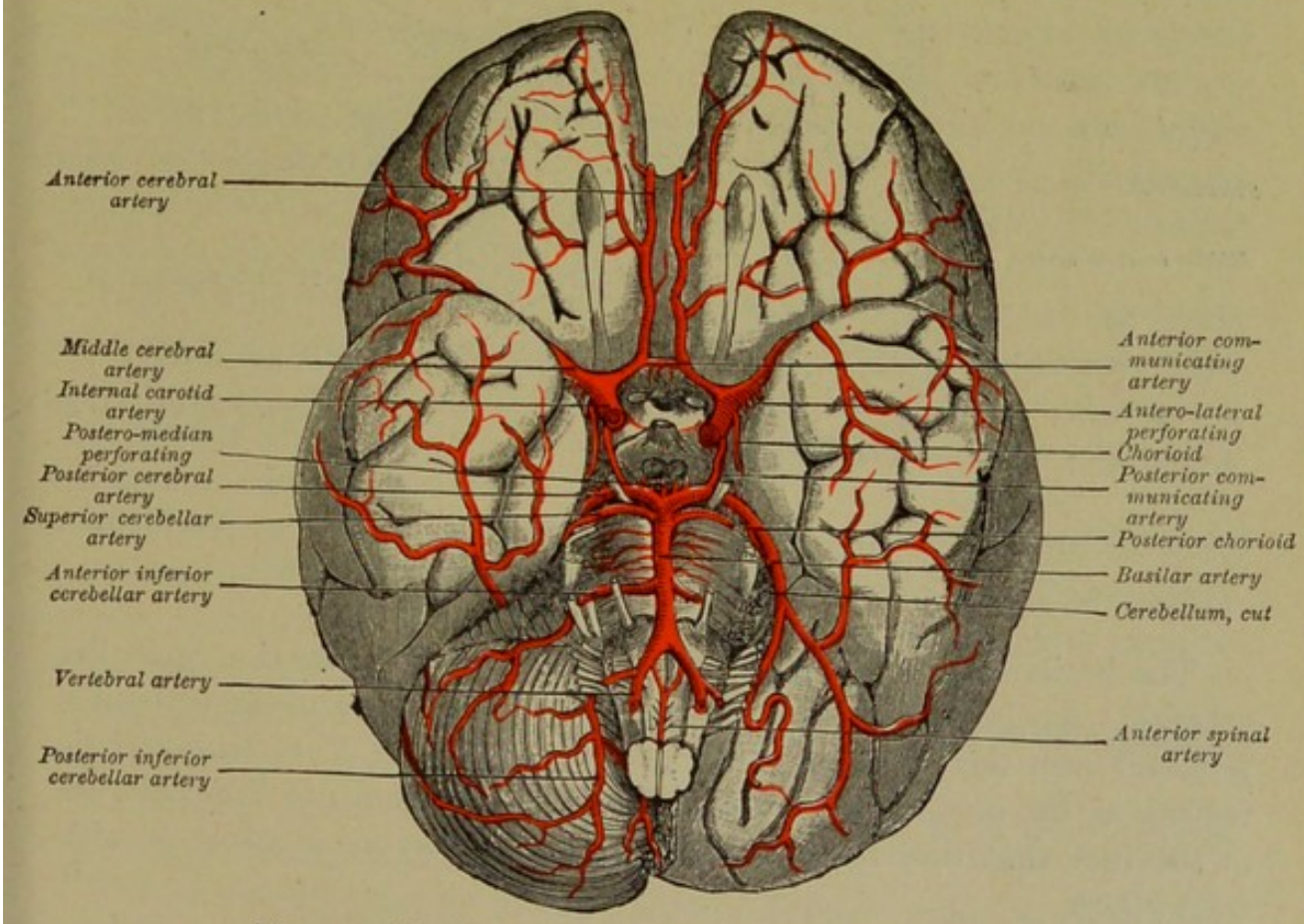


FIG. 45.—THE ARTERIES OF THE BRAIN. (*Morris' Anatomy.*)

(The cerebellum has been cut away on the left side to show the posterior part of the cerebrum. From a preparation in the Museum of St. Bartholomew's Hospital.)

The **middle cerebral arteries,** the largest of the internal carotid branches, supply the frontal, parietal and temporal lobes, and through the anterior perforated space branches to the basal ganglia. The latter branches are the "ganglionic." One of the lenticulo-caudate arteries which is the largest is called **"artery of cerebral hemorrhage"** (Charcot).

The **posterior communicating** arteries join the posterior cerebral arteries.

B. The **Vertebral arteries,** branches of the subclavian, meet at the lower border of the pons, unite and form one trunk, viz. **basilar** artery, which, running in the middle line of the pons, divides at its upper border into two **posterior cerebral arteries.**

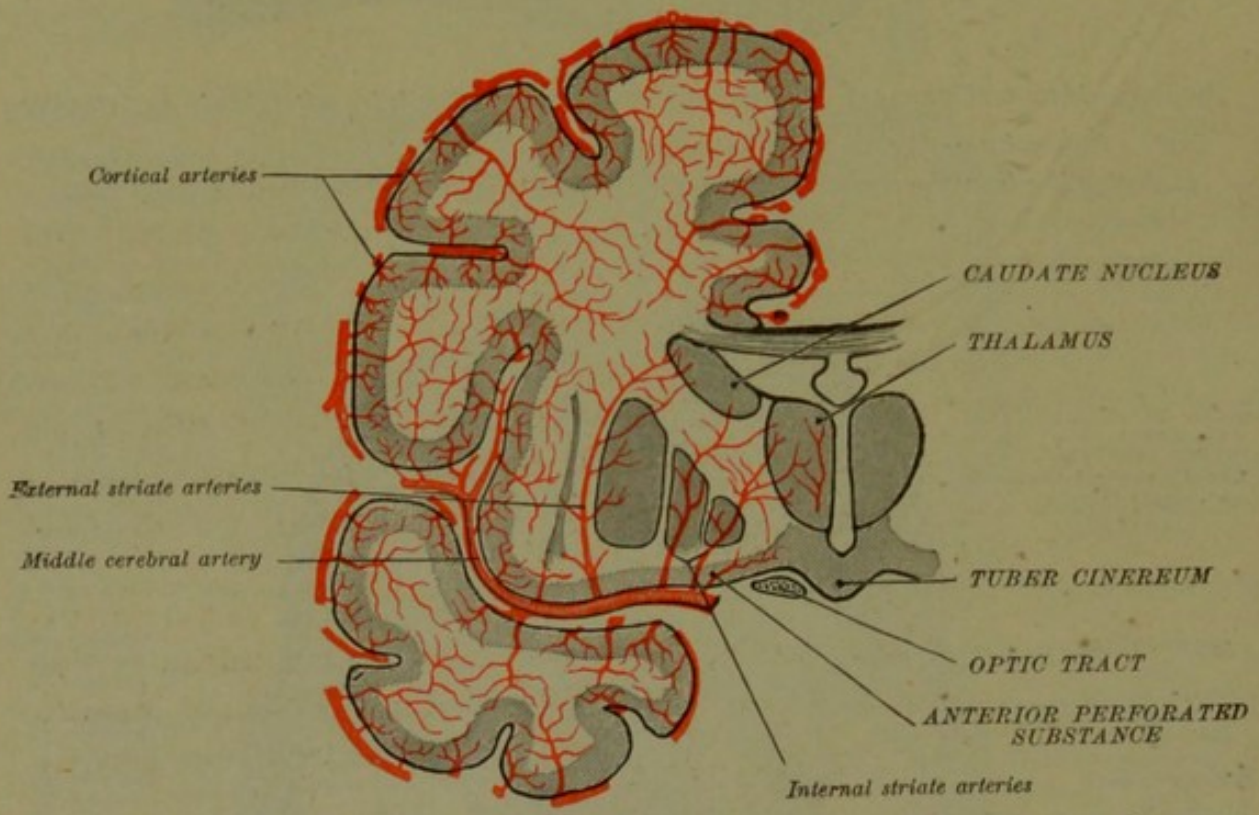


FIG. 46.—DIAGRAM SHOWING THE MANNER OF DISTRIBUTION OF THE CORTICAL AND CENTRAL BRANCHES OF THE CEREBRAL ARTERIES. (*Morris' Anatomy.*)

The basilar artery gives off anterior, inferior and superior cerebellar arteries. At the base of the brain the internal carotids and the vertebrals join and form the **Circle of Willis** (see illustration). The characteristic feature of the blood vessels distributed in the substance of the brain lies in the fact that they are **terminal,** that is, they do not anastomose with each other.

Cerebral veins do not accompany the arteries, but open in various **sinuses** (see chapter on Meninges). The latter are without valves (Figs. 44, 45 and 46).

HISTOLOGICAL ELEMENTS OF THE CENTRAL NERVOUS SYSTEM

Neurone Doctrine and Secondary Degeneration

The chief histological elements of the central nervous system are: nerve-cells, nerve-fibers, neuroglia.

Nerve-cell.—It consists of a protoplasmic body in the center of which is a nucleus with a nucleolus. It has no capsule, but the cells of the sympathetic ganglia and of the spinal ganglia have a capsule. The protoplasma contains granular masses, stainable by Nissl's stain, and they are called Nissl's bodies. Ramon y Cajal has recently (1903) devised a stain with which it can be demonstrated that the cell is composed of delicate fibrils (**neurofibrils**). In the majority of the cells, particularly in those of large size, exists a granular mass of yellowish pigment.

Each cell is provided with processes, one of which is the most important and called **axone.**

Varieties of Cells.—They may be **bipolar** and **multipolar.**

The latter are found throughout the entire nervous system, but predominate in the anterior horns of the spinal cord, the cortex, medulla, basal ganglia. Bipolar cells are found in the posterior horns of the spinal cord, spinal ganglia, and in the molecular layer of the cerebellar cortex.

In the **Cerebellum** there are two special forms of cells, viz. **Purkinje's cells** and **basket cells.** The first are **flask-shaped** with a very slender and long axone having a vertical course. The second have also a long axone which has a horizontal course.

Cell Processes.—Some branches of a cell branch out, tree-like, divide and subdivide, but **do not anastomose.** They are the **dendrites.** One process, called **axone,** is the most important. It gives off collaterals. Soon after leaving the cell it receives a coating, called **myelin** and becomes a medullated nerve-fiber.

Nerve-fiber.—It is the prolongation of an axone. It consists of an axone, surrounded by a myelin sheath and of a delicate membrane, neurilemma, surrounding the latter. The myelin and neurilemma present constrictions (nodes of Ranvier).

The sympathetic system contains nerve-fibers, called **non-medullated,** viz. deprived of a myelin sheath.

Connective tissue joins nerve-fibers into fasciculi or bundles, which when united form **peripheral nerves.**

The nerves terminating in the muscles or skin present at their ends special arrangements. The **motor nerves** end in motorial end plates, which are special expansions of granular protoplasm. The **sensory nerves** end in corpuscles, composed chiefly of connective tissue.

Neuroglia.—It is the framework of the central nervous system. Before Virchow (1846) it was considered as a connective tissue. This tissue is composed of spheric cells (spider cells, Deiters' cells) containing a large nucleus and a great many processes. The function of neuroglia tissue is to replace diseased nervous elements. As soon as a lesion of the latter occurs, the neuroglia tissue begins to proliferate. Its function therefore resembles that of connective tissue.

Neurone.—Under this name, given first by Forel and then by Waldeyer, is understood an anatomical **unit** of the nervous system. It consists of the **cell-body** with all its outgrowths, viz. **dendrites** and **axone** with all the ramifications, collaterals, and terminal arborizations. The nervous system is made up of a multitude of such units which, according to the generally accepted view, are trophically and genetically independent and are only contiguous, but not continuous. In spite of their independence they are functionally correlated. This can be explained by a supposed amœboid function of the dendrites and by appearance and disappearance of buds or gemmules on them caused by the contractility of the protoplasm. Such a mechanism will naturally bring about a contact influence of one neurone upon another. The problem, however, is far from being settled.

There are three types of neurones:

(1) Neurone with a long axone which may give off collaterals. It is found in brain and spinal cord.

(2) Neurone with a short axone which breaks up promptly into branches. It is found in brain and cerebellum.

(3) Neurone with several axones. It is found in the first layer of cerebral cortex.

The **function** of neurones is (1) **efferent** (motor), (2) **afferent** (sensory) and (3) **associative.**

(1) **Efferent (Motor) Neurones.**—They present two groups: **upper** and **lower.**

Upper Motor Neurones.—This is the pyramidal system. The fibers originate in the large pyramidal cells of the motor area (see Localizations, page 75), descend through the centrum ovale, reach the internal capsule, pass downward into the crura, medulla oblongata, where they mostly decussate, descend in the spinal cord in a small number as non-decussated or direct fibers in the antero-internal region of the same side, and in a larger number as decussated or crossed on the opposite side as low as the fourth sacral segment. During their passage through the mid-brain and hind-brain a large number of fibers are given off to the motor nuclei of the cranial nerves, viz. third, sixth, fifth, seventh, ninth and twelfth.

In the spinal cord the crossed pyramidal fibers give off collaterals to the gray matters. The direct pyramidal fibers decussate through the anterior white commissure and terminate in the cornua of the opposite side. The upper motor neurones connect, therefore, the motor cortex with the bulbo-spinal centers.

Lower Motor Neurones.—They are constituted by the motor nuclei of the medulla and spinal cord (anterior cornua), their roots and peripheral nerves. In the medulla the nuclei give origin to the motor fibers of cranial nerves. In the spinal cord the cells of the anterior cornua give origin to the motor nerves of the muscles of the extremities and the body.

(2) **Afferent (Sensory) Neurones.**—They commence in the sensory end-organs, ascend in the peripheral nerves to the spinal ganglia, posterior roots and their extension into the spinal cord as posterior columns. The latter continue their course through the mesial fillet, where they decussate and reach upward the optic thalamus. Another set of fibers originate in the cells of the posterior cornua, decussate in the posterior commissure, reach the antero-lateral tract and ascend through the medulla and pons to end in the optic thalamus (spino-thalamic system). The sensory fibers of the cranial nerves also enter the sensory pathway. Finally, fibers ascend from the optic thalamus to the cerebral cortex of the same and opposite side. (For details of the pathways see their respective chapters in Anatomy.)

(3) **Association Neurones.**—They connect the other neurones together. They are neurones with short axones.

The integrity of a neurone depends upon the integrity of its main element, viz. cell-body. Waller has shown that if a nerve was severed from its mother cell, it would degenerate in the direction in which the impulse is conducted. For example, a section of a posterior (sensory) nerve-root ventrally to its spinal ganglion in which it originates, will be followed by a degeneration of the ascending posterior columns of the cord. If an anterior (motor) root will be severed from its origin in the cells of the anterior cornua, the degeneration of the peripheral nerve will be descending and reach its termination in the muscle. This process is called **"secondary degeneration."**

MALFORMATIONS OF THE CENTRAL NERVOUS SYSTEM

The brain and spinal cord may be the seat of malformations. Pathological and traumatic influences may disturb the growth of the fœtus in its intra-uterine life. The ealier the disturbances occur, the more pronounced are the malformations.

Brain.—**Hyperplasia** or **Hypoplasia** of cerebral tissue are the chief abnormalities.

A. **Hypoplasia.**—Absence of the brain is called

1. **Anencephalia.** (Fig 47.) Instead of brain tissue there is a cavity filled with a dark vascular membrane. This condition is inconsistent

FIG. 47.—ANENCEPHALIA.

with life. Partial absence (*agenesis*) of brain tissue is compatible with life. When parts of the cerebrum are not properly developed, the local defects are covered with the pia-arachnoid; the subarachnoid space is filled with fluid. To this variety belongs

2. **Porencephaly.** (Fig. 48.) It is characterized by a fissure-like or funnel-shaped **depression** showing a want of normal tissue. It is confined

usually to the cortex (central and parietal lobes), and the Island of Reil, but it may also be intracerebral. The depression is bridged over by the arachnoid, but the pia follows the gyri. It may reach the ventricles and even communicate with then. Porencephaly is frequently associated with other developmental defects, as multiplication of fissures and lobes. Microscopically there is atrophy of cells and white matter in the defective area. Clinically hemiplegia, contractures, imbecility, idiocy, mutism and deafness are observed (see page 112). Depressions in the cerebrum are sometimes observed when partial destruction of cortical and subcortical tissue occurs from some acquired morbid process in extra- or intrauterine life. This is **pseudo-porencephaly.**

3. **Microcephalia** is characterized by a diminution in size of one or

FIG. 48.—PORENCEPHALY. (Feeble-mindedness. Epilepsy.) (*Original.*)

both hemispheres. Normally the weight of a male brain is about 1375 grm. and of a female brain 1245 grm. In microcephalia the weight may be as low as 900 grm. and even 200 grm. Either the entire brain or hemisphere is atrophied or there is a defective development of a certain portion of them. It is usually due to a defective development. It may also be due to a premature closure of the cranial sutures. There is often a deficiency of the sulci on the cortex; the lobes and convolutions are imperfectly outlined or abnormally small. The spinal cord is also very small; the postero-lateral columns are mostly affected. Clinically the condition is characterized by a small size of the head and defective mental development (Fig. 49).

4. **Microgyria** consists of multiplication of fissures and consequently

of convolutions. The latter are very small. The condition is not infrequently associated with microcephaly.

5. **Absence of Lobes, imperfect development or absence of some of the basal ganglia and of Corpus Callosum** also occur in conjunction with microcephaly.

6. The **Cerebellum** may present the same developmental defects as the cerebrum. Atrophy of the cerebellum was the subject of an exhaustive

FIG. 49—MICROCEPHALY. (Idiot.)

study by Preisig (*J. f. Psych. u. Neurol.*, 1911). He reaches the following conclusions: (1) There is a form of meningo-encephalitis of fœtal period of life with a special localization in the cerebellum which results in atrophy of the latter. (2) Idiocy which exists in such cases has an anatomical basis in conjunction with other alterations of the cerebral cortex. (3) All the systems of fibers ending in the cerebellum are in a state of atrophy.

B. **Hyperplasia** is characterized by an excessive development of the

constituent elements of the brain. **Macrocephaly** is due to hypertrophy of the brain. There is anatomically an increase of gray substance and of connective tissue in the white matter. The skull is proportionally increased and the head appears large.

The etiology of macrocephaly is alcoholism and syphilis of the parents. Imbecility, idiocy and convulsions are observed in macrocephalic individuals.

Other Malformations.—To this category belong cases associated with defects in the development of the cranium. **Encephalocele** is characterized by a protrusion of brain tissue between the cranial bones, which for

FIG. 50.—SPINA BIFIDA.

some reason failed to coalesce. If the meninges alone form the mass, it is called **Meningocele.** There is usually a combination of both anomalies.

Cyclopia is characterized by a mass in the forehead consisting of fusion of both eyes. When in addition to the latter there is also absence of the nose, the anomaly is called **Arhinencephalia.**

Spinal Cord.—The most common variety of malformations of the spinal cord is **Spina bifida.** It is due to a defect in the development of the vertebral arches, which failed to coalesce and therefore permitted the cord with or without the meninges to protrude dorsally.

Spina bifida may present itself as a **Meningocele,** viz. a saccular tumor containing the meninges; as a **Meningomyelocele,** consisting of meninges and cord, also the nerve-roots; as a **myelocele,** consisting only of cord tissue; the latter is rare and occurs when not only the vertebral arches, but also the medullary folds failed to coalesce.

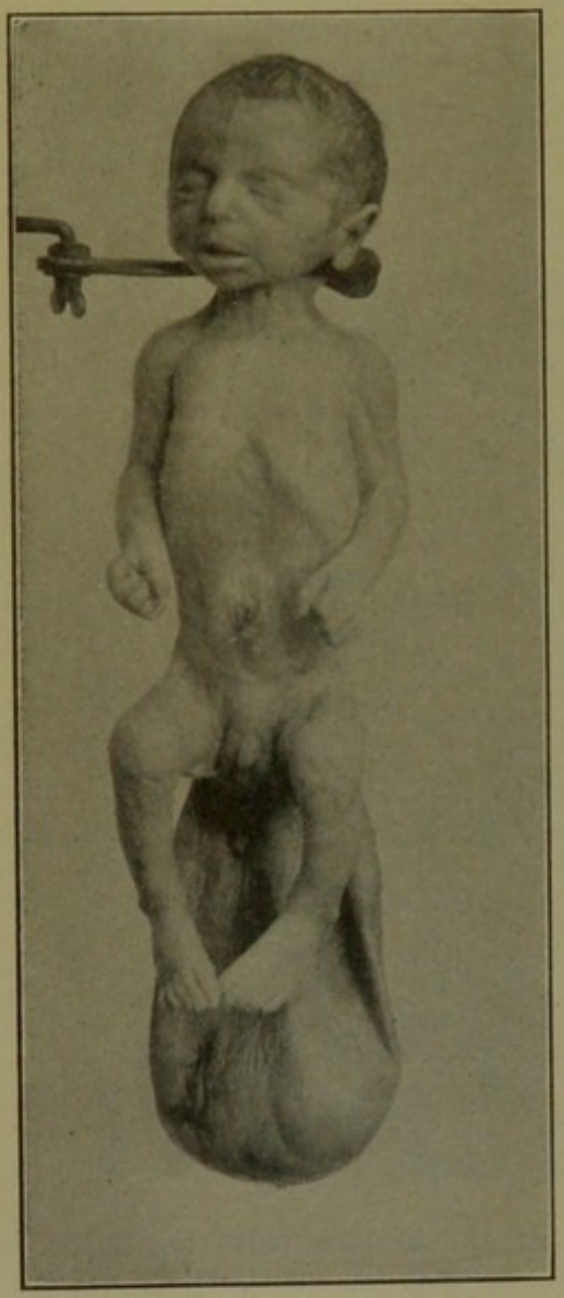


FIG. 51.—MENINGOMYCLOCELE WITH DERMOID CYST. (Rigidity of all extremities.)

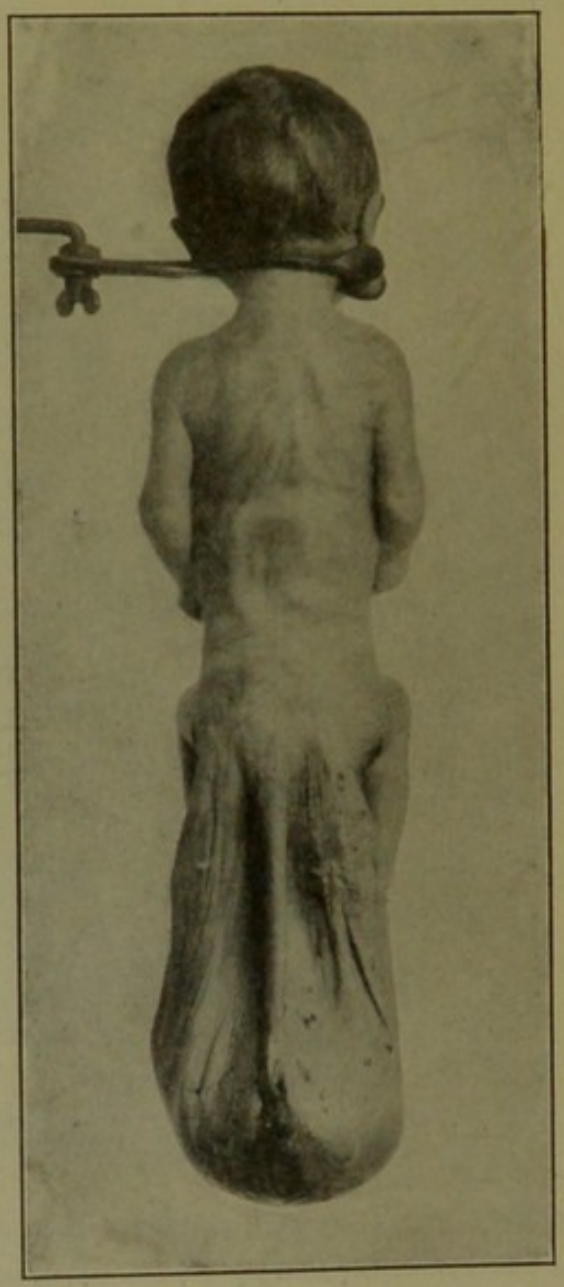


FIG. 52.—MENINGOMYCLOCELE WITH DERMOID CYST. (Rigidity of all extremities.)

The most frequent variety is the **Meningomyelocele.** Clinically there is usually paralysis of the legs, of bladder and rectum, clubfoot, also anæsthesia. The lumbo-sacral region is its place of predilection (Fig. 50, 51, 52).

Children born with a spina bifida usually do not live long; their average life is from three months to a year. In some cases where the tumor is very small, adult life may be reached. The covering of the tumor may be only a thin membrane or else the skin. In the latter case the prognosis to life is better, as the skin is a good protection.

The tumor may be a round mass covered by and continuous with the surrounding skin or may be pedunculated. In the latter case there is usually a meningocele. Operative procedures are practically the only means that may be employed for remedying the condition. In the most favorable cases paralysis of the extremities will remain. In meningo-

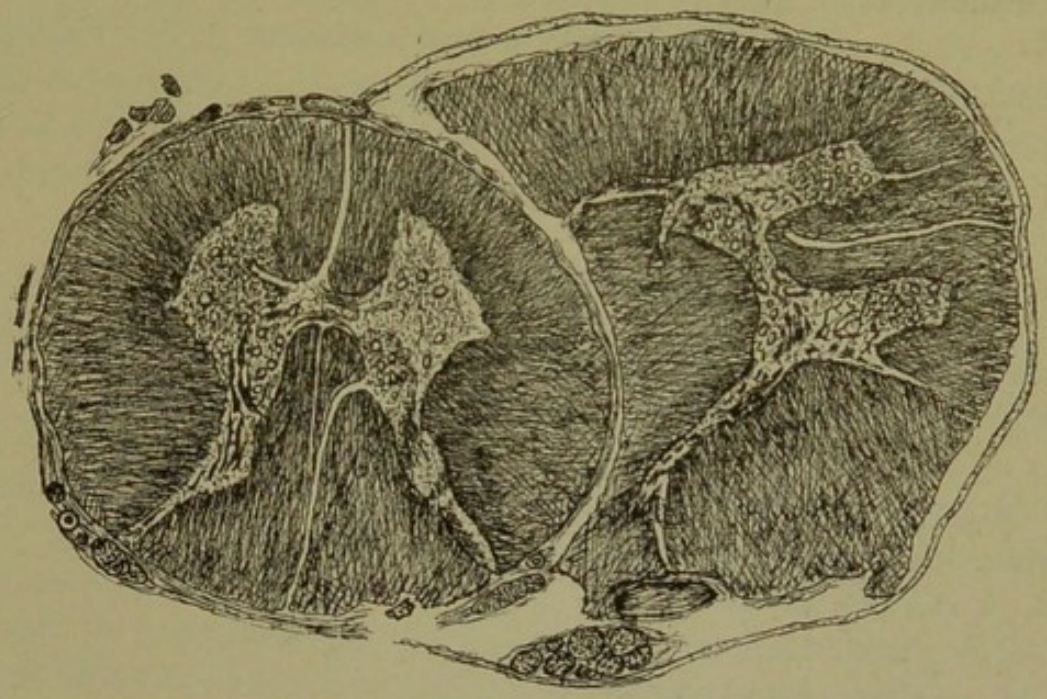


FIG. 53.—DIPLOMYELIA. (*Original.*)

myelocele the contents of the sac cannot be removed. Only in pure meningocele an operation can give satisfactory results. Tournier and Ducuing basing themselves on a large experience formulate the following indications for operative treatment. Excision of the tumor is permissible when it is complicated with nervous symptoms of no longer than a year's duration at most, or when the tumor is free from complications, but is of annoying size. When the nervous symptoms are of long duration, palliative treatment is the rule, some orthopedic operation on other parts of the body than on the spina bifida offering the only prospect of benefit. Under all other conditions operative treatment is contraindicated (*Arch. génér. de Chir.*, 1912).

Among other malformations can be mentioned: **Craniorrhachischisis**

and **Rhachischisis.** The first consists of lack of closure of the cranium and vertebræ. The entire medullary groove is patent and filled with a vascular tissue, rudiments of cord and brain.

In the second variety only the spinal portion of the groove is patent.

As a great rarity may be mentioned **diplomyelia** (double cord) (Fig. 53).

Heterotopia of gray matter is also a very rare condition. It consists of the presence of gray nodules in the white matter of the cord.

CHAPTER II

METHOD OF EXAMINATION FOR DIAGNOSIS OF NERVOUS DISEASES

THE diagnosis of cases of nervous diseases is based chiefly upon the knowledge of the physiological and pathological states of the following elements: I, **motor phenomena**; II, **sensory phenomena**; III, **special senses**; IV, **cranial nerves**; V, **speech and writing**; VI, **reflexes**; VII, **sphincters**; VIII, **vasomotor and trophic disturbances.**

In order to arrive at a correct diagnosis, every case of functional or organic nervous diseases that comes under observation should be investigated from the above standpoints.

I. MOTOR PHENOMENA

An examination of the motor phenomena embraces the following points: station, gait, attitude, muscular power, various muscular movements, state of nutrition of muscles.

Station.—The patient is observed while standing with open and closed eyes. Normally when the eyes are closed and the patient stands with his feet close together, there is a very slight oscillation of the body. When the latter is somewhat more than slight or very pronounced, so that the patient shows a tendency of falling, the station is pathological. In neurasthenia, where the muscular fatigue is pronounced, an instability may be present. The degree of the latter depends upon the intensity of fatigue. It never, however, reaches the instability of organic nervous diseases. In hysteria the restlessness and inability of controlling the latter may also render the station with closed eyes abnormal.

In tabes the patient cannot hold himself straight not only with closed but also with open eyes. In the latter case he stands with the feet wide apart and the least attempt to bring them together makes him lose the equilibrium. When the eyes are closed the instability is great (**Romberg's sign**).

The same condition may be encountered in other organic nervous diseases. Whether it is due to organic or functional nervous diseases an inability or difficulty of standing erect with open or closed eyes is a morbid phenomenon.

Gait.—Disturbance of gait is very frequently observed in nervous diseases. The patient is told to walk with open and closed eyes. Normally the body follows a straight line. In certain nervous affections the coördination of movements is disturbed. This is called **ataxia.** The latter may be very slight, moderate or much pronounced. It is particularly marked when the eyes are closed or when the patient is told to turn. In **ataxic gait** the patient throws his leg forward in an exaggerated manner; the moment he is ready to touch the ground, the foot makes a backward movement and strikes the ground with the heel. Such a gait is seen in **tabes.** In some cases the gait may simulate a zigzag movement. It is observed particularly in cerebellar diseases (**titubation**).

Ataxia may also be revealed by asking the patient to touch with the heel of one limb the knee of the other or place one foot on the other. He will then form circles in the air with the foot before it reaches the desired spot.

Besides an ataxic gait, there is also a **high steppage** gait, which is due to foot-drop. The thighs are flexed on the abdomen more abruptly and higher than normally; the foot is thrown forward and then it falls on the ground first by the toes and last by the heel, so that two successive knocks on the ground are heard.

Wh ether the incoördination is mild or great, it is a morbid phenomenon and may be encountered in organic and in functional nervous diseases. In hysteria there may be an inability to stand or walk while all the other functions of the limbs are intact when the patient is in bed. The condition is known under the name of **Astasia-abasia.** In Paralysis Agitans the patient, once started voluntarily or slightly pushed, may keep on walking or running forward, backward or laterally for a long while until stopped. These movements are called, respectively, **propulsion, retropulsion** or **lateropulsion.**

Ataxia may be also observed in the upper extremities. The patient is told to close his eyes and bring the tips of the fingers of both hands together, or to touch the ear or the end of the nose with one finger. This test will reveal whether the movements of the upper limbs are coördinate or not.

In connection with the gait the **attitude** of the patient must be noticed, viz. whether he holds himself in an erect position or else in inclined forward or backward, also whether the movements of the spinal vertebræ are free or not. Special attitudes are acquired in some functional and organic nervous diseases.

Muscular Power.—It may be impaired from a simple weakness to a complete loss. When a muscle is suspected to be diseased, its power can

be tested by the resistance method. The patient is told to perform the function of this muscle and the observer endeavors to resist the given movement. If, for example, the forearm is voluntarily flexed (biceps), an effort is made to extent it. When the resistance of the biceps is easily overcome, we say that there is diminution of power of that muscle. If the desired function of a certain muscle cannot be performed at all, it may be due either to **paralysis** or **atrophy** of that muscle.

When **paralysis** is present, it may be **complete** or **incomplete** (paresis). It may be **spastic** when associated with rigidity of the muscles (**hypertonia**) and **flaccid** when the muscles are flaccid (**hypotonia**). The spastic form can be revealed by the presence of enormous resistance of the muscles to passive movements. The flaccid form is detected from the total absence of resistance. In the latter case if the hypotonia is pronounced, the limbs may be made to assume unusually abnormal positions, such as raising the foot to the head.

Paralysis may affect one limb (**monoplegia**), two symmetrical limbs (**paraplegia**) or one side of the body (**hemiplegia**). When the four extremities are affected with a spastic paralysis, we speak of **diplegia.** Paralysis or paresis may affect the musculature of an entire limb or only a certain group of muscles, as, for example, in cases of wrist- or foot-drop, in which the extensor groups of muscles alone are affected.

Muscular Movements.—The manner of using the lower extremities, of grasping or handling objects or else doing certain acts with the upper extremities, the range, rhythm, regularity or irregularity, constancy or inconstancy of the movements, the effect of emotion on movements, must all be closely observed in studying the motor apparatus. Ataxia, paralysis, loss of muscular resistance were mentioned above. **Tremor** is observed in functional and organic nervous diseases. It may be fine, coarse, passive (when at rest) and intentional (upon voluntary act). It may be also temporary or permanent. **Choreic** movements consist of irregular, involuntary and continuous muscular contractions.

Athetosis consists of slow and arhythmical movements, affecting chiefly the fingers.

Tic is a quick involuntary contraction of a certain muscle or group of muscles continuously repeating itself in a regular manner and imitating ordinary mimicry.

Myoclonia is characterized by involuntary clonic contractions of a group of muscles occurring in paroxysms. **Tetany** consists of a sudden and special contraction of the muscles of the fingers; they assume a writing position.

Muscles may also be affected by **convulsive movements, spasms or**

cramps. All these muscular phenomena occur either independently of or in association with various organic or functional nervous diseases.

State of Nutrition of the Muscles.—The nutrition of the muscles depends upon the integrity of their trophic centers. When a muscle is diminished in volume and cannot therefore perform its function, we speak of its **Atrophy.** When the atrophy is symmetrical and slight, it may escape our notice. When it is unilateral, it will be revealed by comparison with the corresponding muscles of the opposite side.

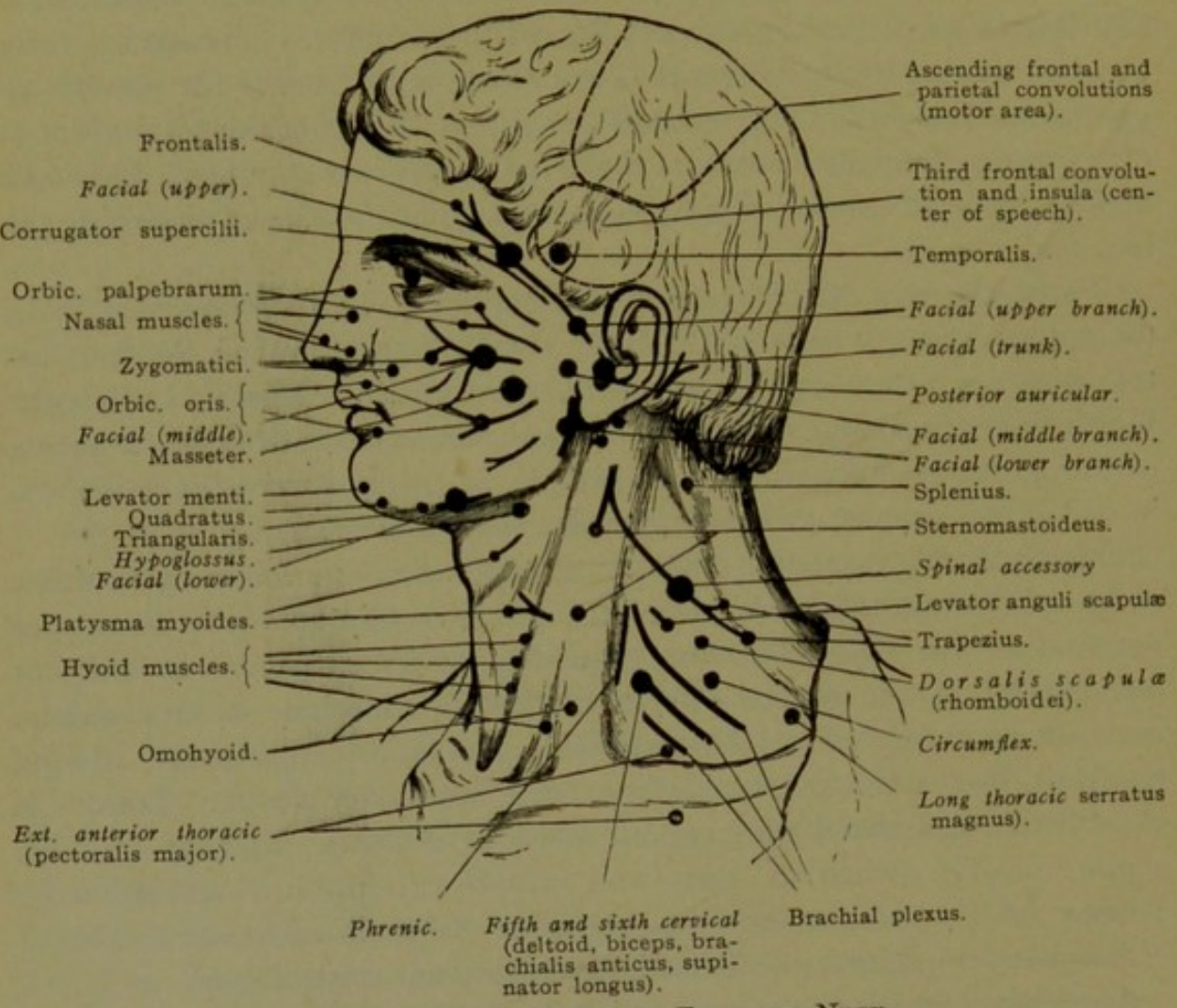


FIG. 54.—MOTOR POINTS OF FACE AND NECK.

It is not sufficient to determine the size of muscles. It is also necessary to study the state of their functions: a diminution of muscular force will be a valuable information in case the reduction of the volume of the muscle is not perceptible. If the size of a muscle is increased, we speak of **Hypertrophy.** The size of a muscle or of a group of muscles may be only apparently increased when the atrophy is masked by an increase of superimposed adipose tissue. Such a condition is known under the name of **Pseudo-hypertrophy.** Finally in cases where inspection or palpation

make the diagnosis of atrophy doubtful, an electrical examination must be resorted to.

Electrical Contractility.—Faradism and galvanism are employed for the purpose of ascertaining the state of nutrition of muscular tissue and of the nerves distributed in them. Muscular contractions may be ob-

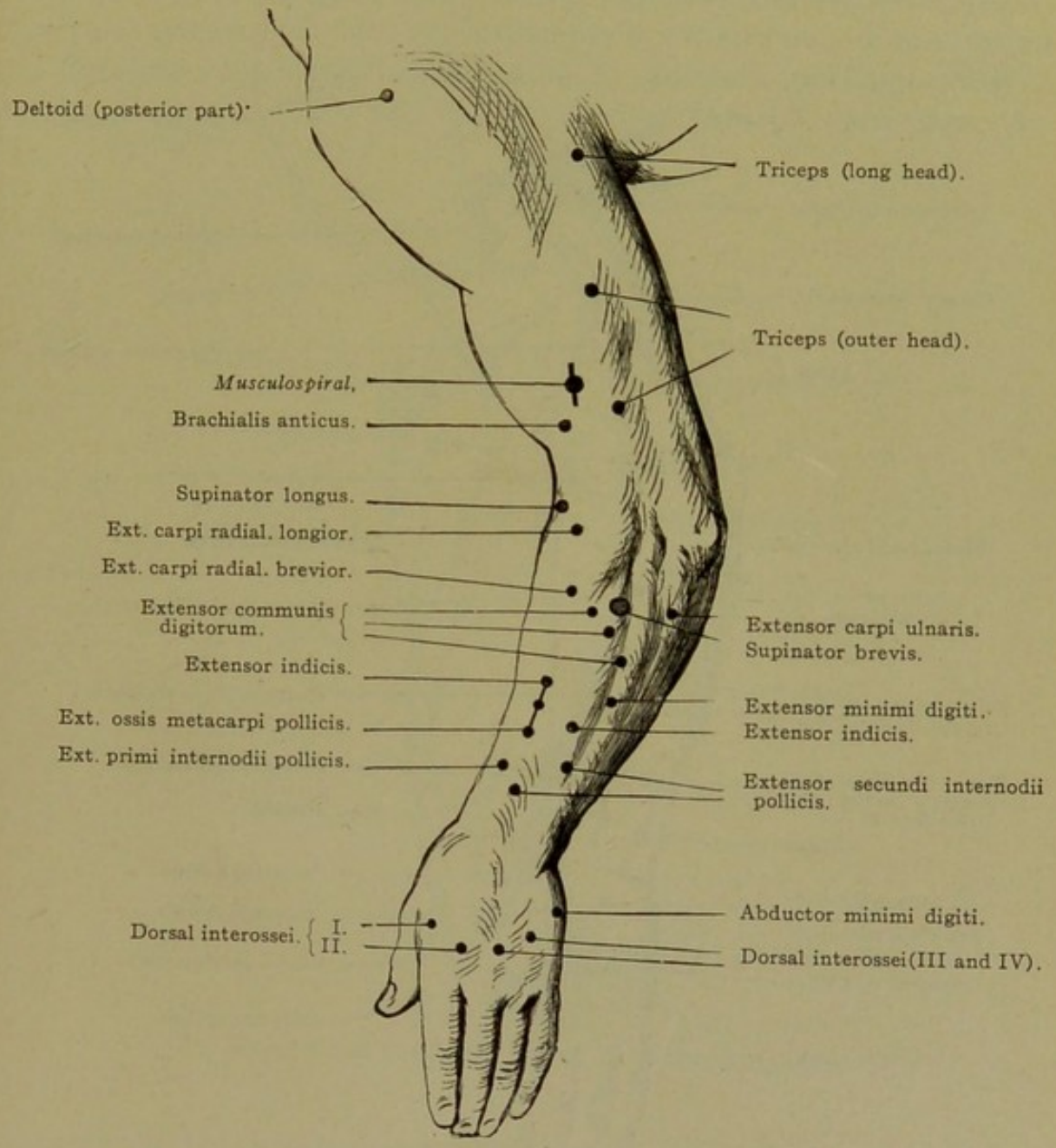


FIG. 55.—MOTOR POINTS ON UPPER LIMB, EXTENSOR SURFACE.

tained from direct applications of the electrical current to the tested muscle or from its application to the nerve trunks.

The examination for electrical reactions must be conducted in the following manner.

Faradism.—The large or indifferent electrode attached to the positive

pole (anode) is applied to the neck or sternum, the small or testing electrode attached to the negative pole (cathode) is held in the operator's hand. Both electrodes must be well wetted in hot water.

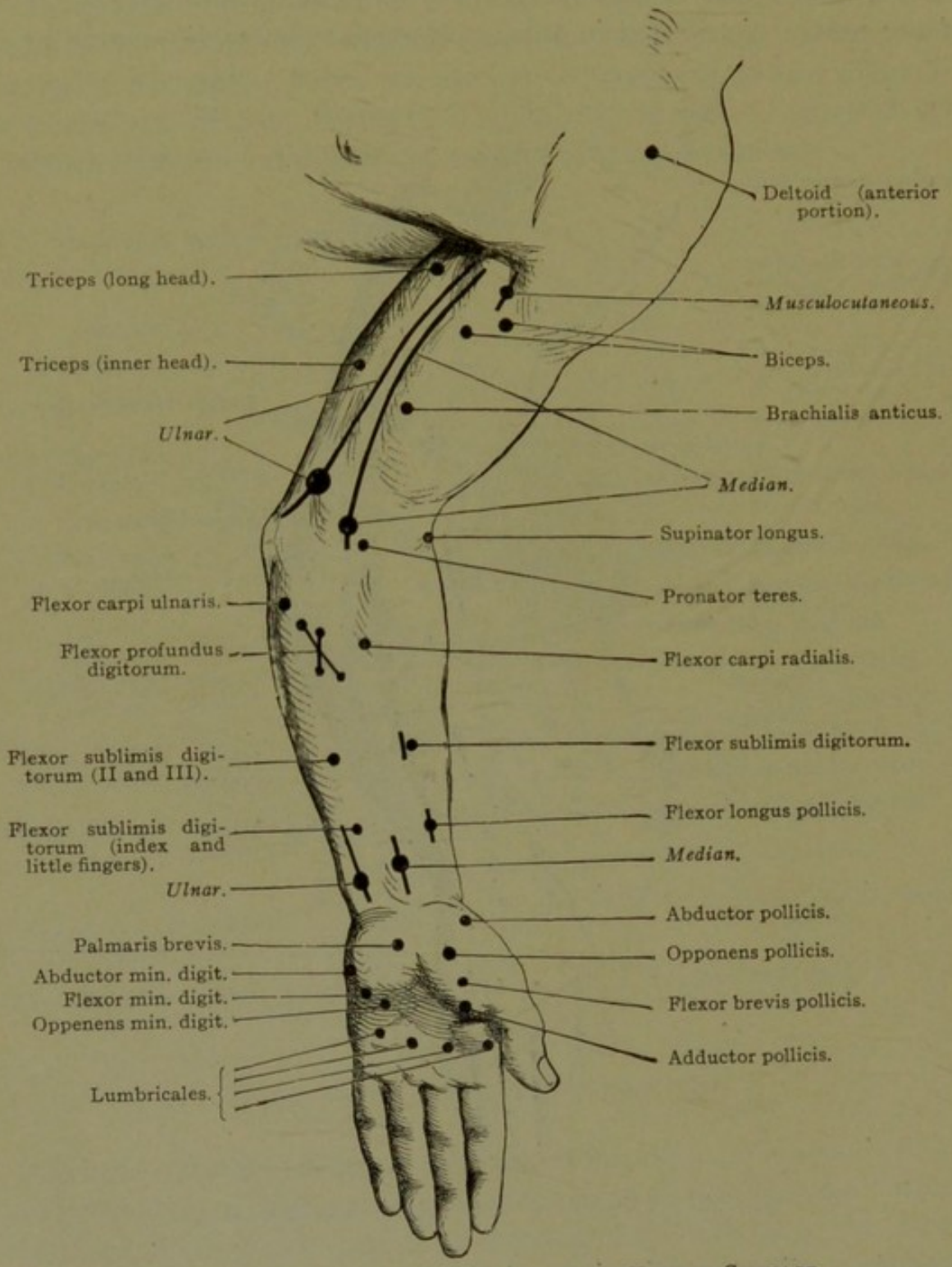


FIG. 56.—MOTOR POINTS ON UPPER LIMB, FLEXOR SURFACE.

The current is slowly turned on and the contact breaker of the battery having been put to its slowest rate of vibration, the testing electrode is placed over the motor point of the muscle or on the nerve trunk.

Normally a muscular contraction from a faradic current is short, stronger in opening than in closing the current. If the current is rapid and follows one another, a tetanic contraction is obtained.

Galvanism.—The electrodes are placed like in faradism. The testing electrode is attached to an interrupting handle and placed upon a motor point. At each closure a short, sharp muscular contraction appears. The galvanometer will register the intensity of the current. Contractions are obtained in cathodic closure (CaCC), cathodic opening (CaOC), anodic closure (AnCC), and anodic opening (AnOC) operations. In

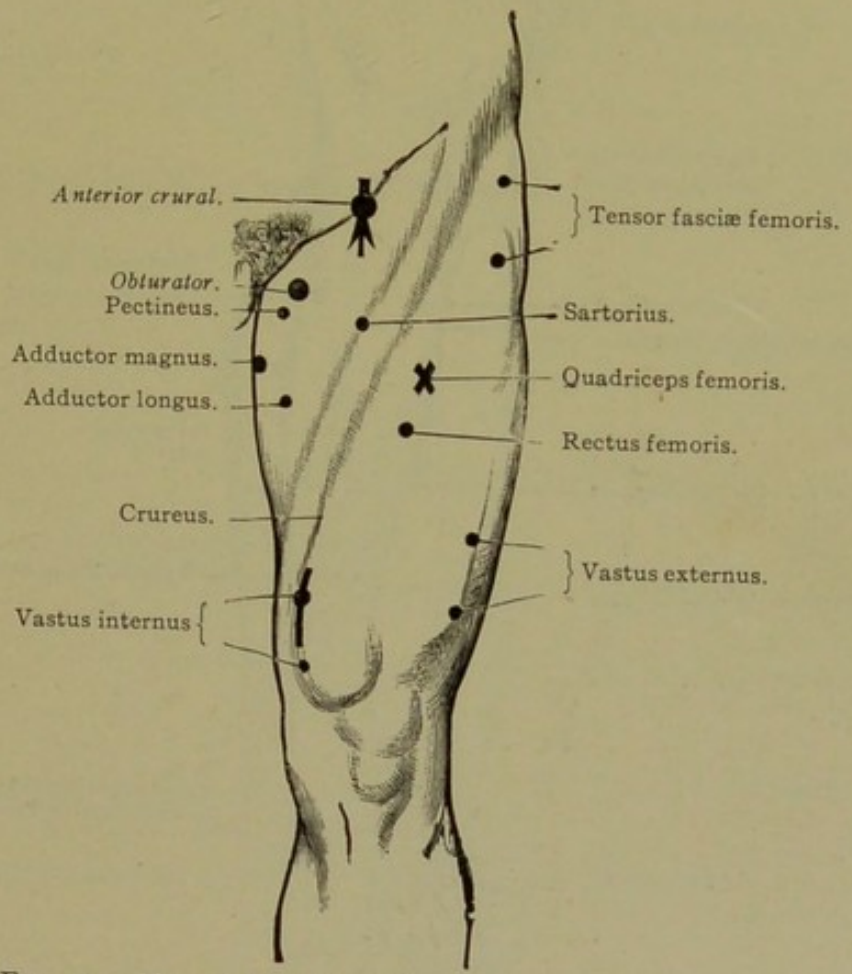


FIG. 57.—MOTOR POINTS ON THIGH, ANTERIOR SURFACE.

health nerves and muscles react differently whether the testing electrode is connected with the Ca or An and whether the current is closed or opened. The following law may be formulated for the nerves: (1) with a weak current a reaction is obtained only from CaCC; (2) with a medium current the reaction from CaCC is the strongest, although anodal contractions are also obtained; (3) with a strong current the CaCC is tetanic, AnOC and AnCC is strong, especially the first, and CaOC is weak. In

direct muscular contractions only the closure contractions are important. The CaCC comes before the AnCC.

Pathological Reactions.—In diseased conditions of the muscles **quantitative** and **qualitative** changes of the electrical reactions are important. As to the first they are of less value than the second. An increased reaction has an importance only in one disease, viz. tetany. There the CaCC and AnOC appears even with very weak currents. A decreased response occurs oftener than an increased, but without any special diagnostic value. Ordinary diminution of electric irritability is observed in

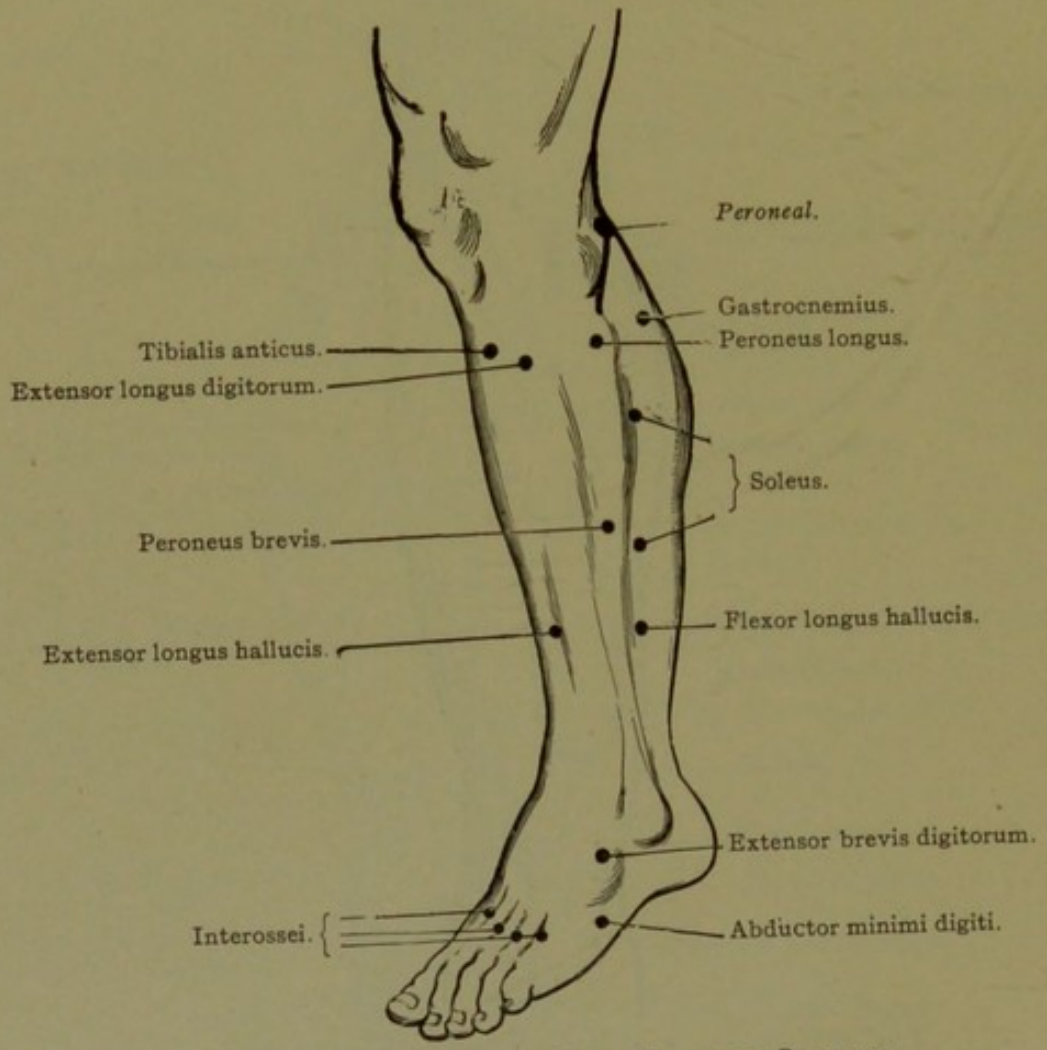


FIG. 58.—MOTOR POINTS ON LEG, EXTERNAL SURFACE.

muscular atrophy without degeneration of nerves and muscles, in muscular wasting accompanying diseases of the joints or cerebral diseases. Complete loss of electrical contractility is observed, when the muscles are entirely destroyed. Of considerably greater importance are the **qualitative** changes, viz. **reactions of degeneration** (**RD**). They may be **complete** or **partial.**

Complete.—1. If a **nerve** is gravely injured, we have in the peripheral portion an increased reaction during the first two days which

gradually decreases and in ten to fourteen days disappears totally. (The reactions are identical with galvanic and faradic currents.)

2. In case of a **muscle** being gravely damaged the galvanic irritability decreases first, but in a couple of weeks increases and becomes exaggerated. The quickness of each individual response is lost and the contractions become sluggish. At the same time there is a change in the normal law of contractions (see above), viz. the AnCC is equal to or stronger than the

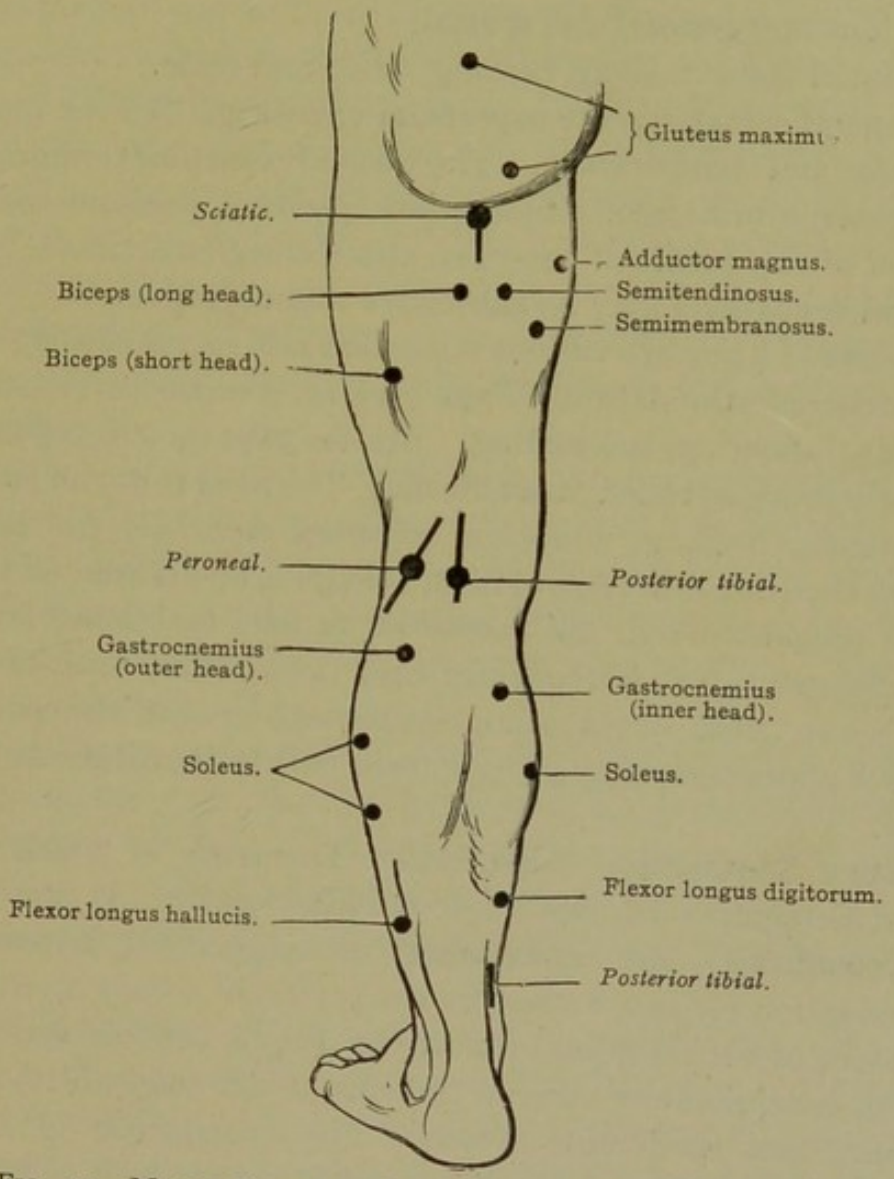


FIG. 59.—MOTOR POINTS ON LOWER LIMB, POSTERIOR SURFACE.

CaCC (reversed formula). Then appear the CaOC and AnOC, which normally do not exist. The increased galvanic irritability exists for one or two months, then gradually disappears, while the RD persists. In incurable cases the galvanic change grows deeper and deeper, has yet a weak AnCC and finally the latter disappears. As to the faradic contractility of muscles in complete RD, it is lost.

Partial RD.—Here the change in the galvanic muscular reactions is the same as in complete RD, but there is no loss of galvanic nerve reaction and no loss of faradic muscle and nerve reactions.

II. SENSORY PHENOMENA

The study of **sensory disturbances** constitutes a very important chapter in diagnosis of nervous diseases.

Sensations are: **general** and **special.**

The special concern sight, hearing, smell and taste.

The general sensations are **superficial** and **deep.** To the first belong: touch, pain and temperature. The second constitute: muscular and osseous senses, vibration of a tuning fork, position, pressure, localization, recognition of two points of compass, also stereognostic sense.

General sensations may be **subjective** and **objective.**

Subjective.—They are various sensations felt spontaneously and without any external stimulation. **Pain** may be continuous or paroxysmal, dull or sharp, shooting, lancinating. Besides **pain** there is a great variety of peculiar sensations called **paræsthesia.** The most common among them are: numbness, tingling, vibration, affecting especially the extremities. Sometimes there is a sensation of heat, cold, pins and needles or electricity. They are encountered in the course of various functional and organic nervous diseases. Not infrequently they precede the onset of a disease.

Pain is a frequent symptom and sometimes its seat, the character and the mode of appearance play an important part in the diagnosis of nervous affections.

Objective Sensations. Superficial.—The sense of **touch** is a very important element in diseases of the nervous system. It may be diminished (**hypæsthesia**), lost (**anæsthesia**) or exaggerated (**hyperæsthesia**). The examination for touch must be conducted in a very careful manner. With a piece of cotton-wool, or with von Frey's hairs or with the finger or with an æsthesiometer various portions of the body are touched very superficially and then more closely. The promptness of response is noticed. If one portion of the body presents changes, it is advisable for comparison to perform the same manipulations on the corresponding part of the opposite side.

The same rules can be applied to the tests for **temperature** (heat and cold) and **pain.** For **temperature** two glass tubes half filled with very hot and very cold water are used. For **pain** the sharp end of a pin or æsthesiometer is used. The three senses may all be abolished, diminished or exaggerated. They may be **dissociated** so that touch is preserved,

pain and temperature are abolished. One of the three may be altered to a certain extent. The heat and cold may be reversed, viz. application of heat will give the sense of cold or vice versa. Sensory **dissociation** is characteristic of syringomyelia, but may be observed also in myelitis, hematomyelia, Brown-Séquard's syndrome.

They may be altered on a half of a body, on a portion of it or on a segment of a limb. Sensory disturbances may follow the course of nerve-trunks (**radicular anæsthesia**). They may be distributed in **islets** on various portions of the body or in **segments** the upper border of which is perpendicular to the axis of the limb (glove-like, stocking-like). Under the name of **Allochiria** is understood an inability to recognize the spot touched or otherwise stimulated or else the patient points to the side opposite to the one touched, as for example when one leg is touched he thinks that it was the other leg. It is met with in hysteria and in some organic nervous diseases.

Deep Sensations. Pressure.—Various weights are used for this purpose. The superficial senses may be abolished, but sense of pressure may be preserved.

Localization.—It can be determined by three procedures: (1) naming, which consists of having the patient tell the spot touched while his eyes are closed; (2) looking, which consists of pointing out the touched spot by the patient (eyes closed); (3) spacing, which consists of touching with two points of the compass or with one point after another. The distance between the points should be noticed.

Muscular Sense and Sense of Attitude.—The patient is told to execute certain movements, the eyes being closed. If errors are made, the muscular sense is disturbed. It is seen particularly in tabes. The sense of **attitude** can be tested by placing a limb or a portion of it in certain positions and by asking the patient with the eyes closed to indicate the position.

Osseous Sensation.—It is tested with a tuning fork. The latter is applied to various accessible portions of the skeleton. A sensation of trepidation is obtained. It may be present in spite of cutaneous anæsthesia. It may be absent in tabes, syringomyelia, hematomyelia (Egger).

Stereognostic Sense.—It is the faculty of recognizing by palpation the form, shape, consistency of objects, also the material of which they are made. The patient is told to close his eyes and the objects are placed in his hand. The loss of this faculty is known under the name of **Astereognosis.**

Asymbolia (or tactile agnosia) is loss of ability to recognize by palpation the nature of an object and therefore to name it. Astereognosis

and asymbolia may exist independently of each other and each of these two senses may be present while all other sensibilities, superficial or deep, are absent. (See my contributions in *Med. Record*, 1910, and *J. of Abnormal Psychology*, 1911.)

Head proposes the following grouping of sensations:

1. **Epicritic** sensibility, to which belong: light touch, light temperature of heat and cold, appreciation of two points of compass and localization on the skin.

2. **Protopathic** sensibility to which belong: extreme temperature of heat and cold, appreciation of pain sense and sensibility of the viscera.

3. **Deep** sensibility to which belong: pressure, position, movement.

Experimenting on himself Head ascertained that section of a peripheral nerve produces a loss of protopathic sensibility over the area of distribution of this nerve and a loss of epicritic sensibility over a larger area. If the nerve contains sensory and motor fibers, deep sensibility is also abolished. When a nerve is only slightly injured, the epicritic sensibility alone is impaired. When a nerve-root is involved, the protopathic sensibility is more impaired than the epicritic. The nearer the lesion lies to the central nervous system, the more definite and more extensive is the loss to pain; the nearer to the periphery, the greater is the loss to cutaneous touch. In this way a diagnosis of the position of the lesion can be made.

III. SPECIAL SENSES

Sight.—Changes in the eyes, especially in the fundi, are of great diagnostic importance. Optic neuritis, optic atrophy, swelling of the papilla are frequent phenomena of cerebral or spinal lesions. The examination should also include the state of the pupils, their light and accommodation reflexes, their shape, their relative size.

The condition of the ocular muscles should be ascertained. Contraction of the visual and color fields is of importance. The visual acuity, partial or complete blindness, hemianopsia—all these symptoms present valuable data for diagnosis. Subjective visual phenomena (sparks, etc.) are not infrequent in functional nervous diseases (for details see Diseases of the Second Cranial Nerve).

Hearing.—Subjective auditory symptoms are of great importance. The acuity of hearing should be investigated, either with a watch, tuning fork or whispered voice. There may be also perverted hearing (for details see Diseases of the Eighth Nerve).

Smell.—There may be a diminution, increase or loss of sense of smell.

A perverted smell also occurs. Smell can be tested by means of various odorous substances.

Taste.—It should be investigated with various substances and electrical current. It may be perverted or lost in nervous affections.

IV. CRANIAL NERVES

The cranial nerves are very frequently involved in organic diseases of the nervous system. Every case should be investigated from this standpoint. Sometimes in doubtful cases the pathological condition of a cranial nerve decides the diagnosis and the localization of the lesion (see Diseases of the Cranial Nerves).

V. SPEECH AND WRITING

Disturbances of the speech are quite frequent in cerebral diseases of a localized nature, in some affections of the spinal cord and sometimes in hysteria.

The function of speech should be investigated from the following points of view: articulation of words, intonation, reading, writing, copying, counting, singing, hearing spoken words. A disturbance of each of these faculties has a special significance. See chapter on Aphasia and Agraphia.

A tremulous speech, a certain manner of pronouncing certain letters or syllables are met in some organic affections of the spinal cord and in neuroses. A careful examination of the speech is necessary and any deviation from normal noticed. Finally the speech may be altered because of intellectual deficiencies, such as idiocy, etc. On the other hand, the patient's previous knowledge or illiteracy should be taken into account in forming an opinion upon his ability to write, read, etc.

VI. REFLEXES

The study of reflexes is of paramount diagnostic importance in neurology.

A reflex action is the result of a peripheral stimulation. The latter follows a centripetal course to a center, from which a motor act is transmitted through the centrifugal pathway.

Reflexes are divided into two great groups: **Tendinous** and **cutaneous** or **deep** and **superficial.**

1. Normal Tendon Reflexes.—To observe a reflex complete relaxation of the muscles must be obtained before the test is made.

(a) **Patellar Tendon Reflex (Knee-jerk).**—The patient is placed comfortably on a chair. He is told to **relax** his muscles and cross his knees. When relaxation is difficult to obtain, he is engaged in a rapid conversation and told not to observe the test. The tendon is outlined and a sharp blow is given with the ulnar border of the hand or with a percussion hammer. A contraction of the quadriceps is obtained and a forward jerk of the leg follows. When the response is very feeble or doubtful, Jendrassik's method should be tried. The patient is told to grasp firmly one hand with the other and pull. If the reflex is present, it will then be obtained or if it is feeble with the ordinary test, will be prompt with this methods.

(b) **Achilles' Tendon Reflex.**—The patient kneels on a chair, relaxes his muscles and a short blow is given in the preceding manner over the tendon. Normally there is a plantar extension of the foot.

(c) **Triceps Reflex.**—The patient's arm is held on its anterior surface and the forearm allowed to hang down, forming a right angle at the elbow. The tendon of the triceps is then percussed. An extension movement follows.

(d) **Biceps Reflex.**—A semiflexed and relaxed position is given the elbow. A slight blow over the tendon will be followed by a flexion movement.

(e) **Masseter Reflex.**—The mouth is slightly opened, a hard object is placed on the teeth of the lower jaw. A slight blow over the latter produces a contraction of the masseter.

The above described tendon reflexes may be increased, diminished or lost. These three conditions are extremely important for diagnostic purposes. They are indications of involvement of the nervous system.

Abnormal Tendon Reflexes. (a) **Ankle-clonus.**—When the patient is seated, the calf-muscle of his semiflexed leg is grasped and held gently in the palm of the hand. With the other hand his foot is brought first downward and then abruptly flexed dorsally. A to and fro movement of the foot is then produced, which may last from a fraction of a minute to several minutes.

(b) **Contra-lateral Movement.**—When the patient lies on his back with the lower limbs semiflexed and the patellar tendon is percussed, besides an extension of the leg on the same side, there is also an adduction of the opposite limb.

2. **Normal Cutaneous Reflexes.**—They consist of muscular contractions produced by irritation of the sensory cutaneous nerves. The contractions may be limited to the area stimulated, but when the stimulation is very marked the contractions spread to other muscles and may invade the entire body.

(a) **Plantar Reflex.**—When the sole of the foot is slightly irritated the toes flex, but when the response is pronounced there will be at first a contraction of the tensor fascia lata, then a dorsal flexion of the entire foot, flexion of the leg on the thigh and of the thigh on the pelvis.

(b) **Abdominal Reflex (Rosenbach's Sign).**—It consists of a contraction of the abdominal muscles upon an irritation of the skin of the abdomen along the outer border of the rectus abdominalis muscle.

(c) **Cremasteric Reflex.**—It consists of a sudden ascension of the testicle when the skin of the internal aspect of the thigh is stimulated.

(d) **Anal Reflex.**—Irritation of the perianal cutaneous surface produces a contraction of the sphincter.

Abnormal Cutaneous Reflexes. (a) **Babinski's Reflex (toe phenomenon).**—It consists of extension of the great toe and sometimes of all the toes, when the sole of the foot is slightly irritated. It is best elicited by stroking the outer margin of the sole or across the balls of the toes. The extension of the great toe may be accompanied by a fan-like abduction of the other toes.

(b) **Oppenheim's Reflex.**—If the handle of a percussion hammer (or any object) is passed from above downward along the inner border of the tibia, producing at the same time slight pressure upon the soft tissue, extension of the great toe or all the toes follows.

(c) **Paradoxical Flexor Reflex** (described by the writer) consists of extension of the great toe or of all the toes when the deep muscles of the calf are pressed upon. To elicit it the patient is seated and his feet (not the legs) are placed on a stool. The limbs are everted and completely relaxed. The examiner places the thenar and hypothenar muscles of his hands on the antero-internal surface of the tibia and turns his fingers downward to the middle of the calf muscles. Pressure on the latter is then produced. Instead of pressing the fingers may roll the calf muscles from side to side.

Other abnormal reflexes will be described in the respective chapters of various organic diseases.

Significance of the Abnormal Tendon and Cutaneous Reflexes.—They are all manifestations of an involvement of the **motor** area and pathway. They are associated with exaggerated normal reflexes. The paradoxical reflex in particular, makes its appearance frequently long before the Babinski's and Oppenheim's signs developed. It is also present in very slight lesions or irritation of the motor tract and motor center.

Abolition of all reflex responses is observed in some organic diseases, such as transverse lesions of the cord, in poliomyelitis, in tabes, finally in coma.

VII. SPHINCTERS

The involvement of the sphincters of the bladder and rectum is a frequent manifestation in organic nervous diseases. It also occurs occasionally in the course of functional nervous disorders.

Retention, incontinence, imperative and frequent micturition (or defecation) are the symptoms to be investigated.

VIII. VASOMOTOR AND TROPHIC DISTURBANCES

They are very frequent in nervous diseases. Localized œdema, localized adiposis, local asphyxia of the extremities, herpes zoster, disturbed, function of sweat glands, bed-sores, hemiatrophy, localized or generalized atrophy—may all be encountered in nervous diseases.

CHAPTER III

CEREBRAL LOCALIZATIONS

ANATOMICALLY the brain presents homogeneous masses of gray and white matter, but physiologically it is composed of portions, areas or **centers,** the functions of which are different from each other. The conception of centers was admitted even as far back as 1825. In 1861 Broca first localized the speech center. But a solid foundation to the existence of cerebral localizations was laid by the experiments of Fritsch and Hitzig (1870) and later of Ferrier on animals, and the clinical observations of Hughlings Jackson. Since then pathological observations began to accumulate and they corroborated the experimental researches. Thus regional diagnosis of diseases of the brain became facilitated and gained an anatomical basis. Surgery benefited considerably from this new acquisition of cerebral physiology.

MOTOR CENTERS

Until recently the motor area of the cortex was considered as consisting of the ascending frontal and ascending parietal convolutions, also the paracentral lobule. Considerable doubt has arisen lately as to the motor function of the ascending parietal convolution. First of all, Sherrington and Grünbaum have demonstrated on anthropoid apes that excitation of the ascending parietal by strongest electrical currents failed to produce movements, while identical stimulation of the whole ascending frontal convolution and of the Rolandic fissure evoked movements on the opposite side of the body. Also stimulation of the bases of the frontal convolutions was followed by movements of the eyeballs. Observations have also been made in man as to effect of electrical stimulation of the Rolandic area (Mills, Keen, Horsley, Dana, Cushing, Lloyd, Gordon, etc.). By using the unipolar method they obtained movements only from the ascending frontal convolutions. There are cases on record showing that lesions of the ascending frontal alone produced during life paralysis of the opposite side, while no motor symptoms were present when the lesion was found in the postcentral convolution. Of considerable importance are also the differences in the structure of postcentral and the precentral convolutions. According to Campbell the cortex of the precentral convolution contains the

giant-cells of Betz and an abundance of nerve-fibers. The postcentral convolution has no Betz cells and is not so rich in nerve-fibers. The Betz cells seem to be the origin of the pyramidal tracts. As a last argument in favor of the exclusive motor character of the precentral convolution is found in Flechsig's embryological studies.

On the other hand, there are some facts which tend to prove that the so-called motor centers have also a sensory function, that they are **sensori-motor.** Horsley (*Brit. Med. Jour.*, 1909, p. 125) removed the arm center of the precentral convolution in a patient suffering from convulsions limited to the arm. The result was: paralysis of the arm, astereognosis, anæsthesia to all forms of sensations. A year later both motion and general sensations improved. In view of the absence of the precentral portion of the arm the postcentral controlled the movements and sensations. Clinical studies of motor paralysis of cerebral origin show that not infrequently sensory disturbances are present in the paralyzed limbs. Tripier, Golgi, Exner, Starr, Soury, Dejerine and others have given ample anatomo-clinical proofs of this contention. In my personal study (*Jour. Nerv. and Ment. Dis.*, 1903, p. 144) of 35 cases, I found in some a distinct parallelism between the motor and sensory disturbances in regard to distribution and degree. My investigations by means of faradization of the cortex (*Jour. of Amer. Med. Assn.*, June, 1907) show that the ascending parietal convolution participates in formation of motor area, although to a considerably lesser degree. In Rossi and Roussy's two cases (*Revue Neurologique*, 1906) and in Souques and Barbé's case there was a slight involvement of the ascending parietal convolution. Considering all the accumulated evidences, clinical, pathological and experimental, one is more inclined to admit that the motor and sensory cortical areas are distinct, that the precentral convolution is purely motor, that the postcentral is only sensory, and that the motor area includes the precentral, anterior part of paracentral, the bases of the first two frontal convolutions, also the Rolandic fissure.

The motor nature of the special portion of the cortex is easily recognized from the convulsive and paralytic symptoms produced by an irritation or a destructive lesion of these centers. The latter control each individual muscle or groups of muscles of the **opposite** side of the body. It is therefore important to describe the seat of each individual motor center. For practical purposes it is sufficient to consider three chief portions of entire motor zone corresponding to the limbs and head. The center for the lower extremities occupies the anterior part of the paracentral lobule and the upper fourth of the ascending frontal convolution, and that of Rolandic fissure. The two middle fourths represent the center

for the upper extremities. The center of the head lies in the lower fourth and in the rolandic operculum. The center for the trunk lies between those of the extremities.

Clinical observations on focal epilepsy or electrical stimulation of the motor centers have shown that in each of the above areas exist secondary centers which correspond to the function of muscles of segments of the limbs. Thus there are centers for the shoulder, elbow, wrist, fingers, thigh, knee, ankle, toes; for the head individual centers control the movements of the face, tongue, lips, pharynx and larynx. The larynx has two centers—one for each of its two functions, viz. respiration (abduction

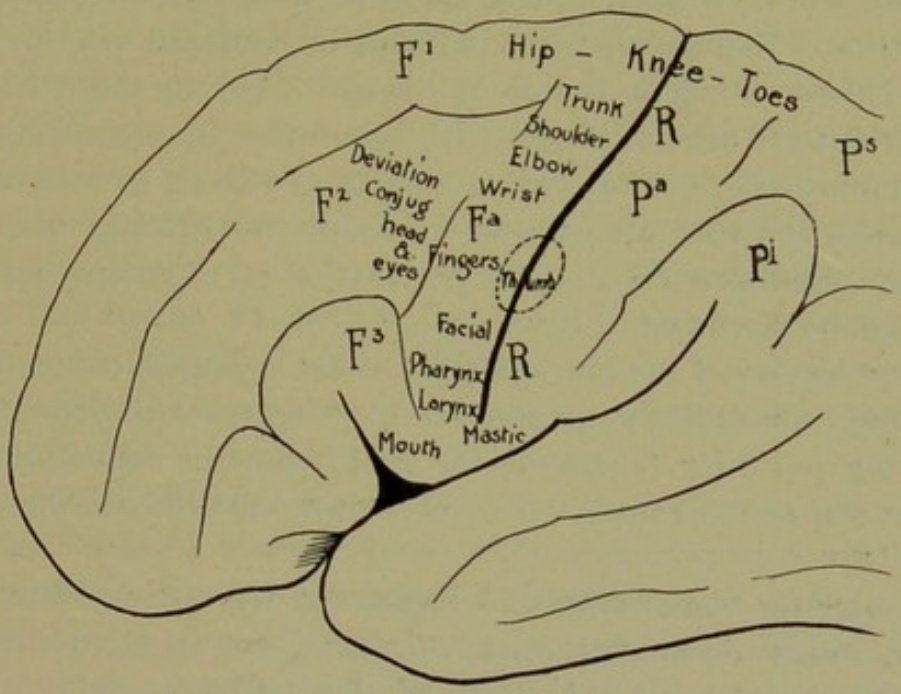


FIG. 60.—MOTOR CENTERS. (*After Lamacq.*)

of the vocal cords) and phonation (adduction of the vocal cords). The center for the conjugate deviation of the head and eyes lies in the foot of the second frontal convolution. The accompanying illustration shows the relative seat of these various centers.

It is well understood that **each center** controls muscular groups of the **opposite** side of the body and the reason of it lies in the decussation of the pyramidal fibers. There is, however, a certain group of muscles which are under the influence of centers of both hemispheres, in other words, some centers control simultaneously muscles of both halves of the body. Such **bilateral centers** exist for: (1) The muscles of the eye; elevation and lowering of both eye globes are produced simultaneously. (2) The muscles—orbicularis palpebrarum and frontalis. The integ-

rity of the superior facial nerve in organic facial palsy (see hemiplegia) is also due to bilateral innervation of the upper part of the face. (3) The muscles of mastication. (4) Some muscles of the tongue. (5) Muscles of deglutition. (6) Muscles of the larynx. (7) Muscles of respiration—diaphragm and intercostal.

SPEECH CENTERS

They are situated along the Sylvian fissure and in the left hemisphere for right-handed, in the right hemisphere for left-handed individuals. There are four speech centers: one serves to receive words, another to receive read words, a third to express in an articulate language what is heard or seen. Expressing what is heard or seen can also be done by **writing.** Hence a fourth center has been added to the above three. These four centers are associated with each other by commissural fibers of great importance, so that destruction of one leads to interruption of its communication with others. In the chapter on **aphasia** various phenomena of speech produced by the destruction of these anatomical connections will be discussed at length.

The localization of the **four speech** centers is distributed as follows:

The center for **auditory images,** viz. for comprehending spoken words, occupies the posterior fourth of the first temporal convolution (**left**). A lesion of this center causes a form of sensory aphasia known as **"word deafness."**

The center for **visual graphic images,** viz. for distinguishing written or printed words, occupies the **left** angular gyrus (postero-inferior part of the left parietal lobe). A lesion of this center produces a form of sensory aphasia known as **"word blindness"** (**alexia**). These two centers, a lesion of which is responsible for "sensory aphasia," are known as **Wernicke's area** and the speech defect as **Wernicke's aphasia.**

The center for the faculty of **writing** lies in the foot of the second frontal convolution, a lesion of which produces inability to write—**agraphia.**

The motor center for pronouncing and articulating words, as discovered by Broca, occupies the foot of the third frontal convolution (**left**), immediately in front of the centers of phonation utilized in speech. The destruction of this center produces **motor aphasia** (aphemia). The speech and writing functions are frequently involved together because of the proximity of their two centers and of the commissural fibers connecting these centers with the auditory and visual centers. As to the **new view** advanced recently by P. Marie, see chapter on Aphasia.

SENSORY CENTERS

(a) **General Sensations** (touch, pain, temperature, muscular sense).—Although the question is still somewhat debatable, it is nevertheless admitted by the majority of competent writers that the motor and sensory spheres of the cortex are separate. In the chapter on motor center it was shown that the prevalent opinion is presently in favor of the ascending parietal convolution being the center for general sensations. The parietal lobe presents three important portions: superior lobule, supramarginal lobule and angular gyrus. The parietal lobe forms a part of Flechsig's posterior **association area.** The superior parietal lobule together with the postcentral convolution can be considered as a cortical center for general sensibility. An irritative lesion in this area will cause Jacksonian epilepsy with a sensory aura. Flechsig believes that the sensory cortical area corresponds to the large surface sensory area which comprises the skin, mucous membrane, muscles, articulations and viscera. All forms of sensations (touch, pain, temperature and muscular sense) are represented in this center, but nothing is known in regard to a separate localization for each individual form. There is reason to believe that the sensory area contains also centers for the nerves of secretory glands and for vaso-motor nerves (Adamkiewicz, Bechterew).

Stereognostic Sense.—Under this term is understood the faculty of recognizing the form, size and consistency of objects by touch. To elicit it various objects are placed separately in each hand of the patient, his eyes being closed. Inability to recognize the characteristics of the objects constitutes **asterognosis.** Although muscular sense, tactile and temperature sensations, also those of weight and pressure, participate in perception of an object placed in the hand, stereognosis is nevertheless considered as a special sense which has a special cortical center. It was mentioned on page 69 that in spite of the integrity of various elementary sensibilities the stereognostic sense may be abolished. Astereognosis has been often observed in lesions of the superior parietal lobule (Mills, Burr, Starr and others) or of the supramarginal gyrus. This is, however, not the exclusive seat of the stereognostic sense, as in lesions of the Rolandic area asterognosis has also been noted. On the other hand, in lesions of the parietal lobe the stereognostic sense has also been found intact as it can be seen from my case (*Med. Record*, 1908) in which a bullet entered the left parietal region and subsequent operative procedures lacerated very extensively the superior parietal lobule without involving this special sense.

SPECIAL SENSATIONS

(a) **Visual Centers.**—The cortical centers for vision occupies the cuneus and the calcarine fissure in either hemisphere. Most frequently a lesion of this area in one hemisphere will produce a **hemianopsia,** viz. a blindness in one-half of both eyes on the side opposite to the lesion and total blindness if the lesion is bilateral. The convexity of the occipital lobe is also concerned in vision so that hemianopsia may also be the result of a lesion of the occipital cortex.

The occipital lobe is connected with sub-cortical centers of the visual apparatus, viz. with anterior quadrigeminal bodies, pulvinar and external geniculate body—by means of the fibers of optic radiations, which pass through the retro-lenticular segment of the internal capsule (see Anatomy). Does a lesion of any of these portions cause blindness of the visual field? The most recent investigations show that an involvement of the optic radiations, of the chiasma or of the optic nerve produces hemianopsia, a lesion of the pulvinar may (exceptionally) do it, but there are no indisputable proofs for hemianopsia due to diseases of the quadrigeminal bodies or of external geniculate body. As to the angular gyrus, it is the center for word-seeing. A lesion of it will produce **word-blindness** (**alexia**) (see chapter on Aphasia).

(b) **Auditory Centers.**—Very few cases of pure deafness came to autopsy, but they show that the cortical center of hearing is in the **temporal lobe.** According to Ferrier, Seppilli and others, the first temporal convolution is particularly concerned in the function of hearing. Flechsig, Bechterew and Monakow have shown that the posterior quadrigeminal bodies, the internal geniculate body are in relation with the cortex of the temporal lobe, consequently with the auditory area. The function of "word-hearing" (see above) is not necessarily connected with that of hearing in general, as patients affected with "word deafness" are not deaf, generally speaking. It is, therefore, wise perhaps to conclude that while "word deafness" is associated with a lesion only of the left hemisphere, the general audition is controlled by **bilateral centers.** Broadbent observed a case of a deaf-mute in whom atrophy of both superior temporal gyri was found. Mills describes a case of a woman deaf for thirty years in whom both superior temporal gyri were atrophied.

(c) **Olfactory and Gustatory Centers.**—Long ago Ferrier observed from his experiments that destruction of the **apices of both temporal lobes** led to loss of sense of taste and smell (anosmia). Some writers corroborated this observation. According to Flechsig the olfactory

center is in the **cornu Ammonis** and **hippocampus.** Campbell's histological investigations (Cambridge University Press, 1905) suggest that the lobus pyriformus, which consists of the external root of the olfactory lobe and of the gyrus hippocampi, must be considered as the principal cortical center of the olfactory sense. On the other hand, some anatomo-clinical observations point to the uncinate gyrus as the cortical seat of taste or smell senses. J. Hughlings Jackson speaks of cases of Epilepsy with intellectual auræ (dreamy state) associated with sensations of smell. Buzzard in 1905 reports similar cases in which lesions were found in temporo-sphenoidal lobes (*Lancet*, 1906, p. 1808). Although the few records found in the literature speak in favor of the mentioned cortical areas, nevertheless there is no absolute proof of the existence of cortical centers for these two special senses. Zuckerkandl's observation is, however, significant in this respect. Animals with a highly developed sense of smell possess a large **limbic lobe** (see Anatomy); the latter is atrophied in animals whose olfactory sense is rudimentary or absent.

Center for Intelligence.—There is no unanimous opinion as to the localization of human thought in the brain. Intelligence is the result of function of all the centers combined and of the association fibers which serve to connect the centers. However, observations made by Flechsig, Hitzig, Ferrier and others point to the **prefrontal lobe** as the most prominent part of the brain where superior psychic processes are elaborated. The history of tumors or of other lesions of the prefrontal area seem to corroborate this view (see Tumors of Brain, also my article on Function of Prefrontal Lobes in *Jour. Amer. Med. Ass.*, 1907). Mabille and Pitres (*Revue de Méd.*, No. 4, 1913) report the history of a patient who lost the ability of recalling perfectly events of his past life, of fixing any new memories, also of appreciating his present surroundings. Autopsy showed two symmetrical areas of softening, one in each hemisphere in the midst of the white substance of the prefontal lobes;

CHAPTER IV

APOPLEXY

HEMORRHAGE. EMBOLISM. THROMBOSIS

THIS morbid condition is characterized usually by a sudden loss of consciousness with complete or partial loss of power and sensations on one side of the body (in some cases consciousness is preserved).

Morgagni was the first to show that apoplexy has an anatomical reason in the brain or on its surface. For a long time apoplexy and cerebral hemorrhage were considered synonymous, but in 1819 a new era in the history of the subject commenced with a series of anatomopathological works which rapidly followed one another, all tending to prove that other factors besides hemorrhages are apt to cause apoplexy. The most common immediate causes as accepted at the present are: **Hemorrhage, Embolism** and **Thrombosis.**

These three conditions lead to one final result, namely hemiplegia, but the onset, the course of the disease and the pathological lesions are not identical. A separate description is therefore justifiable.

A. HEMORRHAGE

Pathology.—Rupture of a blood vessel may occur in the brain substance or on its surface. The most frequent seat, however, is the internal capsule and the neighboring central ganglia between which the capsule is located (see Anatomy, page 31). The reason of it lies in the fact that the small vertical arteries which are distributed to these parts (namely the lenticulo-striate and lenticulo-thalamic arteries), having no collateral branches, are under a relatively high pressure and therefore are more apt to rupture than the cortical vessels, for example, which branch a great deal. Durand-Fardel has shown that these same arteries develop frequently miliary aneurisms, which means degeneration of vessel wall and hence rupture. They were deservedly called by Charcot "arteries of cerebral hemorrhage." Degenerative conditions of the blood vessels are therefore the chief factor in cerebral hemorrhage. When the vessel-wall is altered (peri-endarteritis), miliary aneurisms are apt to form (Charcot and Bouchard). But the important exciting element is the rise of arterial pressure, which, when it reaches the short terminal arteries of the central ganglia, facilitates their rupture. In aged individuals

Ferrand called attention to a special state of cerebral tissue characterized by small cavities (**lacunes**) in the center of which is seen an artery. The cavities are due to a disintegration of nervous elements. As the blood pressure is invariably increased in advanced age, the atheromatous central artery is easily ruptured when the blood vessel is under high tension.

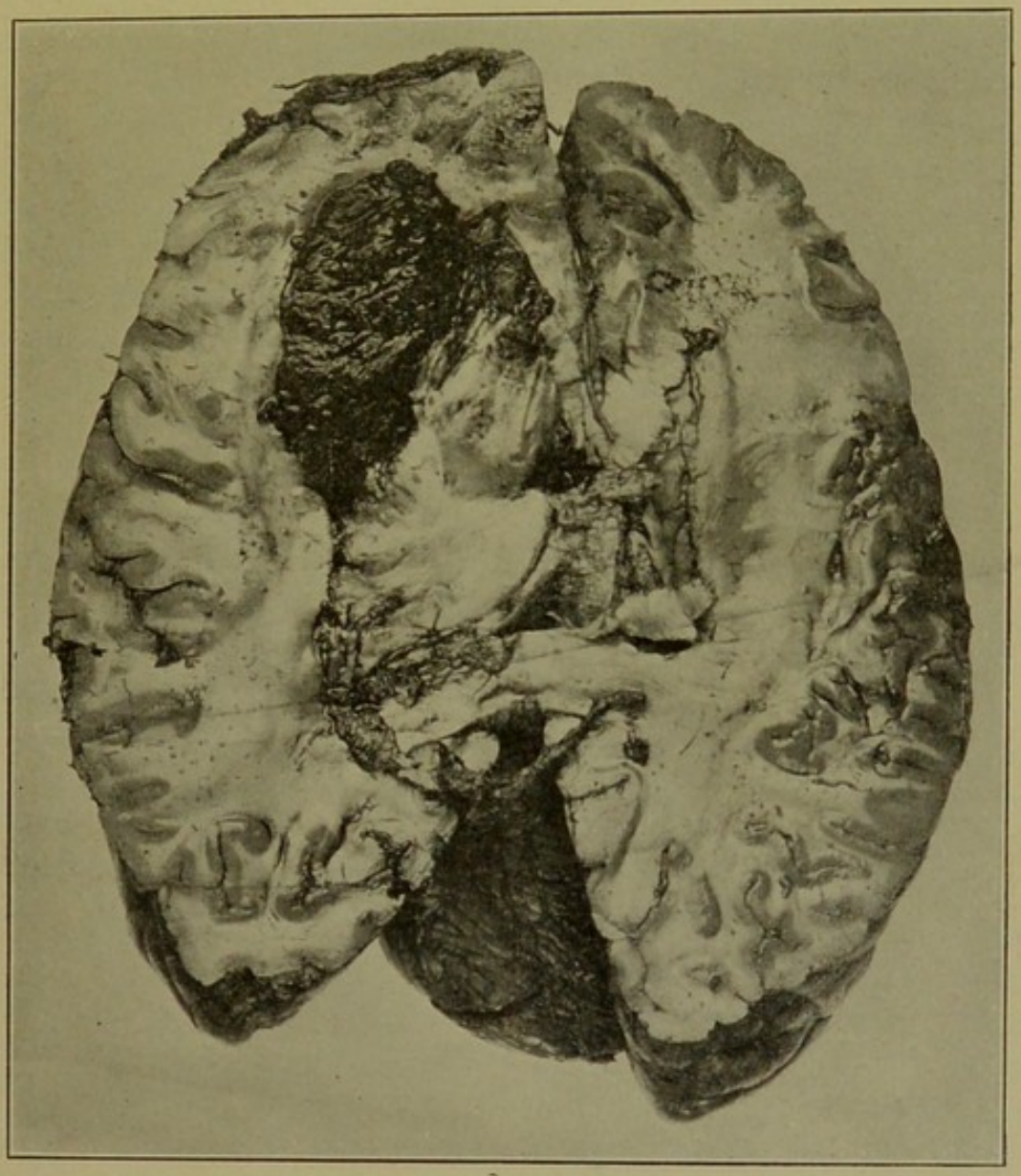


FIG. 61.—EXTENSIVE HEMORRHAGE IN THE ANTERIOR CORNU OF THE LEFT LATERAL VENTRICLE. SUDDEN DEATH. (*Original.*)

The clot which forms immediately after a cerebral hemorrhage and the adjacent brain tissue undergoes certain changes. The red color of the clot gradually changes into yellow. The nervous tissue, which is affected by the clot, is liquefied and eventually becomes absorbed; the neighboring tissue, being compressed, suffers in its turn; the connective tissue and the neuroglia proliferate and form a thick capsule around the

softened area and a cyst containing a yellowish fluid is the result. When the contents of the cyst is absorbed its walls shrink and a cicatrix is formed. Frequently the course of events is not so favorable. The ruptured artery continues to ooze and the destruction of brain tissue continues. If the

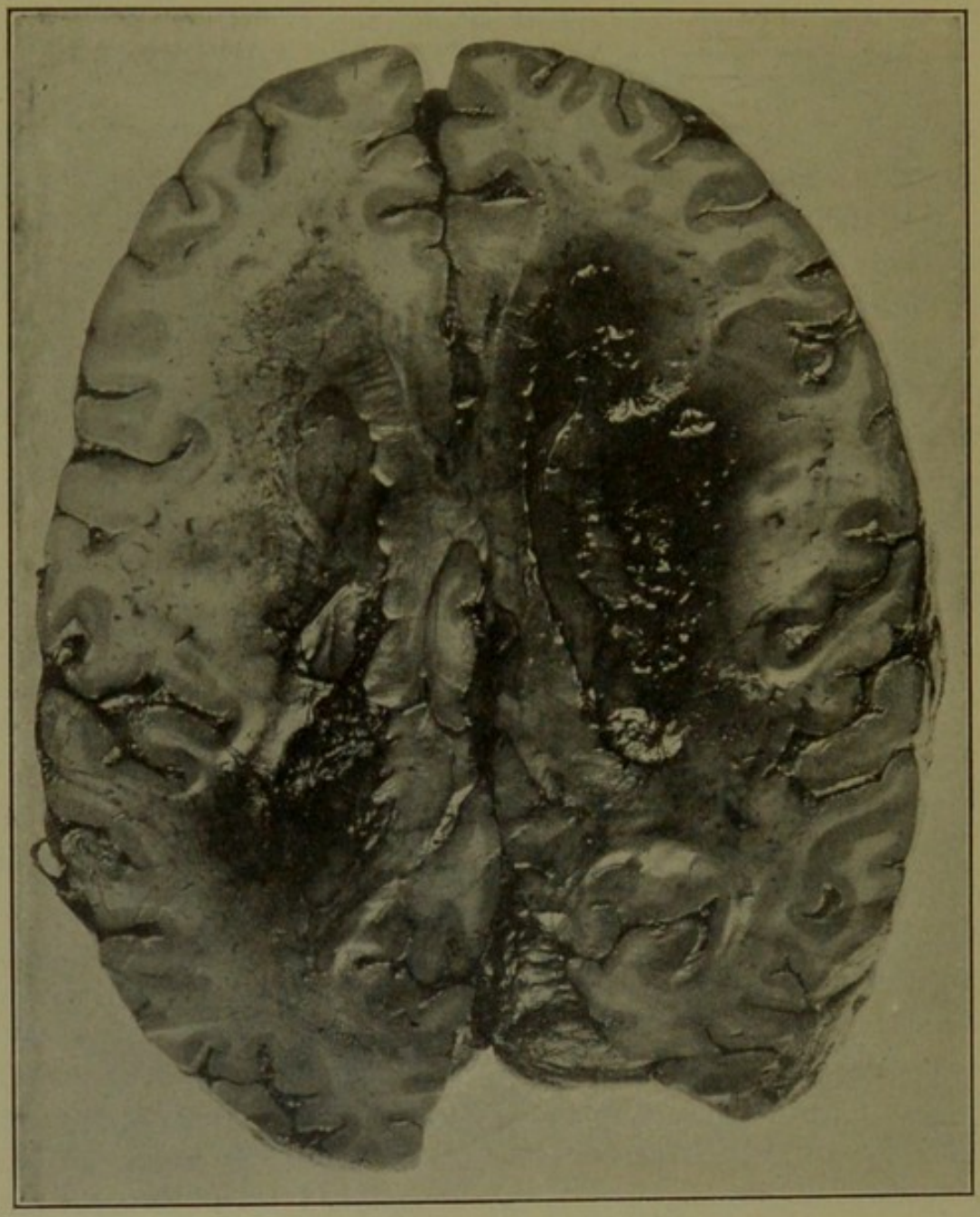


FIG. 62.—EXTENSIVE HEMORRHAGE IN BOTH HEMISPHERES. RAPID DEATH. (*Original.*)

hemorrhage is not stopped, it may reach the ventricles: various structures of the brain undergo then considerable pressure (Figs. 61 and 62).

When a hemorrhage occurs on the surface of the brain, beneath or within the membranes, the clot may not become organized and destruction of cortical tissue may be avoided, so that the brain will resume its function when the clot is removed.

The most important consequence of cerebral hemorrhage is its remote effect on the nervous tissue beneath and in the immediate vicinity of the area destroyed by the clot. Secondary degenerations, which promptly set in, are the usual result. They may be traced through an entire tract. A hemorrhage, for example, in the posterior limb of the internal capsule will be followed by a descending degeneration in the entire motor pathway, even in the lowest portion of the cord. The degeneration is characterized at first by disappearance of the myelin and nerve-fibers which are ultimately replaced by connective tissue, and a scar is thus formed.

Etiology.—Disease of the blood vessels is the chief cause of hemorrhage. The most important factors producing changes in the vessel walls are: **advanced age, intoxications** and **infection.** Although no age is exempt from cerebral hemorrhage, nevertheless degenerative condition of arteries resulting in arteriosclerosis is proper to the degenerative period of life and the majority of cases of cerebral hemorrhages occur after forty years of age. According to the records of St. Bartholomew's Hospital the largest percentage is between the ages of forty and fifty and decrease with the following decades. Among intoxications, syphilis and lead occupy the first place and their influence is augmented when they are present in advanced life. Alcohol also predisposes to atheromatous state of blood vessels. Degenerative conditions of cerebral blood vessels are also observed in pernicious anemia, purpura, scurvy; in Bright's disease; finally in conditions producing cardiac hypertrophy, which cannot be compensated.

As to sex, men are more liable to cerebral hemorrhage than women. Sometimes a hereditary predisposition is observed. In a case under my observation, a young man of twenty-seven died suddenly after a violent paroxysm of whooping-cough from ventricular hemorrhage. His arteries and other organs were found totally normal. Three members of his family died from apoplexy when young and in good health (Fig. 62).

In the majority of cases of cerebral hemorrhage the altered vessel wall will rupture under the influence of raised blood pressure and this occurs in severe muscular efforts, as, for example, in lifting heavy weights, in the act of difficult defecation, in parturition, in paroxysms of severe cough, coitus, attacks of anger, excitement, etc. Finally, intra-cerebral hemorrhage may occur irrespective of arterial alterations, namely from injuries to the head: fractures of the skull, blows or a simple contusion. Hemorrhages on the surface of the brain are usually the result of direct cranial trauma, although it may occur under the conditions related above.

B. SOFTENING OF THE BRAIN (EMBOLISM AND THROMBOSIS)

Pathology.—Cerebral softening is the result of an interruption of blood supply which is usually produced by an embolus or thrombus. When this occurs, the first effect will be a local anemia and a change in the consistency of the tissue supplied by the obstructed vessel. At the end of twenty-four

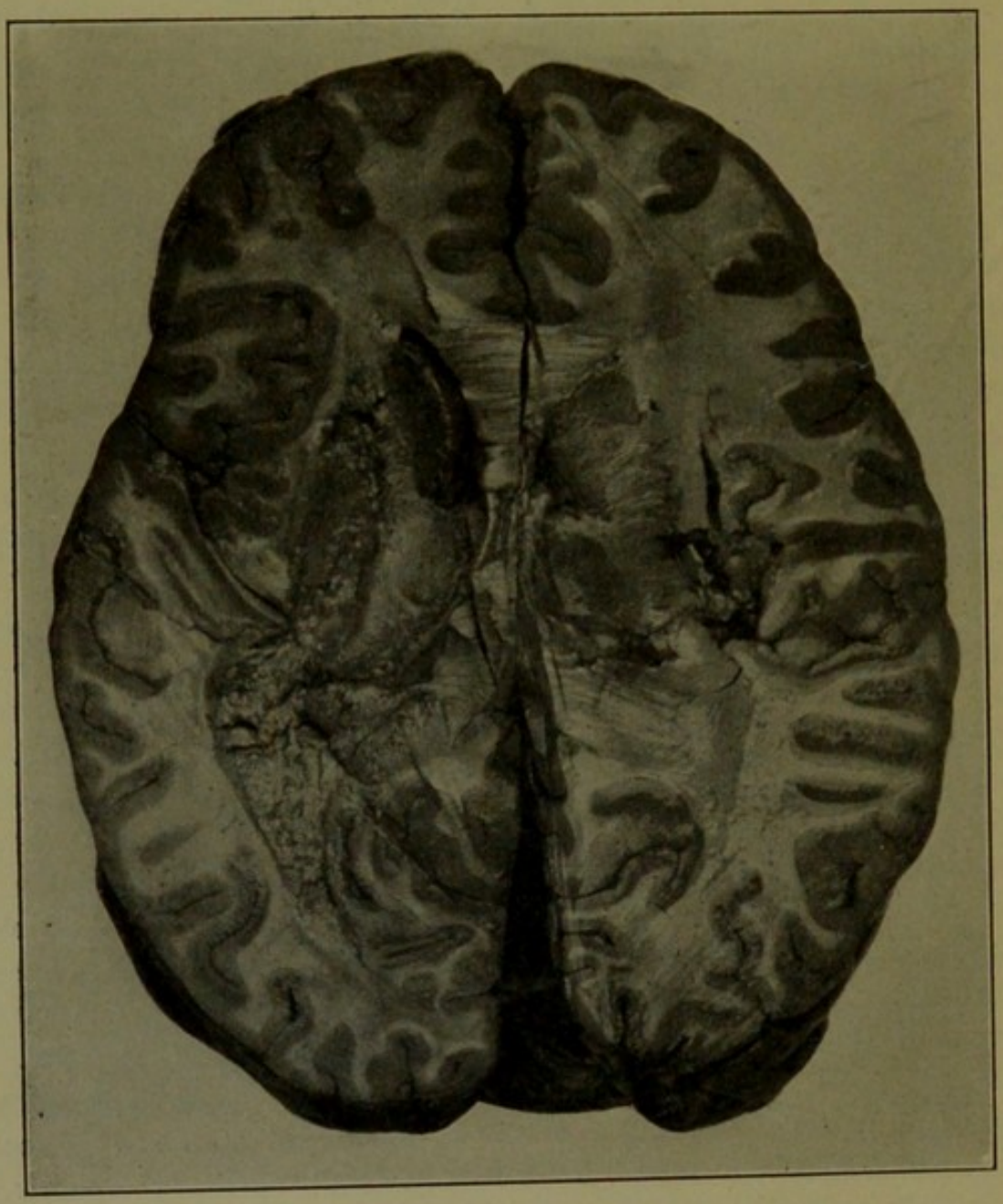


FIG. 63.—EXTENSIVE SOFTENING IN THE LEFT HEMISPHERE, INVOLVING THE INTERNAL CAPSULE, BASAL GANGLIA AND CORONA RADIATA. (*Original.*)

hours effusion of serum (œdema), breaking up of nerve elements and softening take place. The color of the affected area is at first white because of the local anemia, but later on, when the return circulation is established, some corpuscles escape with the serum under high pressure

into the tissue and give the softened area a red color (red softening) which still later (in the fourth week) changes to yellow because of the degenerative changes in the effused blood. Yellow and red softening is observed chiefly in the convolutions, viz. in the gray matter; white softening in the white substance of the brain.

In but a few days after an onset the brain tissue is in a state of degeneration. Cells, neuroglia, myelin are dissociated, and the leucocytes which escape from the capillaries are in abundance. The entire affected area undergoes fatty degeneration. In an advanced state of degeneration (red softening) punctiform hemorrhages in the midst of softened tissue are seen. Yellow softening belongs to old cases: the coloring matter of the blood is modified and the softened area is transformed into fatty substance. The ultimate result of softening is either formation of a cyst in the place of the softened focus or formation of a cicatrix. The latter is due to proliferation partly of the neuroglia and partly of the adventitia of the blood vessels. As to the obstruction in the vessel itself, it is constituted either by a clot or a particle of fibrin carried from the diseased valves of the heart, or by a fragment of an atheromatous plaque. The clot usually undergoes changes, degenerates and sometimes calcifies. The obstructed blood vessel may become a fibrous cord. As some cases of embolism recover, it is probable that the collateral circulation interferes in such cases and assists in keeping up the nutrition of the area supplied by the blocked artery. In other cases sudden death may occur long after the onset of the apoplexy. Aneurisms were found in these cases on one side of the embolus and death was due to their rupture. In thrombosis recovery cannot take place, because the diseased vessel is surrounded by other diseased vessels (atheroma) and collateral circulation is of no special usefulness.

Softening may occur in any part of the brain, but the most frequent seat is in the basal ganglia and internal capsule. The left hemisphere is more frequently affected by embolism than the right. The middle cerebral arteries and their branches are the usual seat for embolism and the arteries at the base for thrombosis, (Fig. 63).

Etiology—Embolism and thrombosis, which are the causes of cerebral softening, have a different etiology. In a general way it can be said that embolism is of cardiac origin, while thrombosis is of arterial origin. In the majority of cases an inflammatory condition of the heart (endocarditis) is the cause of embolism, although an embolus may come from other sources, from the lungs, for example. Vegetations on the mitral valve, clots from a diseased heart may become detached in a physical effort, in a shock or a severe cough and thrown into circulation, producing an embol-

ism in the brain. In ulcerative endocarditis the detached particles will carry microörganisms into the cerebral arteries and thus produce besides embolism an inflammation of the surrounding tissue. A disease of the pulmonary vein, a disease of the lung, such as empyema, are apt to send embolic clots into the cerebral blood vessels.

In **thrombosis** the primary lesion lies in the arteries. An endarteritis narrows or obliterates the lumen of the blood vessel, coagulation of the blood follows. The usual causes of formation of thrombus are syphilis in young individuals, arteriosclerosis in advanced age, chronic intoxication, such as alcohol, lead, etc. There is another condition which deserves special mention that leads to thrombosis, viz. weakness of the myocardium with the resulting retardation of circulation. This is seen in cachexia without atheromatous changes in the blood vessels, in infectious diseases, in chlorosis, in typhoid fever, in diphtheria, in pneumonia. The state of blood in diabetes and gout favors its coagulation and therefore thrombosis. In old individuals unusual exertion is apt to produce thrombosis; the latter commonly occurs in the night during sleep after fatigue.

The question as to the cause of the development of thrombosis in these diseases is not entirely settled. It is, however, probable that microörganisms when present in the blood increase its coagulability or develop an endarteritis, which leads to formation of thrombi.

Symptoms (of Hemorrhage, Embolism and Thrombosis).—The most conspicuous manifestation of apoplexy among other symptoms is a paralysis of one side of the body. The onset, the course and the accompanying symptoms may vary in apoplectic seizures caused by embolism, thrombosis and hemorrhage, but the inevitable result of all these conditions is almost always hemiplegia. It is therefore appropriate to indicate first the differences and then conclude with a chapter on hemiplegia.

Symptoms of Hemorrhage

An apoplectic stroke may be preceded by prodromes, may set in suddenly or gradually; with or without loss of consciousness. The usual precursory symptoms are: vertigo, sensation of fullness in the head, headache, tingling in one-half of the body, nausea, vomiting, ringing in the ears and speech disturbance. The loss of consciousness follows rapidly. The latter may present various degrees; in some case it is absolute, in others partial. The comatose state in which the patient is found after a stroke is usually attended by a loss of all voluntary movements and sensations, by a congestion of the face, by a difficult, superficial and noisy breathing. The pupils are usually dilated and do not react to light.

In view of the hemiplegia which ordinarily follows apoplexy the cheek on the paralyzed side is flaccid and puffed out during each act of expiration. The cutaneous reflexes are abolished. Frequently there is involuntary evacuation of urine and feces. The temperature drops down one or two degrees at the beginning, but a few hours later rises to normal; in fatal cases it may rise to 105° and even to 108°. In fact, elevation of temperature in the course of cerebral apoplexy is of a very grave omen. If recovery is to follow, the patient recovers consciousness; symptoms of amelioration begin to appear at the end of twenty-four or forty-eight hours. Pulse, respiration, temperature and the various disturbed functions gradually return to normal. Then symptoms of **local paralysis** become evident. They will be discussed in detail in the chapter on hemiplegia. In grave cases the loss of consciousness may become deeper and deeper and the coma may last for days before death (**Ingravescent apoplexy**). Irregular breathing during the coma and especially of Cheyne-Stokes' type presents a grave outlook. The same can be said of an irregular pulse.

There is an important symptom which deserves special mention, as it frequently accompanies apoplexy and has, in my opinion, a diagnostic value. The symptom is called **"conjugate deviation of the head and eyes."** At the onset, especially of cerebral hemorrhage, the deviation of the head and eyes is toward the paralyzed side. When the irritating lesion is only temporary, the condition will gradually change: the patient will turn head and eyes toward the lesion; the latter is the most common attitude. Milian (*Progrès Médical*, 1908, No. 18) has recently called attention to two symptoms which during the comatose state will enable to determine the side which is going to be paralyzed. They are: loss of the abdominal reflex and of the corneal reflex.

Symptoms of Embolism and Thrombosis

The apoplectic stroke is not as **intense** and not as **durable** as in cerebral hemorrhage. The loss of consciousness, which is constant in hemorrhage, is frequently missing here. If the lumen of a large artery is occluded, sudden loss of consciousness will follow, but in occlusions of small arteries with emboli and in thrombosis of arteries of any size no loss of consciousness will occur.

Whether the loss of consciousness is present or absent, the onset in both cases may be **sudden** or **gradual,** although, speaking generally, in embolism a gradual onset is rare. The symptoms of coma, when the latter is present, are milder and sometimes opposite to those of coma in

hemorrhage (the pulse is not retarded, face is pale, temperature rises immediately). When the onset is gradual prodromal symptoms are **always** present. Twenty-four or forty-eight hours prior to the onset of the paralysis there is a sensation of numbness or slight pain in the extremity or extremities which are to be paralyzed. This is soon followed by a weakness at first in the fingers or toes, which gradually progresses and involves the entire limb. The paretic condition becomes a genuine paralysis at the end of two days. Sometimes collateral circulation intervenes and then amelioration of symptoms is seen instead of complete paralysis. It is therefore advisable to reserve the prognosis for two or three days. The gradual onset of apoplexy, due to a gradual softening, is of course met with in the progressive arterial degeneration of advanced age.

The moderate intensity and durability are not the only characteristic features of apoplectic seizures caused by cerebral softening. When the obstructed blood vessel is cortical, instead of coma epileptiform convulsions set in; they may be focal and limited to the limb or limbs, which will eventually become paralyzed, or else generalized.

Hemiplegia

As mentioned above, paralysis is the final result of apoplectic seizures in hemorrhage, embolism and thrombosis. When the paralysis affects one-half of the body, the condition is **hemiplegia.** The latter is **total** when the face is involved in addition to the arm and leg. The loss of power may be **complete** or **incomplete.** When one limb is affected, the condition is called **monoplegia.**

Two phases should be considered in the course of hemiplegia. One is that of **flaccidity,** the other that of **rigidity** with later contractures. At the onset and a short time after the onset flaccidity is the main feature. When the arm and leg are raised and abandoned, they fall as inert bodies; all movements are abolished. Gradually, however, the power returns and this is noticed earlier in the leg than in the arm, so that in a well-established hemiplegia the arm is usually more involved than the leg. As to the individual muscles, the extensors are usually more affected than the flexors and the abductors more than the adductors. The deep reflexes, which at the beginning were abolished, soon return and become increased: the knee-jerks, the tendo Achillis' reflex, the biceps and triceps reflexes also the wrist reflex of the upper extremities and the jaw-reflex are all very active.

The presence of ankle-clonus during the flaccid phase of the paralysis

is not constant. [This phenomenon when present is elicited in the following manner: the patient's leg is placed in a semi-flexed position on the palmar surface of the left hand of the observer; the latter grasps with his right hand the sole of the foot, extends it and abruptly flexes it on the ankle; rapid clonic movements of the foot follow.] Babinski described a toe phenomenon which may make its appearance even immediately after the onset of hemiplegia. It consists of extension of the great toe or of all the toes when the sole of the foot is gently irritated. This sign is of great diagnostic value, as, according to the generally accepted view, it is present whenever the motor area or the motor pathway in the brain or cord are diseased or only disturbed in their function. There is another sign which was first described by the writer and which is also frequently observed at the onset of hemiplegia. It consists of extension of the great toe or of all the toes when the calf muscles of the leg are deeply pressed upon; by this procedure the flexor muscle-group is brought into action, and since the result is extension instead of flexion I gave it the name "paradoxical flexor reflex." The latter is found in the same conditions as Babinski's sign. Moreover, it was found in a large number of cases of hemiplegia before Babinski's sign made its appearance. (See also page 73.) Oppenheim's sign which consists of extension of the great toe when downward pressure is produced over the extensors of the leg close to the tibia, is also sometimes present in hemiplegia. Mendel observed that percussion of the external aspect of the dorsum of a hemiplegic foot, there is a flexion of the four last toes, but especially of the second and third toes. In normal condition there is extension.

Babinski's toe phenomenon may be also obtained by striking with a percussion hammer the dorsal base of the great toe (Throckmorton) or by a light scratch with a pin below the external malleolus (Chadwick), finally by pinching the tendo Achillis (Shäfer).

During the same flaccid phase of hemiplegia the **face** also shows involvement. The paralysis affects **only the lower half** of the face; the angle of the mouth is lowered, the cheek on the same side is flaccid and is raised at each expiration, the naso-labial fold is smoothened. The asymmetry of the face is particularly noticeable when the patient speaks or laughs. The tongue protrudes toward the paralyzed side. The integrity of the upper branch of the facial nerve can be seen from the normal function of the musculus frontalis and m. orbicularis palpebrarum. The reason of a partial involvement of the face is not very well known; the following possibilities are given by various writers: (1) different course of the fibers of the lower and of the upper portions of the facial nerve; (2) bilateral innervation of the orbicularis palpebrarum muscle

(Broadbent). The neck and the trunk are only very slightly involved because of innervation of their muscles by both hemispheres. Bilaterally associated movements are controlled by the motor cortex of both hemispheres of the brain. This is seen from the inability to move voluntarily one side of the chest without moving the other or one side of the forehead without the other.

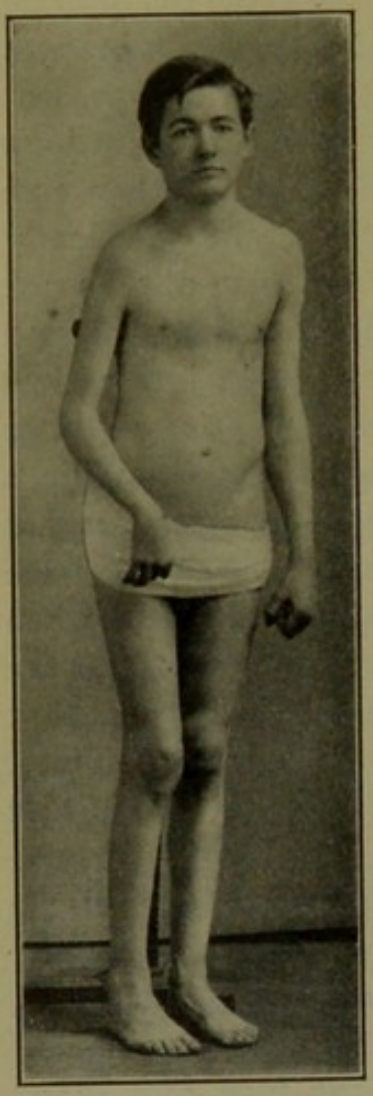


Fig. 64.—Attitude in Hemiplegia showing also the Position of Contractured Left Arm and Leg. Right Hempilegia.

When the symptoms of paralysis do not disappear, the affected portions of the body gradually enter into a phase of **secondary contractures.** It is characterized by an accentuation of all the preceding symptoms. The rigidity of the muscles becomes pronounced, especially in the flexors, and this is seen in the upper as well as in the lower extremities and in the face. Hence deformities of the extremities are frequently observed; they will be various according to whether the flexor or extensor groups of muscles are affected. As to the various reflexes, they become more evident when the contracture of the muscles is moderate, but when the latter is pronounced, all the reflexes may be entirely absent, viz., not obtainable. The **attitude** and **gait** of hemiplegics are quite characteristic. The position of the contractured upper extremity in flexion at the elbow, the scraping of the floor with the paralyzed foot, the bending of the trunk toward the normal side in walking are some of the typical features of hemiplegic persons (Fig. 64).

The opposite side of the body presents also some peculiarities. Exaltation of all deep reflexes, some weakness of muscular power, the presence of Babinski's sign and of the paradoxical flexor reflex and occasionally ankle-clonus—these are the symptoms observed on the "sound" side in some cases of hemiplegia. The reason of it lies in the fact that sometimes the homolateral tract (see Anatomy) is involved and sometimes there is a bilateral lesion in the hemispheres.

In recent years the study of organic hemiplegia became enriched by a number of new symptoms which in doubtful cases may render considerable assistance. They are as follows:

(1) **The sign of the interossei** (Souques). It consists of extension

and abduction in a fan-like form of the fingers, when the patient attempts to raise the paralyzed arm.

(2) **The sign of the thumb** (Klippel and Weil). It consists of involuntary flexion of the thumb when an effort is made to extend slowly the four other fingers which are in a state of contraction in the hemiplegic hand.

Signs (1) and (2) are met with only in cases of hemiplegia with contracture of the upper extremity. The following sign can be demonstrated only in flaccid organic hemiplegia.

(3) **The sign of the hand** (Raimiste). The elbow of the paralyzed arm is on a table. The forearm and hand are both raised and kept in a vertical position by the observer's hand. The latter is gently removed from the patient's hand and slides down on the forearm; the paralyzed hand at once falls and forms an angle with the forearm of 130°. This sign can be detected immediately after an apoplectic stroke and during coma.

(4) **Exaggerated flexion of the forearm.** When the supinated forearm is elevated and flexed over the arm, the degree of flexion is greater than in a normal arm when the movement is persisted in.

(5) **The finger phenomenon** (A. Gordon). It consists of extension of all the fingers, or of the thumb and index, when pressure is produced by the observer's thumb against the pisiform body. This sign is particularly prompt in recent hemiplegias.

(6) Chaddock's **"wrist sign"** consists of flexion of the wrist and simultaneous extension and separation of all the fingers, when scratching with a pointed instrument is done on the ulnar side at the junction of the palm and the wrist.

(7) **Adduction and abduction sign of the leg** (Raimiste). When the patient lying flat on his back is told to bring the sound leg close to the paralyzed one and at the same time the movement is resisted by the observer, the paralyzed leg instead makes an involuntary adduction movement. An abduction of paralyzed limb is noticed when the patient is told to abduct his sound leg and at the same time opposition is made by the observer.

(8) **Hypertonicity of flexors sign** (Néri). When in a lying position the paralyzed leg is raised, the angle it forms with the bed is but 40°, while the angle formed with the sound leg and bed is 70°. When standing patient is told to bend his trunk forward, the sound leg will remain straight, but the paralyzed leg will flex.

(9) **Sign of Grasset and Gaussel.** When a hemiplegic lying on his

back attempts to raise his paralyzed leg, it falls heavily back at the moment the sound leg is elevated by another person.

(10) **Babinski's associated movements.** When lying on his back the hemiplegic attempts to raise himself without support of his hands, the paralyzed leg extends and the entire extremity is raised off the bed.

(11) **Claude's reflex hyperkinesia** is very important from a prognostic standpoint. It consists of voluntary movements of a totally paralyzed limb when it is placed in a forced attitude of pronation or supination for the upper extremity; for the lower extremity when the skin of the external aspect of the thigh is pinched. When this sign is present, the prognosis is very favorable.

(12) **Marie and Foix' retraction sign.** When the toes are forced slowly downward, the thigh flexes over the pelvis, the leg over the thigh, and the foot assumes a position of adduction.

(13) **Anticus leg sign** (Piotrowski). It consists of reflex dorsal flexion together with supination of the foot, when the tibialis anticus muscle is percussed at its point of origin between the tuberosity of the tibia or head of the fibula or else at its belly. It is pathognomonic of organic diseases of the central nervous system, if the reflex response is excessive or when it can be elicited only or is predominant on one side.

Sensory, Motor and Trophic Complications of Hemiplegia

Sensory.—In the majority of cases there are some sensory disturbances on the hemiplegic side. Numbness, tingling or even pain may precede or follow the attack of paralysis, but the most important sensory symptom is **hemianesthesia.** The subject is of great importance, from a diagnostic standpoint, as the same phenomenon may be observed also in hysteria. When the paralyzed side is carefully examined, some diminution of sensations to all forms will almost always be observed. Sometimes the loss of sensations is absolute or in proportion with the loss of motor power. As to the special senses, their involvement has not always been observed. Although the question of disturbed sensations in hemiplegia is not yet entirely settled, nevertheless it is admitted by the majority of writers that while complete hemianesthesia is rare, partial loss of sensations is common. There are also cases in which sensory disturbances are only transitory, and most of the time they develop shortly after the onset of hemiplegia. The characteristic feature of sensory disturbances of cerebral origin (cortical or capsular) is their predominance in the distal ends of the limbs. The existence of partial or complete hemianesthesia on the paralyzed side of hemiplegics finds its explanation in the anatomical

conception of sensory centers. (See Anatomy.) The posterior limb of the internal capsule was according to the old teaching divided into two portions: the anterior two-thirds were exclusively motor and the posterior third exclusively sensory ("carrefour sensitif" of Charcot). We are now in possession of a considerable number of pathological data showing that sensory disturbances have been observed in hemiplegia caused by diseased foci in the anterior two-thirds of the posterior limb of the internal capsule, also motor disturbances as well as sensory in hemiplegia caused by a lesion in the posterior third of the same limb of the capsule. On the other hand there are a few isolated facts which are apt to throw some doubt on this conception of sensory and motor localization. The involvement of special senses was found to be present in hemiplegias of capsular origin, but not of cortical; however their involvement is not as frequent as that of general sensations. Special mention should be made of the **stereognostic sense.** This name is applied to the perception of form and of physical properties of objects by means of touch. In hemiplegia alongside with general sensory disturbances **astereognosis** is also not infrequently observed. The writer had examined at the Philadelphia Hospital thirty-five cases of motor cerebral paralysis and found the stereognostic sense disturbed in twenty-nine cases, in twenty-two of which the loss was complete (*Journal of Nervous and Mental Disease*, 1903).

Motor Complications.—Hemichorea, hemiathetosis and hemiataxia (frequently associated with hemianesthesia) accompany sometimes hemiplegia. In the first the paralyzed arm and leg are animated with choreiform movements. Athetosis affects the fingers and toes and occasionally the muscles of the face. Hemiataxia consists of incoördination of the hemiplegic arm and leg during voluntary movements. The three conditions make their appearance some time after the onset of the paralysis, although they may precede it. According to Raymond, these motor symptoms are due to a lesion in the internal capsule.

Trophic Complications.—Œdema, desquamation of the skin, ulcerations, disturbed nutrition of the nails are not infrequent occurrences in hemiplegias. Glossy skin of the hand is not rare. Among the very frequent trophic disturbances is muscular atrophy of the paralyzed limbs.

The atrophy is diffuse affecting particularly the small muscles of the hands and feet, also the roots of the limbs viz. the humero-scapular and the gluteal-femoral regions. It may develop early, but most frequently long after the onset of hemiplegia. There are usually no reactions of degeneration. In advanced cases the galvanic and faradic contractility is diminished. The atrophy is not due to the misuse caused by protracted impo-

tence, but it is, according to the generally accepted opinion, of central origin; it depends either on a lesion of the cells of the anterior cornua of the spinal cord, or on the original cerebral lesion. The pathogenesis, however, is not completely established. Very exceptionally hypertrophy of the paralyzed muscles is observed. Arthropathies are sometimes observed in the shoulders, fingers, elbow or knee.

Other Complications.—In right-sided hemiplegias there may be a temporary or permanent loss of speech (**aphasia**). The latter may be complete or partial. This subject is of great importance and deserves a special description, which will be given in a special chapter. Impairment of intelligence, change of disposition, modification of character are not infrequently observed some time after the onset of hemiplegia, especially in people of advanced age.

Prognosis.—During the apoplectic attack it is almost impossible to tell whether the patient will recover or not. If the coma is prolonged above twenty-four or forty-eight hours, the outlook is grave. It is equally bad when the bodily temperature goes down or ascends rapidly. It is also serious when the high temperature is continuous. A cardiac or vascular disease renders also the prognosis uncertain, as repetitions of apoplectic attacks are to be feared. Convulsions and early contractures are an indication of a profuse hemorrhage in the ventricles or in the meninges. Cheyne-Stokes respiration is of grave omen. Bed-sores are also an unfavorable sign. The presence of blood in the cerebro-spinal fluid warns of impending danger even if the clinical picture is at first mild. When the patient recovers from the coma, the question is to determine the future of the paralytic symptoms. An incomplete hemiplegia presents a favorable prognosis. However if the paralysis is at first slight, but during the following weeks becomes more pronounced, the chances for regaining the power in the affected limbs are slight. Appearance of contractures makes the prognosis serious. Hemiplegia due to traumatism or syphilis presents the best prognosis, as it is amenable to treatment.

Complications—sensory, motor or mental, also trophic disturbances, convulsions—all make the prognosis unfavorable.

Diagnosis.—Generally speaking, there is no difficulty in diagnosticating an apoplectic seizure. In exceptional cases, however, it may be confounded with **uremic and diabetic coma, syncope, intoxications** (chloroform, alcohol, lead, etc.) and **hysterical** paroxysms, also **hemorrhagic meningitis.** In uremia the onset of the coma is usually slow and preceded by vomiting, dyspnœa, convulsions and sometimes visual disturbances. In diabetic coma there is a difficult and deep breathing followed by abrupt expiration, vomiting, diarrhœa; the breath has the

odor of chloroform and sugar is found in the urine. In syncope consciousness is generally not totally lost, the pulse is small and the attack lasts but a short time. The breath of the patient will reveal an intoxication, although hemorrhage in the brain is not an infrequent occurrence in acute alcoholism. A hysterical paroxysm may simulate an apoplectic attack, but the facial expression showing emotion, the preservation of the corneal reflexes and the facility with which the intensity of the coma is modified will enable one to make the diagnosis of a hysterical attack. Hemorrhagic meningitis accompanied by coma will be distinguished from apoplectic coma by the presence of convulsions, by absence of focal symptoms and rapid fatal termination. Lumbar puncture will assist in determining the etiology of coma. In cases of meningeal involvement lymphocytosis is found; in diabetic coma acetone, in uremic coma excess of urea, in lead intoxication high pressure of cerebro-spinal fluid. In a hysterical paroxysm sudden loss of consciousness may supervene, but the respiration and pulse remain normal; the face is not congested. Besides, it usually occurs after an emotion.

After the diagnosis of apoplexy is established, the main problem consists in determining the **cause** and the **seat** of the lesion.

In the majority of cases it is difficult to tell whether the apoplexy is due to a **hemorrhage** or **softening.** In favor of the first there is usually absence of premonitory symptoms, its occurrence at a comparatively advanced age (above forty), low temperature, profound and prolonged coma, completeness of the paralysis, redness of the face, high tension of the pulse and strong heart beats. A rapid and progressive improvement soon after the onset is also a characteristic feature of hemorrhage. In favor of **softening** there is usually a rise of temperature, absence of coma or mild and not prolonged coma, incompleteness of hemiplegia (usually a monoplegia), finally the presence of cardiac or arterial lesions.

Convulsions at the onset are more frequent in softening than in hemorrhage, especially if they are unilateral and when the cortex is affected. Early convulsions and early contractures are indicative of a hemorrhage in the ventricles or in the meninges (see Meningitis). Aphasia, if it occurs with a mild hemiplegia, is almost always due to a softening. Persistent aphasia is caused by softening. Hemianopsia suggests softening. Repetition of apoplectic attacks occurs in softening. If all these symptoms are not sufficient to differentiate hemorrhage from softening, lumbar puncture will render assistance. In hemorrhage the effused blood passes into the cerebro-spinal fluid (especially in ventricular hemorrhages) and can be detected by lumbar puncture, while in thrombosis this is not the case.

The next step is to determine whether the softening is due to **embolism** or **thrombosis.** A sudden onset without premonitory symptoms in youth and a cardiac lesion are symptoms of embolism. In ulcerative endocarditis the embolism may be followed by rise of temperature (chills and fever). Thrombosis is often preceded by paresthesias in the limbs about to be paralyzed, vertigo, headache, brief impairment of speech; the apoplectic insult comes on gradually: at first only weakness and then a progressive paralysis without loss of consciousness; the hemiplegia is usually incomplete. Atheromatous condition of the blood vessels with high blood pressure and old age are found in softening due to thrombosis.

The above mentioned differential signs may be sufficient to determine the nature of any given case of apoplexy. When the hemiplegia following apoplexy is definitely established and the patient falls under observation some time after the insult, it is important to ascertain whether the paralysis is of organic origin or functional. In **hysterical hemiplegia** the paralysis is usually slow and progressive, is not always confined strictly to the entire half of the body; one limb or a part of a limb is usually involved; the face is most frequently not involved and if asymmetry of the face is occasionally observed, it is due to a spasm but not to a paralysis. The reflexes are normal (no Babinski sign, no ankle-clonus, no paradoxical flexor reflex or any other tendon or cutaneous reflex observed in organic hemiplegia). The course of the disease is irregular: the paralysis may become ameliorated or aggravated, alternate in its intensity, even disappear and reappear; finally the paralysis may remain flaccid for an indefinite period of time. Spasticity does not succeed the early flaccidity which is observed in organic cases. The gait is quite characteristic. In hysterical hemiplegia the patient drags his leg, while in organic hemiplegia the front part of the foot sweeps the ground with each step. In hysteria there is frequently a **hemianesthesia** on the hemiplegic side. The latter, if present, is absolute and more marked than the paralysis, while in organic (capsular or cortical) hemianesthesia the sensory disturbance is not evenly distributed: the extremities are more affected than the trunk and the upper extremities more than the lower. Cerebral hemianesthesia is usually transitory and it may last but a few hours (see Hysteria).

Localization.—When the lesion is in the **cortex,** the paralysis is frequently either in the face and arm, or in arm and leg; it is accompanied by an incomplete or transient hemianesthesia, frequently by aphasia, and follows frequently an attack of Jacksonian epilepsy.

Hemiplegia which accompanies a **meningeal** lesion is rarely complete and presents early contractures and convulsions.

When the lesion is in the **internal capsule,** the hemiplegia is total and is frequently accompanied by hemiathetosis or hemichorea and hemianesthesia.

When the hemiplegia is due to a hemorrhage in the ventricles, convulsions occur early and are generalized; there is a progressive rise of temperature and death is the usual outcome. Hemiplegia caused by lesions in other portions of the brain will be described in their respective chapters.

Treatment.—It was mentioned above that in the majority of cases it is difficult to determine the cause of an apoplectic seizure. In such cases the following measures should be applied. At once the patient must be put to bed, all clothing loosened, especially at the neck, and a free access of fresh air given. He must not be disturbed in the coma. The pulse and heart should be watched and according to their condition stimulants or sedatives will be administered (hypodermatically preferred). With return of consciousness the patient is given a fluid diet, consisting mainly of milk, his bowels and bladder are closely watched and hypostatic congestion of the lung will be avoided by changing frequently his position.

When it is possible to determine at the outset the cause of the apoplexy, the management will somewhat differ in hemorrhage and softening. When **hemorrhage** occurs, the patient's head will be somewhat raised and the body placed on the back or on the non-paralyzed side. As there is usually a flushed face and a full, strong pulse, a bleeding should be resorted to without unnecessary delay. In individuals with a strong heart ten to fifteen ounces of blood can be taken. No venesection should be practiced in cases of cardiac weakness. Lowering of blood pressure can be accomplished not only by venesection but also by lumbar puncture and withdrawal of a moderate quantity of cerebro-spinal fluid. Compression or ligation of the internal carotid artery on the side of the lesion can be used in extreme cases for arresting a hemorrhage after other measures have failed. Puncture of the brain has been advocated (Franke), but this procedure is quite dangerous. Ice applied to the head may relieve congestion. As purging is a valuable procedure, two drops of croton oil should be placed on the back of the tongue. No medication is to be given during the comatose state. With return of consciousness the treatment should be symptomatic. Restlessness and insomnia are combated by bromides, aconite, trional, sulphonal or veronal. Weakness of the heart will be treated with mild stimulants. Great caution should be exercised in using heart stimulants. Coffee, tea and alcohol should be avoided, unless there are special indications. Great care must be taken of the

patient's skin to prevent bed-sores. Bladder and rectum must be emptied, if there are no voluntary evacuations.

In **meningeal hemorrhage of adults** trephining and ligation of the bleeding blood vessel are indicated.

Meningeal hemorrhage in the newly born deserves special mention. On the first suspicion of a hemorrhage a lumbar puncture should be made. If this fails to relieve, the anterior fontanelle should be punctured. Some advocate puncture of the fontanelle at once before doing a lumbar puncture. Giles (*Revue mens. d'Obstetr., Gynéc. et Pediatrie*, 1912) advises introduction of Pravaz needle to a depth of 5 mm. or even 8, as far from the longitudinal sinus as possible in the fronto-parietal angle of the fontanelle. This draws a certain amount of blood at once before it has had the time to clot and the rest is easily absorbed. Puncture of the fontanelle is merely an exploratory operation, but it may have an actual curative value as in a case observed by Giles (*loc. cit.*). It is an excellent preparatory procedure as it gives an opportunity to wait until the brain develops more resisting power before a radical operation is attempted to relieve pressure on the brain.

When the indications are that the apoplectic stroke is due to an **embolism or thrombosis,** the patient's position should be somewhat different from that in hemorrhage: the head must be somewhat lowered or the entire body laid flat. Heart stimulants must be instantly administered, if the weakness of the heart is evident or when the blood pressure is low: ether, camphor and nitroglycerine should be administered hypodermatically; wine, brandy, tea and coffee by the mouth. Application of ice and venesection, which are sometimes beneficial in cerebral hemorrhage, are contra-indicated in softening. The further treatment of softening after the immediate symptoms have subsided will be symptomatic. The condition of the sphincters and of the skin must be carefully watched. The diet must consist mainly of milk, eggs and fruit. Later on, many weeks after the onset, when only hemiplegia is present, meats and vegetables may be added to the above diet. As internal medication iodides are advisable in both softening and hemorrhage, especially in those cases in which there are evidences of specific arteritis. In cases of arteriosclerosis with a high tension pulse, **occasional** administration of nitroglycerine in gr. 1/100 doses two or three times a day is advisable.

The treatment of hemiplegia should begin as soon as the general condition permits it, and this is usually in the second week. Passive and active movements in addition to massage are the only means of which improvement can be expected. Massage is the most important one.

I have seen mild cases which have so much improved from early massage that the paralysis was hardly noticeable. It is a valuable procedure not only in recent but also in old hemiplegias with contractures. The massage must be given as often as possible—every day and even twice a day. It prevents, when instituted early, the rapid tendency to contractures by improving the nutrition of the muscles and their tendons; it prevents ankylosis of the joints and removes the paresthesias which so frequently accompany the paralysis. Systematic re-education of movements of the affected limbs is important; the patient must persist in exercising them frequently. Contractures may also be treated by warm baths followed by massage and passive movements. Electricity, which is so frequently employed in organic nervous affections, should be avoided in organic hemiplegias, as it is apt to hasten or increase the contracture of the muscles. For the same reason the drug which has a tendency to increase the tonicity of the muscles should never be given in hemiplegia —and this is strychnia. To combat the rigidity of the limbs in paralysis of cerebral origin Foerster has devised an operation on the spinal roots. It will be discussed in the chapter on Diplegia. See also "muscle isolation" method of Schwab and Allison, also that of Stoffel (pages 122 and 123).

The **complications** of hemiplegia do not require any special treatment. The hemiathetosis, hemichorea and hemiataxia may improve with the amelioration of the hemiplegia itself. The aphasia which occasionally occurs depends upon the gravity of the cerebral lesion. However attempts of reëducation of speech have been tried by some and fair results reported. No special rules can be given for the treatment of the mental condition in hemiplegia.

INGRAVESCENT OR PROGRESSIVE APOPLEXY

It is characterized by sudden vertigo and headache, rapidly followed by a hemiplegia. There is no loss of consciousness. Gradually the patient becomes somnolent, stuporous, comatose and dies at the end of a few days. The lesion consists of a hemorrhage caused by rupture of the external lenticular artery. The blood spreads forward and backward and finally invades the lateral ventricle.

DELAYED APOPLEXY

Spätapoplexie

Under this name is understood an attack of apoplexy with loss of consciousness followed by hemiplegia. It occurs some time (but not

immediately) after trauma. It was first described by Bolinger in 1891. Cases have been reported in which the interval between the traumatism and the stroke lasts from six days to four weeks. Autopsy showed in the majority of cases a cerebral hemorrhage. Trousseau has shown that a rupture of a blood vessel may occur some time after a concussion by means of a thrombosis of capillaries; a focus of softening is thus formed in the center of which a secondary hemorrhage takes place. In the cases of delayed apoplexy the blood vessels were found diseased (arteriosclerosis). The trauma therefore plays only an exciting rôle in an individual with an already altered vascular system. It is well to remember that nervous disorders occurring some time after an accident are not always functional; they may be also organic and present a grave prognosis.

INTERMITTENT CLOSING OF CEREBRAL ARTERIES AND TRANSITORY HEMIPLEGIA

Besides hemorrhage, embolism and thrombosis as causes of hemiplegia, a sudden closure of a cerebral blood vessel due to a spasm may also produce a paralysis. Bastian was the first to call attention to this condition in 1875. Not infrequently cases come under observation in which pa ralysis comes on suddenly and disappears rapidly. Sometimes an attack consists only of sensations of tingling or numbness in arm or leg or in both, which may or may not be followed by paralysis or paresis on the same side. Sometimes it may be only an attack of aphasia with or without hemiplegia. The chief characteristic of these attacks is their suddenness of onset and of disappearance, also their reappearance at short or long intervals. Besides, there is no loss of consciousness in the majority of cases.

The spasm of the artery may produce either its complete or incomplete occlusion. As a result there is either suspension or impairment of function. The cause of the spasm lies probably in the disturbance of the vaso-motor nerve supply of the arteries. The existence of vaso-constrictors has been shown by C. F. Wiggers (*Amer. J. of Phys.*, 1905, 1907, 1908). Complete recovery after each of these attacks proves that the hemiparesis or hemiplegia is not due to hemorrhage or softening, but to an irregular vessel action and therefore to an irregular blood flow in the brain. If by proper hygienic measures the irritability of the vascular tonus can be prevented, no further attacks will occur. Otherwise it facilitates formation of thrombosis. Recurrent attacks may be considered as threatening or premonitory signs of eventual cerebral hemorrhage or thrombosis.

The treament of such cases consists of internal administration of vasodilators (nitroglycerine), avoidance of emotion, of proper elimination (bowels and kidneys), avoidance of red meats. As the disorder usually occurs in elderly persons showing evidences of arterial changes, prolonged rest and potassium or sodium iodide should be added to the above treatment.

CHAPTER V

INFLAMMATION OF THE BRAIN (ENCEPHALITIS, CEREBRITIS). ACUTE AND CHRONIC

A. ACUTE ENCEPHALITIS

A PRIMARY acute inflammation of the brain tissue is a rare affection. It is usually a circumscribed lesion and almost always leads to suppuration. **Trauma** is considered as one of the causes, but in such cases the meninges as well as the brain tissue are commonly involved, although in rare cases the former escape. The most frequent cause of primary encephalitis is **infection.** Although the organism is not known, nevertheless recent experimental evidences indicate resemblance of acute encephalitis to poliomyelitis. Both are transmissible diseases and probably constitute acute specific diseases. In the course of infectious diseases, as diphtheria, erysipelas, scarlet fever, measles, acute tuberculosis, pneumonia, ulcerative endocarditis and whooping cough, encephalitis may develop. In these cases the inflammation may and may not terminate in suppuration. Cases in which there is always formation of abscess are those with a history of **caries** of petrous bone, of frontal sinuses and especially with **otitis media.** The term "acute hemorrhagic encephalitis" is applied to those forms of encephalitis which do not end in suppuration. The two forms deserve special descriptions.

(a) Non-suppurative Form

This variety of encephalitis affects the cerebrum more frequently than the cerebellum. Strümpell in 1884 was the first to call attention to a primary cortical cerebritis (**polio-encephalitis**) in the motor area. He observed it in children, and acute cerebral palsy was due, in his opinion, to this cause. A similar condition may also occur in the pons, in the gray substance around the aqueduct of Sylvius and is called then "polio-encephalitis superior of Wernicke," to be distinguished from "polio-encephalitis inferior" or acute bulbar paralysis.

Pathology.—Irrespective of the seat the lesion is iedntical in both cases. The affected area appears swollen and markedly red; its consistency is lessened. The blood vessels, especially the capillaries, are dis-

tended, foci of hemorrhage are abundant, nervous tissue and the blood vessels are infiltrated with leucocytes. The brain tissue—fibers, ganglion-cells and neuroglia—undergoes degeneration. When the nerve tissue is totally disintegrated, it is eventually absorbed; walls will form around the remaining cavity and a cyst will be the result; the latter may become contracted and a cicatrix develops. Restitution of the affected nerve tissue is rare.

Symptoms.—The clinical manifestations of the non-suppurative variety of encephalitis affecting the floor of the aqueduct of Sylvius will not be discussed here. In the chapter on diseases of the medulla this subject will be taken up in detail.

The symptoms of encephalitis appear acutely. The onset resembles that of acute infection. Sudden rise of temperature, chills, headache, sometimes vomiting and convulsions are the usual prodromal signs. Some rigidity of the neck muscles is common. Loss of consciousness or only incomplete coma with delirium, Cheyne-Stokes' respiration and rapid pulse are the actual general symptoms of the affection. The localized symptoms will depend upon the seat of the inflammation (see chapter on cerebral localizations), and as in the majority of cases the cortex of the motor area is involved, hemiplegia or monoplegia with local convulsions with or without aphasia are the most frequent occurrences. Acute encephalitis attacks children oftener than adults. For this reason mental arrest is observed in young individuals who had once an acute attack of cerebritis.

Prognosis.—During the comatose state the prognosis is usually unfavorable. Death may occur in twenty-four hours. The severity of the prodromal symptoms and the degree of the coma generally determine the outlook in a given case. After the acute symptoms subside, the prognosis becomes more favorable. Recovery without some defect is rare: focal epilepsy, impaired speech, some paralytic condition of one or two limbs on the same side of the body are the most frequent sequelæ of acute encephalitis.

Diagnosis.—Meningitis and cerebral hemorrhage are the two affections with which acute encephalitis may be confounded. When focal symptoms, such as described above, make their appearance at the onset and become rapidly more and more pronounced, encephalitis should be suspected rather than a localized meningitis. For the differential diagnosis with cerebral hemorrhage see chapter on Apoplexy.

Treatment.—During the acute stage application of ice to the head and bleeding, in addition to absolute rest, are practically the only measures that could be recommended. If the fever is very high antipyretics

should be used. The treatment of the chronic stage is identical with that of apoplexy.

(b) Suppurative Form. Abscess of the Brain

Etiology.—As mentioned above, the most constant cause of acute encephalitis terminating in suppuration is a lesion of the bony walls of the cranium. Trauma of the scalp, of the meninges and of the brain tissue may also be followed by formation of an abscess. In connection with this factor it should be borne in mind that in some cases abscesses of the brain were observed long after the occurrence of the trauma, also that in a certain group of cases the trauma of the head was slight and still suppuration developed in the brain. A metastatic embolus originating in some purulent focus of the body, like cellulitis, abscess of the liver, purulent processes in the bronchi or lungs, or other viscera, is another cause of abscess of the brain. Suppuration of the mucous membranes lining the cavities of the cranium (nose and accessory cavities) is the third cause of abscess. Finally the most important and, according to some observers, the most frequent cause of abscess of the brain is **Otitis media** and especially its chronic form. Small veins and lymphatics pass from the tympanum to the superior petrosal sinus; similar vessels end in the same sinus from the temporo-sphenoidal lobe of the brain. Identical vessels come from the lateral lobe of the cerebellum and from the mastoid region and all end in the lateral sinus. The channels of infection are thus explained. Diseases of the ear are apt therefore to produce an abscess in the temporo-sphenoidal lobe and in the lateral lobe of the cerebellum.

In cases of abscess caused by diseases of the nasal cavity the abscess is situated in the frontal lobe. The infection is carried through the venous and lymphatic channels or directly from the carious bone.

Whatever the original cause may be, suppuration in the brain necessitates the presence of multiple microörganisms, among which the streptococcus plays the most important rôle. An interesting and rare variety is the cerebral suppuration in which the tubercle bacillus alone was found (Fraenkel, Rendu and Boulloche).

The infection may be transmitted to the brain tissue either through the venous sinuses, which are then in a state of inflammation, or through the arterial system. The latter will occur in those cases in which the initial purulent focus is far away from the brain; the former will take place when the suppuration is in proximity of the brain.

As to the seat of abscess, it is most frequent in the hemispheres, rarely in the basal ganglia; the metastatic form is in the area of distribution

of the middle meningeal artery; the otitic form has its seat in the temporal lobe or cerebellum; the traumatic form is usually confined to the injured region of the cranium. The average age at which cerebral abscess is met with is between twenty-five and forty; it is rare above sixty. Males are more frequently affected than females.

Pathology.—There are two principal conditions to be considered: one in which there is a purulent infiltration with subsequent softening of the involved cerebral tissue, the other is characterized by formation of an abscess which becomes encapsulated. In the latter case the membrane surrounding the purulent cavity is organized by the neuroglia tissue; its formation commences on the fifteenth day. If the capsule is complete, it serves as a protection for the surrounding nervous tissue against invasion of the pus, but not infrequently fistulous channels are

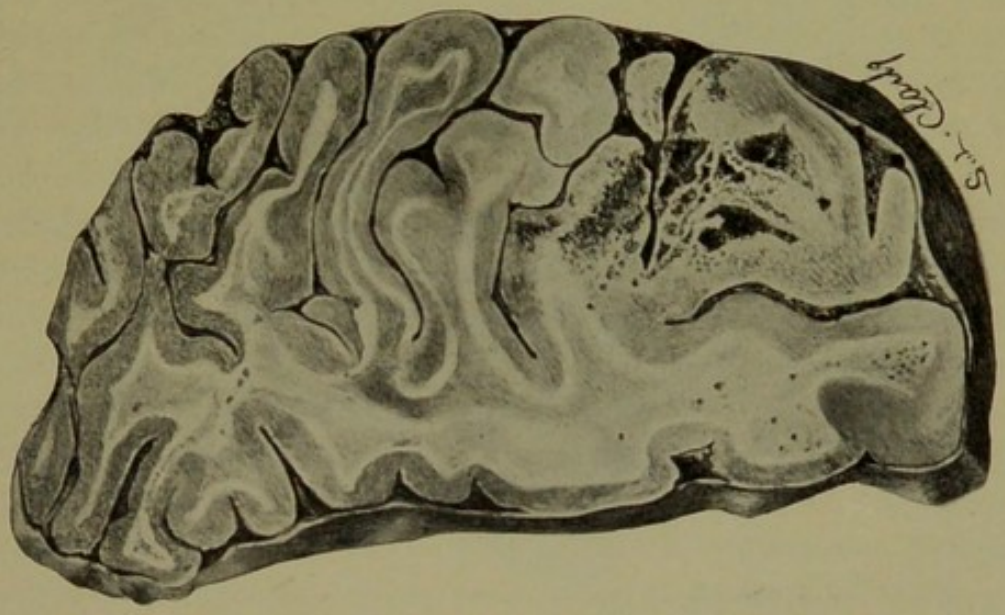


FIG. 65.—HEMORRHAGE IN THE PREFRONTAL LOBE FOLLOWING TRAUMA. (*Original.*)

to be found and formation of pus continues indefinitely. As to the capsule itself, it is thin at the beginning; later it grows thick and calcification may also occur. While it is thin and delicate and the pus continues to accumulate, it is apt to rupture. When the latter occurs on the surface of the brain a purulent meningitis will be the result. When the pus breaks through the capsule into the ventricles sudden death may follow.

Single encapsulated abscesses are met with in the majority of cases, but multiple abscesses may also occur, especially in the metastatic form and in general pyemia. The brain tissue in both cases, namely in **purulent infiltration** and **abscess,** undergoes at first a so-called "red softening" in which disintegrated nerve elements, leucocytes and microörganisms are found. The leucocytes become gradually but rapidly more abundant.

They surround the blood vessels, and cover the nerve tissue, which is in a state of inflammatory œdema. Cells and nerve fibers are gradually destroyed and replaced by pus. Softening of cerebral tissue is observed also in the immediate neighborhood around an encapsulated abscess (Figs. 65 and 66). Cerebral abscesses are more frequent than cerebellar. In the brain abscesses are mostly found in the temporo-sphenoidal lobe. In the cerebellum in its lateral lobe. Abscesses are rare at the base of the brain.

Symptoms.—The clinical manifestations are **general** and **local.**

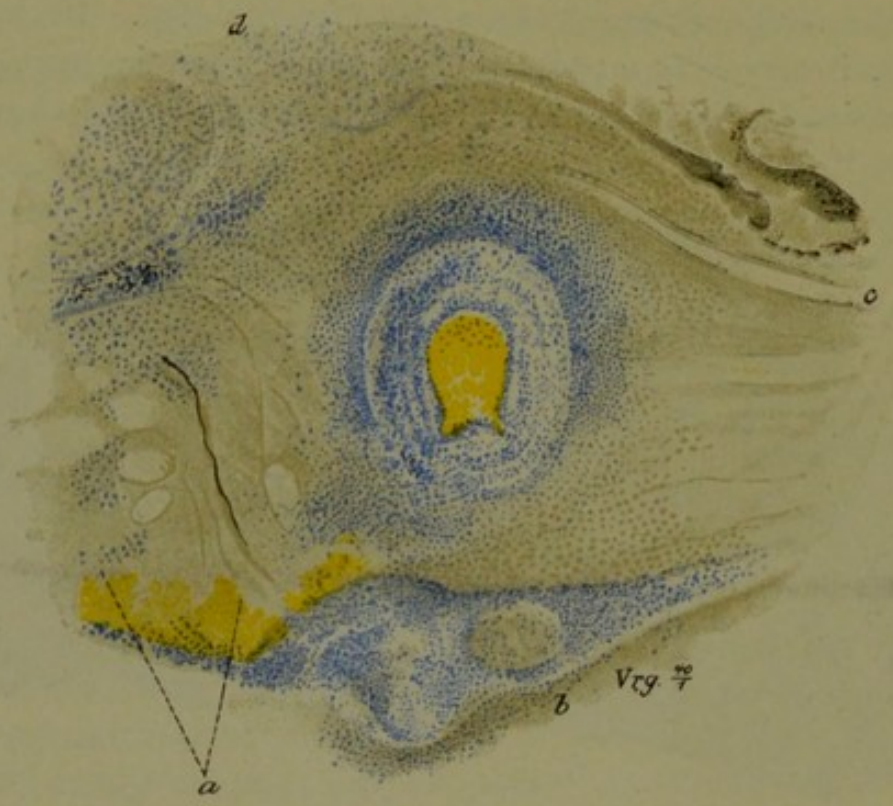


FIG. 66.—HEMORRHAGIC ENCEPHALITIS IN CORTEX. (*From Flatau, Jacobsohn, Minor.*) In center a blood vessel with round-cell infiltration. *d*. Focus of round cells. *c*. Amorphous exudate. *a*. Fresh blood. *b*. Mass of round cells.

(a) **General.**—At the onset the symptoms are those of inflammation. **Fever** is not pronounced, but what is often observed is the lack of parallelism between the pulse and fever; while the latter is high, the pulse may be slow. The fever is not infrequently accompanied by **delirium** and occasionally by **generalized convulsions** especially when meningitis or sinus phlebitis are associated. In the majority of cases there is no fever; the temperature is either normal or sub-normal. The pulse is usually slow; rapidity of pulse indicates either a complication of sinus phlebitis or rupture of the abscess into the lateral ventricles. **Headache** is among the earliest symptoms, and when it is continuous and

accompanied by fever it becomes pathognomonic of acute encephalitis. The localized character of the pain in the head and its exacerbations on the slightest movement make it quite typical of acute suppurative encephalitis. In cerebellar abscess the pain is mostly in the occipital region. **Vomiting** is quite common, especially in cerebellar abscess. **Insomnia** and **restlessness** are frequent. Rigidity of the neck, photophobia and ocular palsies are sometimes observed. **Optic neuritis,** although not constant, has been seen even early in the acute stage. Mental hebetude and apathy are conspicuous.

Leucocytosis is frequent in brain abscess.

(b) **Local Symptoms.**—They are mainly paralysis and focal epileptiform convulsions. The latter are less common than the first. Hemiplegia or monoplegia are usually marked, but may be also absolute; they generally follow a unilateral convulsion. As an abscess may occur in any part of the brain, the special symptoms will depend upon its seat and are therefore similar to those of tumors. Thus motor and sensory aphasia, blindness, hemianopsia, etc., may be observed. They will be described in the chapter on tumors of the brain.

Special mention deserves **cerebellar** abscess, which is not an infrequent occurrence in otitic cases. **Vertigo** and **staggering gait** ard particularly pronounced. The tendency to fall is usually toward the side of the abscess. As the cerebellum forms partly the roof of the fourth ventricle, the abscess may produce pressure on the medulla and cause bulbar symptoms.

Course of the Disease.—The onset of acute suppurative encephalitis is very much similar to that of tubercular meningitis in children. In the large majority of cases the clinical picture is as follows. Fever is the first symptom to appear. Soon violent headache sets in. The patient becomes restless, agitated, cannot sleep, refuses to take food, sometimes has attacks of vomiting. As the disease advances, he becomes delirious, is seized with generalized convulsions; the neck is rigid; ocular palsies make their appearance. Then the patient enters into a second state, in which all the symptoms become ameliorated. This condition, however, does not last any marked length of time, as it is soon followed by a state of apoplexy. Here the coma may be so profound that the patient will not regain consciousness and die. If he does recover, rigidity and paralysis will be present on one side of the body. The further evolution of the disease will depend upon the anatomical course of the abscess. If the abscess becomes diffused or breaks into the ventricles, death will ensue. Some cases of acute abscess run such a rapid course that fatal termination may take place in a very short time. In the course of some cases of cerebral abscess associated with otorrhea the discharge may repeat-

edly stop. At each stoppage there is a marked aggravation of headache. It is due to increase of intracranial pressure. The reestablishment of otorrhea relieves the symptoms. There is a series of cases with a history of trauma or of an old otitis, in which the abscess remains latent for months and even years without being suspected, when death takes place either from rupture of the abscess or from acutely developed morbid processes in or in the vicinity of the abscess. This is the so-called **"latent form."** To the same group belongs those cases in which the abscess is seated in the depth of the frontal lobe or in the postero-external portion of the occipital lobe. In such a case there are very few symptoms during life; sudden death is the usual result. However the "latent form" may in some cases end gradually: an intercurrent acute meningitis may aggravate the few existing symptoms which continue then to increase until death.

Prognosis.—Generally speaking the outlook is grave. The difficulty of making a diagnosis, especially in the "latent" cases, the difficulty of localizing, the damage produced in the brain tissue by the abscess itself or by operative procedures are the reasons why abscess of the brain bears a serious prognosis. However there is quite a considerable number of observations showing that immediate death can be avoided by prompt surgical intervention. Complete recoveries have been also reported by some writers, especially in uncomplicated cases and in those that were operated upon early. In chronic or subacute cases the prognosis is more favorable than in the acute ones. In the latent cases the abscess may remain encapsulated for a long time without producing serious disturbances, but the patient is always threatened with complications, such as meningitis and œdema of the brain.

Diagnosis.—When a history of trauma is given, the diagnosis of abscess is not difficult. It is also comparatively easy to diagnosticate abscess, when in the course of otitis media a localized palsy affecting one or two extremities makes its appearance. In all other cases, especially in the "latent forms" the diagnosis is extremely difficult and can be made only by exclusion. Ear, nose, orbit and other parts of the skull should be examined in every case. A septic otorrhea and a small perforation of the tympanum are in favor of a cerebral abscess; when the opening of the tympanic membrane is large, the pus will have a free exit. Absence of perforation also speaks for a brain abscess.

When there is no external evidence of injury or of bone and ear disease, metastasis should be thought of. Heart, lung and other viscera must be carefully examined. A cerebral **tumor** may give identical symptoms, but its course is uniformly progressive and by far more prolonged than

that of abscess. Moreover convulsions, if they do occur, are more frequent and more regular than in abscess. Headache, which is of great importance in abscess, is more progressive, severer and longer in duration in tumors. Involvement of the cranial nerves and especially double optic neuritis and atrophy are common in tumor and exceptionally rare in abscess. A unilateral optic neuritis is in favor of abscess. Sudden appearance of symptoms of meningitis or of sinus phlebitis is an indication of abscess. Finally a history of ear disease with a long course of brain symptoms points to an abscess. Leucocytosis is also in favor of abscess.

Meningitis can be confounded in certain cases with abscess, but the rapid onset, involvement of cranial nerves, high temperature, rapid and irregular pulse are all symptoms rarely found in abscess. Lymphocytosis and a cloudy appearance of the cerebro-spinal fluid are characteristic of meningitis. In children tuberculous meningitis may be confounded with cerebral abscess. Rigidity of the neck, attacks of focal epilepsy or of paralysis, the elevation of temperature and the above condition of the cerebro-spinal fluid are in favor of tuberculous meningitis.

Traumatism of the cranium is liable to develop a **hemorrhagic pachymeningitis.** The distinguishing symptoms between this form and abscess consist of the character of pain, which is superficial and localized and not augmented by motion in pachymeningitis, and of the fever, which is only transitory in the latter affection.

Sinus phlebitis will be recognized by high temperature, rapid pulse, chills, sweating and particularly by tenderness and swelling over the origin of the internal jugular vein.

In cases of suspected abscess with **otitis** it is important to know whether the abscess is in the temporo-sphenoidal lobe or in the cerebellum. Pronounced occipital headache with other symptoms characteristic of cerebellar diseases will enable one to make the differential diagnosis.

Treatment.—As soon as strong suspicions of abscess are present, prompt surgical intervention should be the only treatment. In a number of cases it is difficult to make a diagnosis. In the obscure cases, in which but a few symptoms are evident, abscess should be thought of and the patient be treated by other means only a short time. Counter-irritation and local bleeding, application of ice to the painful region, purgation, sedatives to allay excitement, stimulants to combat depression are the usual means employed in similar cases. The medical treatment should be kept up for a very brief period, and if there is no marked improvement, an operation must be promptly resorted to.

Great attention should always be paid to cranial injuries, viz. guard against infection, as the treatment may be very efficacious in such cases

before an abscess is formed. Otitis media should never be neglected, no matter how slight it may be. Statistics show that early surgical intervention gave a large percentage of recoveries. When the abscess is due to diseases of the ear, the operation must be performed over the area corresponding to the temporo-sphenoidal lobe or to the cerebellum. In the latter case there is usually an occipital headache, also rigidity of the neck and retraction of the head. Besides, the patient will present the typical symptoms of a cerebellar disease, viz. asynergia, adiadochokinesia, hypotonia, etc., etc. (see Cerebellar Diseases). The temporo-sphenoidal lobe is considered as a silent region and there are usually no localizing symptoms, but if the abscess encroaches upon the first and second temporal lobes, there will be in addition to the general symptoms of cerebral abscess also word-deafness. For localizing symptoms of abscess of other portions of the brain, see Cerebral localizations. The surgeon will therefore be guided by the above considerations. In the traumatic cases a careful examination of the parts injured will have to be made.

B. CHRONIC ENCEPHALITIS

Chronic inflammation of the brain **in adults** is very rare as a primary affection. It is encountered in isolated foci in the vicinity of tumors, in disseminate sclerosis, in old syphilitic cases. It is present in paresis in a diffuse form in association with meningitis (meningo-encephalitis).

In children chronic encephalitis presents special features which are so constant and characteristic that they form an important chapter in Nosology and known under the name of *Encephalopathies*.

Chronic inflammation of the brain tissue may be congenital or acquired in childhood. There are several forms of infantile affections which are due to a chronic encephalitis in intrauterine life or developed early in life. The existence of this condition, although well known before Charcot, was ignored until Strümpell called attention to his **polio-encephalitis of infancy.**

It has been observed that in the fœtus in utero or during labor or shortly after birth traumatic encephalitis, hemorrhagic foci, softening may occur and they may lead to cerebral sclerosis, to diffuse meningitis, to porencephaly, to atrophic condition of one or more lobes. In 1834 Lallemand had shown that various forms of malformation of the brain (agenesis) were the result of an encephalitis. This view was corroborated by Charcot and especially by Bourneville, who perhaps more than any one else had the opportunity to deal with idiots and defective children of all kinds and verify the clinically observed symptoms on the post-mortem tables. There are two special forms which will be considered here, viz. **spastic infantile hemiplegia** and **Little's disease** (Diplegia, Paraplegia).

Etiology.—Traumata of the pregnant uterus, trauma of the fœtus during a protracted labor with or without application of forceps not infrequently lead to meningeal hemorrhages over the motor area and eventually to changes in the cortex. Experience teaches that the abnormal conditions which cause long and difficult labor are more apt to produce birth paralysis than a proper application of instruments. Little lays special emphasis on **dystocia** and especially on **premature birth,** which is a proof that the condition may occur before birth. Strümpell observed that **infectious diseases** are the special cause of encephalitis in extra-uterine life. The hemiplegic form is more frequently acquired than congenital. It occurs usually in the course of measles, scarlet fever, typhoid, diphtheria, whooping cough, mumps and finally, although very rarely, in cases of endocarditis. Whether it is due to the infectious element itself or to an embolism or venous thrombosis (Gowers) or to a hemorrhagic influence in the motor area, it is difficult to say. As to **predisposing** causes, **syphilis** (Fournier) and **alcoholism** may be mentioned. Bourneville's researches show that parental alcoholism showed its effect in 41 per cent. of cases. Finally, chronic metallic **intoxications,** as lead, mercury, phosphorus, have also been mentioned as causes of infantile encephalopathies. The present tendency is to find the predominant etiologic rôle in infections which have its effect not only on the fœtus during pregnancy but also on the character of the labor. In case of Little's disease which were apparently due to abnormal birth, lesions have been found which existed prior to birth. Syphilis for example has been observed with premature labor or in still-born children.

Meningeal hemorrhages during birth deserve special mention. Not only traumatism during labor, but also non-traumatic cases are met with in which spontaneous hemorrhages may occur in the fœtus. The latter are due to a special fragility of the blood-vessels, especially the veins, fragility which is due to hereditary syphilis, alcohol, lead intoxication or to debility from any cause. The largest of the sub-arachnoid veins, which are usually ruptured, are located along the anterior and posterior borders of the parietal bone, which correspond to the lambdoid and coronary sutures. The coronary suture corresponds to Rolandic area and consequently a hemorrhage at this level produces motor symptoms, which is ordinarily the case. They are hemiplegia, monoplegia, tremor, convulsions. If convulsions do not disappear, eventually the following manifestations will be observed: arrested mental development, Little's disease, disturbance of vision, ocular palsies, deafness, various malformations in the limbs, disturbances of speech. It is therefore evident that the recognition of meningeal hemorrhages in newly born infants is a matter of

great importance. As soon as they are recognized, operative procedures become urgent (see Treatment).

Pathology.—Various conditions may be found in the brain. Foci of softening or hemorrhage do not differ to any marked extent from those of adult brains; the only pecularity lies in the retraction of brain tissue around the old morbid foci. Evidences of chronic meningitis are frequently seen with the naked eye. The retracted nervous tissue forms a depression, is yellow in color (yellow plaques) and is particularly noticeable in the motor area. The thickened dura is adherent to the cranium, the thickened pia is adherent to the convolutions, from which it cannot be detached. Sometimes small cysts are seen; they are the result of old hemorrhagic foci. The cortex itself undergoes changes: cells and nerve fibers are in a state of degeneration (see pathology in Apoplexy). Briefly speaking, the condition is one of a **meningo-encpehalitis.**

Very probably in many cases of congenital paralysis intra-cranial hemorrhage is the chief cause. H. Cushing (*Amer. J. Med. Sc.*, 1905) operated on a series of such cases immediately after birth and found the above condition; he removed the clots and some of the children continued to develop normally.

Old lesions may lead to a cerebral peculiarity named **"porencephaly."** It is characterized by a cavity which opens on the surface of the brain. The depth of the cavity is various: it may involve only a small portion of the brain tissue or communicate with the lateral ventricles. These cavities are usually bilateral and they are found mostly in the motor area, third frontal and first temporal convolutions. It is remarkable that they have always been observed in areas having a well defined arterial supply. This led to the view that a vascular influence is the cause of porencephaly: a hemorrhage or softening will cause a destruction with a subsequent cavity; in this case the cavity does not reach the ventricles. The communicating cavities may be also the result of a congenital deficiency of the blood supply in a certain area of the brain. An old lesion in the form of an inflammation, limited to the walls of the lateral ventricles, involving the ependyma (ependymitis) leads to another peculiarity, viz. **internal hydrocephalus.** It is characterized by a considerable accumulation of fluid in the ventricles. The cerebral tissue, which is in immediate contact with the walls of the ventricle, is destroyed, and the convolutions being under continuous pressure, become atrophied. **Atrophy of brain** tissue was also observed in a number of cases as a result of a primary **sclerosis** of the brain. This condition usually affects one hemisphere. The convolutions are found thin, retracted, indurated, lighter in color than normal cerebral tissue. If only one hemisphere is affected, the difference

between the two halves of the brain is striking. It may happen that only one or several lobes of the same hemisphere are affected (**microgyria**). The microsopical lesion consists of a pronounced proliferation of neuroglia, also of thickening of the walls of the capillaries and multiplication of the latter. The retraction of the newly formed fibrous tissue leads to a dilatation of the perivascular spaces. These changes are present in the gray as well as in the white matter, more marked in the former than in the latter: the cells gradually change their form and finally disappear. Instead of atrophic sclerosis the brain presents sometimes a **hypertrophy** of some of its portions. As to the cause of cerebral sclerosis, it is generally admitted after Virchow to be due to a **congenital chronic encephalitis,** in which all the elements of the cerebral tissue are affected.

The above described morbid conditions developed in the fœtus or early in life present a special feature which makes them so different from similar states in adults that special mention should be made. In the chapter on apoplexy we saw that a lesion in the brain inevitably leads to secondary descending degenerations in the pyramidal tract. In the fœtus or in early infancy the pyramidal tract is not yet completely developed, as the myelin covering the axis-cylinders begins to appear only during the first months after birth. It stands to reason that a lesion in the brain at those periods of life leads to an arrest of development and **atrophy** of the portions of the nervous tissue beneath the initial lesion. Atrophy and diminution in size of nervous tissue (brain and cord) are therefore characteristic of the lesions in question.

Symptoms. A. **Infantile Spastic Hemiplegia.**—The symptoms of spastic paralysis may be noticed at birth or only when the child makes the first attempt to walk. In some cases the onset of paralysis is preceded by a group of acute symptoms: fever, restlessness, vomiting. Convulsions appear early if not first; they are epileptiform in character and confined at first to the side which is to become paralyzed. The spasms soon become generalized and increase in frequency. During these epileptic attacks, which usually last about forty-eight hours, the hemiplegia suddenly sets in. It is at first flaccid, affects leg, arm and face; the distal ends of the extremities are the most affected and the upper extremity more than the lower. At the end of ten or fifteen days spasticity gradually develops, all the reflexes become exaggerated, ankle-clonus, Babinski's sign and paradoxical reflex are easily elicted (see Apoplexy). The hemiplegia is established and remains permanent (Fig. 67). While in a general way it is similar to hemiplegia of adults, it nevertheless presents these peculiarities that the contractures with the subsequent deformities are very marked: the flexion of the hand and

fingers, the varus equinus are pronounced to a great extent. The striking and distinguishing feature of infantile spastic hemiplegia is the **atrophy** of the entire affected side: skin, fat, connective tissue, musculature and bone participate in the atrophic process; not only the limbs, but also the face and thorax show diminution in size. Deformity of the skeleton, particularly scoliosis, is observed. The atrophy is more marked in the upper extremity than in the lower. Vasomotor disturbances, as well as sensory, are present: the skin is cold, the sensations are diminished. Astereognosis (see page 69) is always present in view of the arrested development of the hand at an early age.

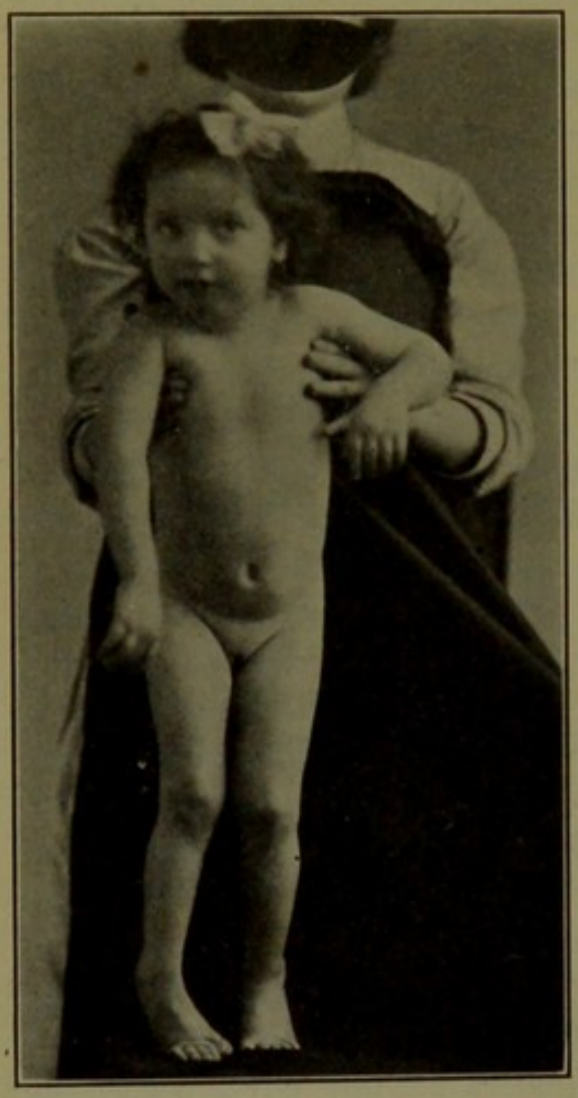


Fig. 67.—R. Infantile Hemiplegia.

The paralyzed limbs are not infrequently affected with **athetosis** or **choreic** movements (hemiathetosis and hemichorea). In these cases the spasticity is not pronounced. Occasionally both varieties of muscular movements are combined. Sometimes the athetosis is bilateral. In some cases there is an intention tremor, such as is seen in disseminated sclerosis. Athetosis, chorea, tremor may vary: they may change in intensity at a certain time of the day or they may substitute each other. They totally disappear during sleep.

Among other complications of infantile spastic hemiplegia may be mentioned **Aphasia,** but the latter is an exceptional occurrence. More frequently there is a delayed development of speech faculty, which begins to develop only at the age of four or five or even later, or after the speech has developed it remains deficient for a long time and even permanently. True aphasia is not present. The intellectual faculties are quite frequently affected in hemiplegia, but more frequently when the frontal lobe is congenitally diseased than the Rolandic area. In view of the tender age of the child there is a retardation of mental development in almost every case. Imbecility and idiocy are observed in some cases.

Children born hemiplegic sometimes present defects in the sphere

of the **special senses,** as congenital strabismus, hemianopsia, nystagmus, deafness.

Epilepsy is quite a frequent complication. It may exist from early infancy or make its appearance later, especially around puberty. The convulsions may be confined to the paralyzed side or be generalized. In the latter case there is usually loss of consciousness with biting of the tongue, frothing at the mouth and other symptoms characteristic of essential epilepsy (see Epilepsy). In many cases the epilepsy has a tendency to decrease with age, but in others remains permanently. Petit mal attacks occur usually in cases in which the paralysis is slight.

B. **Spastic Diplegia. Little's Disease.**

In the chapter on Pathology one could see the various morbid congenital conditions which are apt to create the paralytic states observed during life. When the seat of the lesion, instead of being unilateral, is symmetrically distributed to the **motor areas of both hemispheres,** the clinical manifestation will be a **double hemiplegia** or **diplegia.** When the lesion affects only both **paracentral** lobules a double paralysis of both lower extremities will be observed (**paraplegia**). These are the two clinical varieties usually met with in practice. On the other hand, if during a protracted and difficult labor or after birth hemorrhages should occur in the brain or in the spinal cord, a double hemiplegia (**diplegia**) or paraplegia (palsy of both lower extremities) may equally occur (Figs. 68 and 69). Spastic diplegia or paraplegia may therefore be observed in two different conditions: **congenital** and **acquired.** One of the earliest writers who called attention to the first variety was Little, who in 1862 gave the best description. He pointed out as the most important etiological moment of the affection **premature labor.** He observed that some children born prematurely presented early symptoms of spastic diplegia. It is therefore natural to suppose that in those cases a deficient development of the **pyramidal tract** (agenesis) was the direct cause of the malady. It is reasonable to presume in the light of our present knowledge especially with reference to Wasserman reaction that premature labor and Little's diseases are the consequence of the same cause, viz. hereditary syphilis. In a very large number of cases of diplegic Wasserman test has been found to be positive. At present the writers are divided: some describe under the name of Little's disease both the congenital and the acquired forms, some make a sharp distinction and consider three characteristic features necessary for Little's disease, viz. (1) the affection must be congenital, (2) of cerebral origin, and (3) due to agenesis of the pyramidal tract. The pyramidal fibers become covered with myelin only during the first few months after normal birth.

According to Van Gehuchten, at seven months the pyramidal fibers are absent in the spinal cord and can be traced only in the brain and medulla. It stands to reason that there is no continuity between the cortex, which sends out stimulation, and the peripheral nerves, or else the stimulation sent out from the cortex to the spinal centers is not properly transmitted for want of normal pyramidal fibers. The rigidity of the muscles in diplegia can therefore be explained either by increase of the muscular

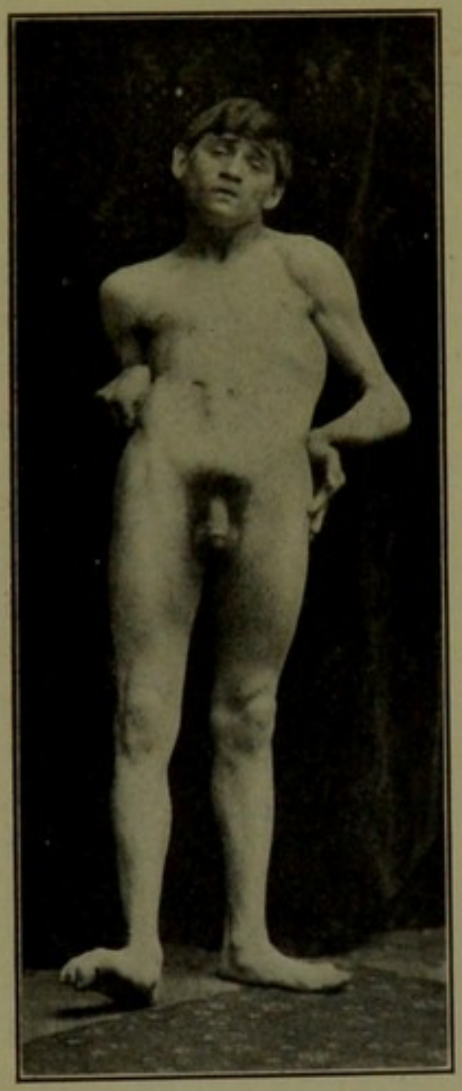


FIG. 68.—DIPLEGIA, LITTLE'S DISEASE.

FIG. 69.—PARAPLEGIA, LITTLE'S DISEASE (Idiot).

tonus or by transmission of a continuous morbid stimulation through imperfectly developed pyramidal fibers.

Fournier had long ago called attention to hereditary syphilis as an etiological factor in Little's disease. In a large number of cases the disease is present in individuals whose parents were distinctly syphilitic. Not infrequently diplegic children present certain stigmata of syphilis, such as hydrocephalus, internal strabismus, etc. In some cases at autopsy

of diplegics lesions of syphilitic nature had been found: sclerotic and gummatous formations, meningeal conditions, cerebral endarteritis, meningo-myelitis. Finally, Wasserman reaction has been frequently found positive in diplegics. On the other hand, a negative Wasserman does not exclude syphilis. The fact that diplegia is observed frequently in association with premature birth is also suggestive of hereditary syphilis.

Symptoms.—The most frequent form is the **paraplegic.** The gait and station are characteristic: the patient's trunk is bent forward, he looks at his feet, which are in a state of varo-equinus. When standing the thighs are in close contact with each other, while the legs are separated. In walking the legs have a tendency to cross each other, the feet make a hemi-circle movement, and scrape the floor at each attempt to advance and the trunk turns laterally. When seated, the legs do not touch the floor; they are extended. The rigidity is so great that flexion of the joints is impossible.

In the **diplegic** form the upper extremities are also affected, but to a milder degree: adduction of the arms, flexion and pronation of the forearms are the principal signs. The face not infrequently participates in the general rigidity. When the patient speaks, laughs or masticates, there is a very marked contortion of the muscles of the face; the speech is therefore indistinct. Should the muscles of deglutition, phonation or respiration be affected, corresponding symptoms will be present. They constitute the congenital pseudo-bulbar paralysis; succion, deglutition are disturbed; later the speech shows slowness and words are pronounced in an explosive manner. The contracture of the extremities may not be evenly distributed and its intensity may vary in different limbs.

The condition of the reflexes is the same as in the hemiplegic form, viz. exaggerated patellar or other tendon reflexes, Babinski and paradoxical flexor signs; ankle-clonus is rare. Athetosis is not infrequently observed in both hands. Choreic movements are less frequent. The mental condition in Little's disease deserves special mention. While the children thus affected present some delay in development of their mental faculties, they nevertheless do not show marked impairment of intelligence, and in some cases, with proper training, their intellect may reach a normal degree of development. There are also cases of infantile encephalopathies of the type of diplegia in which idiocy and imbecility are observed. In these cases epilepsy is very frequently associated. Diplegics not infrequently present a peculiar excitability in regard to noises, what Oppenheim called "abnorme Schreckhäftigkeit." When

they hear an object fall on the floor or a noise of any sort, they are seized with a trembling which may last a long time. The condition is an acustico-motor hyperesthesia.

Prognosis. (a) **In Infantile Hemiplegia.**—Generally speaking, it is unfavorable. The contracture and the atrophy, viz. the arrested development of the tissues of the palsied limbs, render the infirmity permanent. Improvement of the paralytic symptoms may follow, especially during the first year, but it is usually slight. Epilepsy is a very grave complication, as it prevents any possible amelioration of symptoms and retards the development of the mental faculties. Epilepsy at the onset of the affection may cause rapid death. According to Bourneville the attacks of epilepsy become rarer as the child grows and often disappear at the age of thirty. The development of mental faculties is usually delayed, but with a proper training good results may be obtained.

(b) **In Diplegia.**—The prognosis in this affection is less unfavorable than in the hemiplegic form. There is a natural tendency toward improvement, but recovery can never be expected.

Diagnosis.—The spasticity of the paralyzed extremities, marked contractures, athetosis or chorea with epileptiform convulsions, the period of life at which these symptoms make their appearance are usually sufficient facts for making a diagnosis. In some cases, however, difficulties arise. When the infantile spastic hemiplegia presents an acute onset, it may simulate tubercular meningitis. In such cases the diagnosis cannot be made at the beginning: the course of the affection alone will enable one to make a correct diagnosis.

Infantile spinal palsy also has an acute onset, but the flaccidity of the palsy alone besides the diminution or less of reflexes is sufficient to determine the disease.

Obstetrical palsy, limited usually to one upper extremity, can be recognized by its flaccidity.

Tumor of the brain will be recognized from the slow onset, from the history of headache and vertigo and especially from the condition of the eye grounds (optic neuritis or atrophy, or choked disc).

Little's disease, especially the diplegic form, is so characteristic (see above) that it cannot be easily confounded with other affections. The paraplegic variety will be differentiated from paraplegia caused by myelitis mainly by the absence of disturbance of the sphincters of bladder and rectum, also by the history of the case.

Treatment.—In the chapter on etiology, among various causes of spastic paralysis, dystocia was considered as a possible factor. It is therefore evident that prompt obstetrical intervention during difficult

labor is an important preventive measure. When the child is born normally and easily and still presents spastic paralysis, the condition is congenital. When the hemiplegia preceded by the group of acute symptoms (see Symptomatology) appears some time after birth, the treatment will not be practically different from an apoplectic insult in an adult. It is the treatment of deformities and infirmities that we are requested to remedy. Internal drugs are of no value except for epileptiform convulsions, in which case bromides are indicated. However the relation of diplegia to hereditary syphilis (page 349) is a strong indication for the use of antispecific medications, viz. mercury, iodids and especially salvarsan. Mechanical means and surgical intervention is the only possible treatment. It is well understood that conditions like porencephaly, internal hydrocephalus, sclerosis and atrophy of the brain can never be benefited by operative procedures. When the paralytic or epileptic symptoms are due to a vascular lesion or to a cyst, will an operation give a permanent recovery? Is it justifiable to perform even an exploratory operation? Statistics carefully collected show that some improvement was observed in a certain group of cases, but as the cases were not kept under observation a sufficiently long time, it is impossible to draw positive conclusions. Moreover in a small group of cases the slight improvement was only temporary and a number of fatal results were also reported. However, more recent investigations, especially those of Cushing in 1905, show that in many cases of congenital paralysis hemorrhage within the cranium at birth is the main etiological factor. Prompt and early surgical intervention for removal of blood-clots gave him in some cases very satisfactory results. He opens the skull at the level of the parietal region, makes a large opening in the dura, and washes out the blood. A similar operation is done on the other side of the brain. In view of the great gravity of such operations in newly born infants Gilles devised a method which apparently gives promising results. In cases in which a meningeal hemorrhage is suspected a puncture of the anterior fontanelle should be made. It decreases intra-cranial pressure and reduces the danger of infection caused by absorption of toxic products from the blood clot.

This is in Gilles' opinion a very good preparatory treatment. When the brain becomes more developed and the child is stronger, a radical operation may be undertaken, such as Cushing's. Gilles' rule is at first to perform a lumbar puncture and when the latter gives no relief, puncture the anterior fontanelle (see also page 166).

Surgery directed toward the deformities and contractures is apt to give favorable results. Tenotomy and myotomy followed by applica-

tion of plaster casts to the limbs put in corrected positions has given favorable results in certain cases. Various orthopedic appliances, passive manipulations of the limbs, mobilization of the joints and properly regulated gymnastics are highly commendable for counteracting the rigidity of the muscles and of their tendons. Massage is a great adjuvant in this treatment; it should never be overlooked, as very good results are sometimes obtained. It is a good plan to administer frequently warm baths which will facilitate the reduction of the muscular spasticity. Electricity is not advisable as it is likely to increase the muscular rigidity.

Recently Förster (*Beitr. z. Klin. Chir.*, 1909) has suggested to resect the posterior spinal nerve rcots for relief of spasticity. He based this operation upon the following physiological considerations. Spastic paralysis is due to a diseased condition of the corticospinal tract, especially the pyramidal tract. The latter carries two kinds of fibers: those bearing the motor impulse, a disease of which causes the paralysis, also those containing inhibitory fibers whose function is to check the sensory stimuli received from the sensory roots of the spinal cord. The spasticity is therefore a reflex act. As a result of the damage to the inhibitory fibers, the continuous sensory impulses act unrestrained, hence the spasticity. When in such cases the posterior roots are cut, the sensory afflux is removed and the spasticity becomes diminished or disappears entirely. A large number of operations have since been performed and in the majority of cases satisfactory results have been obtained. Spastic diplegia and hemiplegia can be relieved considerably by Förster's operation. For the upper extremity the last four cervical roots and for the lower extremity the last three or four lumbar, also the first and second sacral roots should be removed on both sides. The after-treatment is very important. Förster insists upon careful exercises of the limbs, which must continue for years after the operation. Immediately following the operation the limbs should be placed in removable plaster splints in corrected positions. The splints will be kept on for a very long time and removed off and on only for the exercises, which must be carried out several times a day.

Recently, Schwab and Allison (*Trans.* Amer. Neur. Ass'n., 1910), devised a method of treatment of spasticity and athetosis which they called, "muscle group isolation."

It consists of isolating muscles or groups of muscles which are at fault in the production of contracture, deformity or athetosis. It is done by cutting off from the central nervous system the connection along which the abnormal impulse is transmitted. This is accomplished by isolating the nerve innervating the affected muscles and injecting it with alcohol. A paralysis of these muscles will thus be induced with-

out interfering with the antagonistic muscles. The subsequent treatment will consist of massage and physiological exercises. In Little's disease for example the obturator nerve which supplies the adductor muscles of the thigh is to be injected with alcohol. This method is also advisable in deformities of anterior poliomyelitis.

Another method for "muscle isolation" in treatment of spastic paralysis has been devised by Stoffel (*Münch. med. Wchn.*, 1911 and 1912). His operation aims to weaken the contracted muscle by depriving it of a certain part of its innervation, while at the same time the antagonist muscle is strengthened by massage and exercises. When the nerve is exposed for 3 or 4 cm., its different fibers are touched with a needle electrode carrying a current so weak that the innervated muscle barely twitches. It is thus possible to single out the nerve fibers involved. Thus instructed the tract innervating the muscle involved in the contracture is worked loose for a distance of 5 or 7 cm. The nerve bundle is then severed in cases of severe contracture, or only part of the fibers. The after-treatment is very important. When the operation is correctly performed and suitable after-care with massage and exercise is given, there will be a certainty of success in correcting spastic contracture of any form and of any duration. Stoffel gives a final review of his many cases, almost all ending successfully, in *Therap. Monatshefte*, Dec., 1912.

CHAPTER VI

JACKSONIAN OR FOCAL EPILEPSY

The existence of a special form of epilepsy and different from the ordinary generalized epilepsy was observed first in hemiplegics on the paralyzed side of the body by Bravias, but H. Jackson (1869) deserves the credit of demonstrating the relationship between a unilateral epilepsy and a **cortical lesion** of the brain.

Pathology.—It is definitely established that Jacksonian or focal or partial epilepsy is caused by an irritation of the cortex of the motorarea of the opposite side. The focus of irritation may be situated in the kull (pressure of a fragment in traumata), in the meninges (pachymeningitis, syphilitic meningo-encephalitis), in the cortex itself and in the ubcortical tissue (tumors, etc.). Whether the irritation is from without or from within, the excitation of the motor cells of the Rolandic area is indispensable to produce a focal epilepsy. Sometimes the irritative lesion is found outside the motor area. In such cases the irritation of the cortical cells of the motor region is set up secondarily. Irritation of the cells produces tonic followed by clonic convulsions, while irritation of the subjacent nerve-fibers alone produces only tonic convulsions.

Symptoms.—An attack of Jacksonian epilepsy consists of tonic and mainly clonic convulsions which begin, mostly without loss of consciousness, in a very limited group of muscles on one side and which rapidly spread to a half of the body and sometimes to the entire body. According to the point of departure of the convulsions three types can be considered, viz. **facial, brachial** and **crural.** In the first type the spasm affects the face and neck. Either one angle of the mouth is drawn up or one eye is rolled up; the head is turned toward the same side. The spasmodic contractions become precipitated and soon invade the muscles of the neck and of the arm. The forearm rapidly becomes pronated, the hand closes. Immediately the lower extremity becomes extended and rigid. The clonic contractions follow instantly. In the second type, which is the most common, the initial symptom appears in the thumb, which becomes flexed and the other four fingers follow. The hand is pronated, a rigid flexion of all the segments of the limb sets in and the convulsive movements begin immediately. The face and the lower extremity follow.

In the third type the initial symptom appears in the great toe. Un-

like the upper extremity, here tonic contractions are rare, clonic movements appear usually from the beginning. The upper extremity and face become invaded subsequently. The order of appearance of the symptoms as just described is not always observed. The convulsions may commence in one area and remain confined to the same, or else they may spread only to a portion of the half of the body.

In another group of cases the convulsions may become generalized and affect the entire body. In such cases **the importance of the knowledge of the onset** of the convulsions cannot be overestimated. The onset is the most precious **localizing sign.** Surgical intervention is based almost exclusively upon this information. As to what portion of the cortex causes convulsions of the face, arm and leg, see chapter "Cerebral Localizations."

The attacks of Jacksonian epilepsy may be preceded, accompanied or followed by a few symptoms which deserve mention. The **aura**, which is present in idiopathic epilepsy, is quite frequently met with in focal epilepsy. It may be **motor, sensory** and **psychic.** The motor aura consists of an involuntary movement in some of the muscles, which is then the signal of an oncoming attack. The sensory aura is various. It may be a sharp lightning pain, a sensation of heat, of cold, a tingling, etc. The psychic aura consists of hallucinations (visual and aural): patient sees flashes of light, fire, red or green objects, etc. In some cases a state of confusion precedes an attack. One of my patients would run bewildered immediately before he would fall unconscious. As to **unconsciousness,** during an attack it may be **complete, incomplete** or **entirely absent.** In the latter case the patient witnesses the entire cycle of symptoms. When there is only partial loss of consciousness, the patient has a vague knowledge of his personality and he is then apt to commit acts for which he is not responsible. Pallor of the face, followed by cyanosis and involuntary micturition, are two additional symptoms observed during the attacks.

After an attack is over, the patient lies motionless and appears exhausted. Gradually he regains consciousness, looks vaguely about him and finally recovers; headache frequently follows an attack. Not uncommonly is observed after an epileptic attack a **paralysis** of the limbs involved. The palsy is usually transitory, may last twenty-four or forty-eight hours, although sometimes may remain permanent. In the latter case there is probably a certain permanent damage of the cortical tissue (hemorrhage followed by softening). The transitory post-epileptic paralysis is flaccid, but increased knee-jerk and sometimes Babinski's sign will be observed. The paradoxical reflex has been observed by me

in every case that came under my observation and disappeared when the paralysis disappeared.

Varieties.—Besides the typical attack, in which the tonic convulsions are followed by clonic, there is a form which consists exclusively of **tonic** contractions; the latter then constitutes the entire attack. In such cases the irritative lesion is in the nervous fibers subjacent to the cortical cells. Finally there is a **sensory** variety, in which the entire attack consists of a sensory phenomenon. A patient of the Jefferson Hospital presented sudden brief attacks of coldness and numbness in the entire left side of her body. This lasted only two minutes, during which time she felt dazed; her face was pale and after the attack was over she went to sleep.

Prognosis.—It depends exclusively upon the cause of the cortical irritation. When the condition is amenable to surgical intervention, the outlook is favorable. It is also favorable in syphilitic cases. Generally there is a tendency to destruction of the irritated area, so that eventually a paralysis supervenes.

Diagnosis.—The mode of onset, viz. the initial appearance of convulsive attacks on one side of the body, is sufficient to make a differential diagnosis between focal and essential epilepsy. Hysteria is an affection in which unilateral convulsive movements may simulate Jacksonian epilepsy, but the presence of hysterical stigmata and the character of hysterical muscular contractions will enable a close observer to make a distinction between the organic cerebral disease and the functional nervous disease (see Hysteria). After the diagnosis is established, it is highly important to ascertain the cause and localize the seat of the irritating factor. When an evident injury to the cranium had occurred or when there are evidences of an intracranial neoplasm, it is comparatively easy to explain the pathogenesis of the disease. When convulsions are followed by a paralytic or paretic condition of the face, arm or leg, the diagnosis is easily established. In a number of cases it is difficult, if not impossible, to determine the cause and indeed no organic lesion is found during operation or post-mortem in a great many cases. Syphilitic gummata or localized specific pachymeningitis should be always thought of in obscure cases. Wasserman test will be of considerable assistance in such cases. As to the localization of the lesion, it will be determined from a close observation of the attacks, as the seat of the convulsive attacks or the seat of their beginning will enable us to localize the lesion in the cortex. X-ray examination may render a valuable service in such cases. Pain may sometimes be elicited by pressure over the area corresponding to the suspected irritative lesion.

Etiology.—It has been already mentioned that irritation of the motor zone of the cortex, which is the chief cause of Jacksonian epilepsy, may be produced by fragments of the skull in traumatism, by hemorrhages, localized meningitis, gummata, tumors. All these factors act at first mechanically upon the cortex and then cause an inflammatory condition of the latter. But there are other conditions in which focal epilepsy has been observed. Alcoholism and lead poisoning are two well known

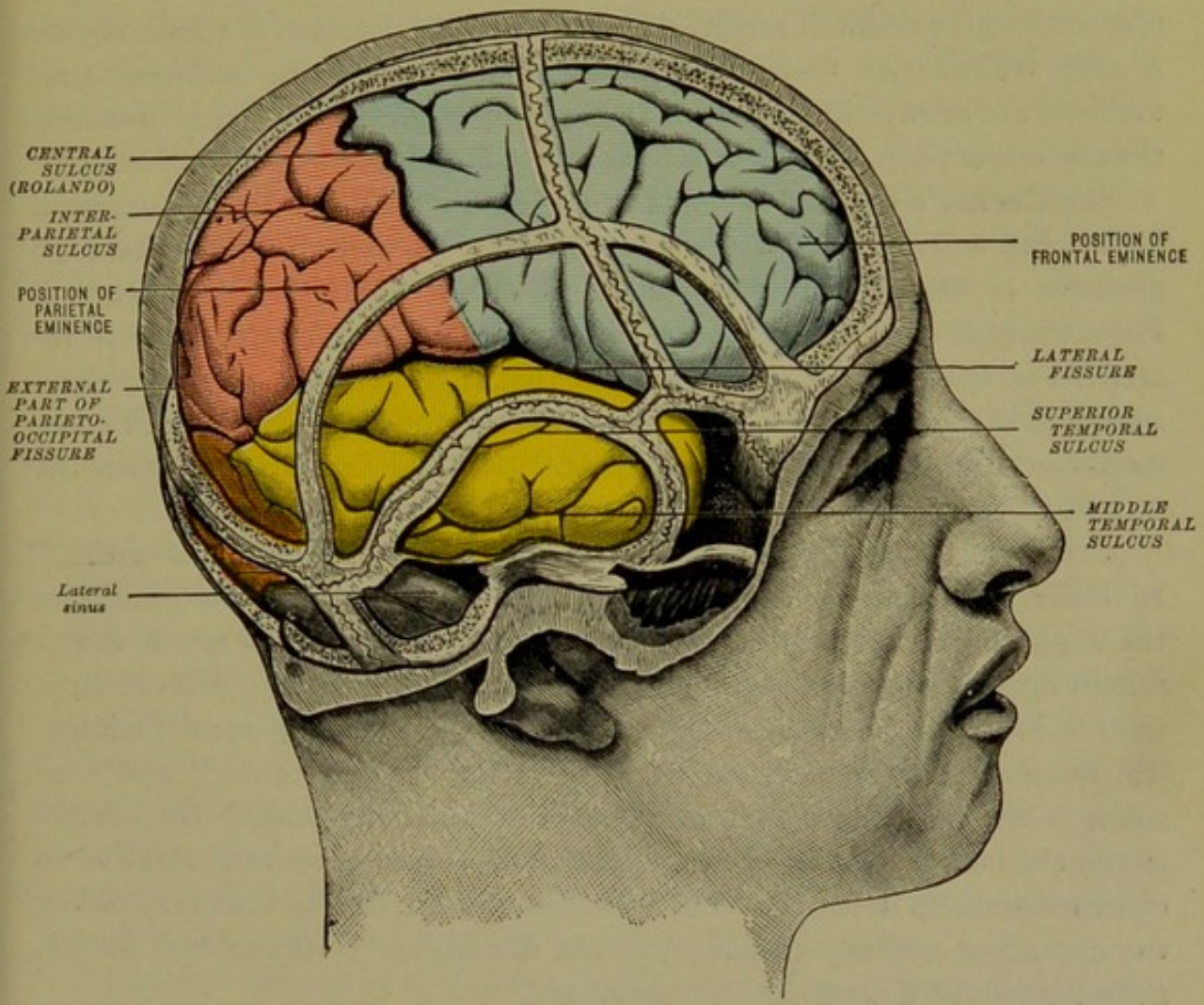


FIG. 70.—DRAWING OF A CAST OF THE HEAD OF AN ADULT MALE. (*Morris' Anatomy.*) (Prepared by Professor Cunningham to illustrate cranio-cerebral topography.)

intoxications, to which Jacksonian epilepsy can be attributed. That uremia may become complicated by attacks of Jacksonian epilepsy is also a well-known fact. On the other hand **peripheral irritation,** as, for example, injury to nerve trunks, burns, cicatrices, polyps, surgical operations, etc., have been the starting cause of unilateral epilepsy. In such cases the pathogenesis being obscure, one is bound to accept the view of a **reflex** origin of Jacksonian epilepsy.

Treatment.—When an intoxication can be traced as a cause of the epilepsy, medical treatment is indicated and it will consist of the removal of the cause. When syphilis is ascertained or suspected, mercurials and iodides also salvarsan may yield excellent results. In all other conditions surgical intervention is justifiable. Operations should be always preceded by a careful mapping out of the area involved (Fig. 70) and aided by X-ray pictures. Also each operation should be preceded by a trial of specific treatment until the point of intolerance. While in some cases operations give brilliant results, in others the amelioration of the condition is only transitory. Outside of intoxications and uremia unilateral convulsions are always justifiable of an exploratory operation. The methods usually employed are those of **Kocher** or of **Horsley.**

(1) **Kocher's method** consists of trephining with incision of the dura. It has this advantage that in cases without an evident lesion the high pressure of the cerebro-spinal fluid is either diminished or removed. Experimental and clinical observations show that the pressure is increased in epilepsy. Kocher believes that high pressure of the cerebro-spinal fluid is the cause of epilepsy. He removes a small round piece from the skull, excises the dura, replaces the bone and covers it with the incised integument.

(2) **Horsley's method** consists of excising the so-called "signal center." In order to determine precisely the seat of this center, he stimulates the supposed center with a very weak faradic current. When a motor manifestation is reproduced, he excises the area even if there is no evident local lesion. A center apparently normal, he thinks, may show histological lesions. Krause adopted as a guide the "signal center" and considers it the most important element of surgical diagnosis. He advises to remove twenty or twenty-five millimeters of cortical surface and five to eight millimeters in depth. The paralysis and anesthesia that may follow the operation are only transitory if the dimensions of the excised cortex, as indicated by Krause, are not increased.

Lumbar puncture when repeated and large amounts of cerebro-spinal fluid removed may sometimes give considerable relief in epilepsy, but this procedure is inferior to the operations mentioned above.

EPILEPSIA PARTIALIS CONTINUA

Under this name Kojewnikoff described in 1894 a special form of epilepsy the essential feature of which is the co-existence of typical seizures and persistent partial convulsions between the seizures.

Symptoms.—The disease may begin either with generalized attacks

or attacks confined to one limb, to the face or to one half of the body. These attacks are typical of epilepsy. In the intervals between the classical seizures frequent involuntary contractions of muscular groups are observed. They are *clonic* in character. Their range varies from fibrillary contractions to very wide movements. They always occur in the same part of the body. They are more violent immediately before the typical epileptic seizures, but after the latter they are very feeble. They cannot be controlled by the will. They increase upon emotion. During sleep they decrease and may disappear. When the movements are pronounced, walking is difficult. The power of the affected side is diminished; atrophy and contracture are occasionally observed. The tendon reflexes are exaggerated, but ankle-clonus and Babinski sign are absent. Pain is sometimes complained of, but the objective sensibilities are normal. In exceptional cases the following additional phenomena have been observed: aphasia or dysarthria, facial paresis, acceleration of pulse and respiration, spasm of œsophagus, involuntary laughing and crying.

Course and Prognosis.—The disease runs a slow course, but it is persistent and interferes with the patient's occupation. It is usually aggravated after an attack of epilepsy. The outlook is serious. Death usually occurs from some intercurrent disease.

Pathology and Pathogenesis.—Autopsies of cases with Kojewnikoff's syndrome are very rare. In Osokine's case (*Rev. de. méd. de Moscou*, 1908) a portion of the motor cortex on the side opposite to the seat of convulsions was excised. It proved to be a meningo-encephalitis. A very recent case of Kramholz (*J. of Nerv. and Ment. Dis.*, 1913) with autopsy also shows that the symptoms were due to encephalitis with a sclerotic process in the motor cortical region. Choroschko incriminates also the optic thalamus and the posterior quadrigeminal bodies. The pathogenesis is still debatable in view of the paucity of anatomical proofs.

Etiology.—Infectious diseases, syphilis, alcoholism, trauma have been observed as immediately preceding the onset of the disease. Men are more frequently affected than women.

Treatment.—Bromides, nitroglycerine are the only drugs which may be useful in decreasing the intensity and frequency of the convulsions. Operative procedures give better results. Horsley's method has been used with great satisfaction (see page 128).

CHAPTER VII

APHASIA

UNDER the old conception (Trousseau) this term expressed merely an inability to express ideas in words. In the light of our present knowledge this conception became naturally broader and now aphasia means an inability to communicate ideas not only in words but with any other sign. It affects therefore two faculties: **comprehension** and **expression.** Consequently there are two kinds of aphasia: (1) aphasia of expression or **motor aphasia** (Broca's aphasia) and (2) aphasia of comprehension or **sensory aphasia** (Wernicke's aphasia).

I. MOTOR APHASIA

Ideas may be expressed in words or in writing. Loss of function to articulate words is called **aphemia,** to write **agraphia.**

Aphemia.—Individuals presenting this symptom are able to hear and understand when spoken to (their inner or intrinsic language is intact); they are able to emit sounds, to move the lips and tongue. What they have lost is their vocabulary; otherwise speaking, the faculty to articulate words.

Aphemia may be **complete** and **incomplete.** In the first case the patient cannot articulate even one syllable. He resembles a mute. More frequently his vocabulary consists of only one or several unintelligible syllables which he repeats at each question. In the second case there are infinite varieties: here the vocabulary may be reduced to one or two words, which the patient always pronounces and repeats whenever spoken to; or to a whole phrase. In mild cases only certain words are wanting. In ordinary cases the aphasic gradually regains his lost words and reëducation may help considerably in this respect. There are also cases which are absolutely incurable. In individuals who speak several languages, aphasia affects first the acquired languages and when improvement takes place, the patient will commence to speak his native tongue long before the foreign. Incomplete aphemia may be modified under certain influences. H. Jackson and Trousseau report cases in which the patients during an emotion (anger) recovered words which they otherwise could not pronounce. Some aphasics repeat words

spoken close to their ear. Here the motor speech center is stimulated by the auditory center. **Echolalia** is thus explained. For the same reason some aphasics while singing are able to pronounce words which otherwise they cannot. Visual impressions may play the same rôle as auditory. One of my patients could pronounce words only when she saw them written. As to the anatomical seat of aphemia, according to the classical view it is the posterior portion of the **left third frontal convolution** (Broca's region) in right-handed and the same portion in the right hemisphere in left-handed individuals. (See chapter on Localizations, and Marie's new conception of Aphasia.)

Agraphia.—The patient thus affected has lost the faculty of expressing ideas in writing. The disturbance of this function may present various forms. It may be complete when the patient is incapable of making the slightest sign on the paper, or incomplete when only one word or certain words can be written. Sometimes the letters are irregularly placed, sometimes the combination of letters or words is such that they do not correspond to the idea the patient wishes to express. In some cases the agraphia affects only figures, not words; in others only copying and not spontaneous writing; in still others the patient can write only under dictation.

In **paragraphia** the patient substitutes to the right words words without meaning. The agraphics who are at the same time hemiplegics exercise often their left hand for writing, but instead of writing normally from left to right they write from right to left. This is **mirror writing.** It resembles a writing as it appears through a mirror.

Agraphia without motor aphasia (aphemia) is exceptionally rare. It is sometimes associated with other forms of aphasia, quite frequently with word-blindness. The function of word-seeing (reading) is frequently necessary for writing either spontaneously or under dictation; if this faculty is abolished (alexia), writing is impossible. Copying, however, is possible as the agraphic is able to imitate letters like drawings even without knowing their meaning. Indeed, there are cases on record in which a lesion of the angular gyrus involved also the faculty of writing. The loss of function of word-hearing may also cause inability to write. The very frequent association of agraphia with motor aphasia can be easily understood: first because of the close proximity of their centers (a lesion of one can hardly escape from involving the other center); also, because of the well known fact that a child as well as many adults pronounce words or articulate them while they write. A loss or impairment of the first function will produce a disturbance of the latter. An independent existence of agraphia is therefore disputed. However, there

are a few cases on record in which there was no other disturbance of speech and of intelligence and still the faculty of writing was lost. The anatomical seat of the function of writing is **supposed** to be in the posterior portion of the left second frontal convolution. This question is still debatable.

II. SENSORY APHASIA

(a) **Word-deafness.**—This condition, to which Kussmaul in 1876 was first to call attention, consists of a loss of the faculty of understanding spoken words. The patient hears the voice, as his general hearing and other faculties are not affected, but the words are to him mere sounds and do not express ideas. Word-deafness may be **complete** (very rarely), when the patient cannot understand one word, and **incomplete,** when only some words are properly interpreted by him. In the latter case there may be many varieties according to the number and character of the words understood. Word-deafness, when it is pronounced, is associated with other speech disorders. In ordinary conditions the acts of speaking, reading and writing are all dependent on inner or mental hearing; it is the inner words that command the outer words (speaking) and dictate the writing, also during the act of reading the mental or inner hearing reproduces the words read. It is therefore evident that visual images are intimately associated with auditory images and that word-deafness disturbs the faculties of speaking, reading and writing.

In the majority of cases the word-deaf shows some disturbance of motor speech. When, for example, he is asked a question in writing his verbal answer will be disturbed, as he is unable to hear his own words. It then frequently happens that the patient's entire speech becomes incomprehensible: either he uses one word instead of another (**paraphasia**) or employs unintelligible words (**jargonaphasia**). Both disorders are then observed in spontaneous and repeated speaking also in reading aloud, because the patient being unable to hear himself, cannot correct his defective speech. Not only paraphasia but also **paragraphia** is observed in word-deafness. The patient writes like he speaks: he substitutes wrong words for the right ones.

There is a variety of deafness referable to music (**auditory amusia**), in which the patient is unable to recognize familiar songs or unable to distinguish one from another. There is also a variety of deafness in which not words but figures are misunderstood and misinterpreted. Word-deafness may be incurable, but in the majority of cases with the aid of reëducation the patient regains some words. Other centers,

especially the visual, render great assistance: when the patient is often spoken to, he learns to observe the movements of the speaker's lips and thus gradually some old lost verbal images return.

The anatomical seat for word-deafness is, according to the majority of neurologists, in the left first temporal convolution (see "Localizations").

(b) **Word-blindness or Alexia.**—It was first described by Kussmaul in 1877. It is characterized by an inability to read printed or written matter. The patient lost the faculty of reading even his own writing, although his vision and intelligence are intact; he sees the form of the letters but not the idea expressed in them. Various degrees of alexia may be present. While on one hand it may be complete, so that not one letter is discerned by the patient (**letter-blindness** of Kussmaul), on the other one or several words or only syllables may be seen. There may be blindness for words, but not for letters; the patient is able to recognize letters, but not able to understand the meaning of combinations of letters, viz. syllables or words. Similarly to word-deafness we find here also varieties of alexia in which the patient is blind not for words or letters, but for **figures** or for **musical** notes.

Patients affected with alexia present usually difficulty in spontaneous writing as the writing itself consists in reality of copying their visual images, but the agraphia is never complete. For the same reason writing under dictation is impossible. Copying is done like drawing. When the word-blindness is not complete, the patient may be able to recall, visually, words either by writing or by hearing. Thus he follows sometimes with his finger the letters which he cannot see with his eyes, or else he finds the letters or words when he hears them pronounced. Graphic or auditory images may therefore assist in recalling visual images.

In another variety of word-blindness the patient is able to read and copy letters and words, but unable to comprehend their meaning. It is exactly what happens when thinking of another subject we keep on reading; then suddenly we notice that we have read an entire page or more and do not know the contents. This is the so-called **"psychic blindness."**

The anatomical seat for alexia is in the left **angular gyrus** (see "Localizations").

OTHER FORMS OF APHASIA

Aphasa resulting from a Lesion beneath the Cortical Centers (Conduction Aphasia of Lichtheim-Wernicke).—In the preceding chapter I discussed the varieties of motor and sensory aphasia in cases when the lesion

lies in the cerebral centers, viz. in the cortex. Anatomy teaches that that various portions of the cortex are connected with each other by **association** fibers and that through the internal capsule (knee) pass motor fibers whose destination is to supply the organs of phonation.

A lesion situated in those fibers, viz. beneath the cortex, will constitute the so-called (1) **"Transcortical aphasia,"** or pure aphasias. Similarly to the varieties of the cortical, the transcortical aphasia also presents to the four main types: (1) aphemia, (2) agraphia, (3) word-blindness and (4) word-deafness. In order to understand the mechanism of these subcortical aphasias it is necessary to bear in mind the anatomical arrangement of the association paths. Supposing, for example, that a lesion has interrupted the connection between the angular gyrus and the foot of the second frontal convolution, viz. between the center for word-reading and the writing center. It is easy to conceive that the patient will be able to see and understand (as the visual centers are preserved), to read aloud (as the fibers connecting the angular gyrus with Broca's region are intact). The patient will **not** be able to copy what he sees because of interruption of the above fibers, but he will be able to copy only when he reads aloud, because of the integrity of the fibers connecting the auditory verbal center (first temporal convolution) with the graphic center.

Another example. A lesion interrupts the association fibers between the common visual center and the center for word-reading The patient will be able to see (as the common visual center is preserved), to copy (as the connection between the common visual and graphic centers is intact), but he will not be able to read; the letters and words are incomprehensible. These are the so-called *pure aphasias;* they are caused by isolation of the respective speech-centers. To the pure motor aphasia variety Marie gave the name of *Anarthria* (see below).

The two examples are sufficient to understand all the varieties of aphasia caused by a lesion of the sub-cortical fibers. It is necessary only to bear in mind the scheme of the association fibers.

It was mentioned above that the internal capsule contains motor fibers for speech. A lesion of these fibers will produce a **transcortical motor aphasia.** In this case the disturbance of speech will not be similar to that of a lesion of Broca's region. It will consist only of a **dysarthria** or **anarthria** similar to that found in lesions of peripheral organs of the speech or in bulbar affections. Here the cerebral speech-function is not defective, as the patient has the power of communicating his thoughts in every way but the words are unintelligible (articulatory defect).

The anatomical distinction between the **cortical** and sub-cortical or **transcortical** forms of aphasia cannot be applied with such a degree of

precision to the clinical picture; otherwise speaking, is there any clinical difference between the two groups of aphasia? While the existence of sub-cortical aphasia is proven beyond doubt, yet clinically it is impossible in the majority of cases to say that such and such disturbance of speech is due to a lesion of the cortical centers or of the subjacent fibers. Dejerine and Lichtheim, however, called attention to a few differential points, but as the latter present some objectionable features and are not accepted by the majority, it is unnecessary to go into details.

(2) **Verbal Amnesia.**—Under this term is understood an inability to recall names. When the disorder is only slight, the individual may after a certain effort recall the name of the object; he will do it either by seeing it written or by recalling again and again its mental image. In complete verbal amnesia all the efforts are in vain; he will not be able to find the name of the object. According to Pitres, this form of aphasia is caused by a rupture of the commissural paths which connect the various centers of verbal image with those portions of the cortex on which the higher mental acts depend. It is therefore also a transcortical aphasia. On the other hand, cases have been reported by Bianchi, Mingazzini, Mills, Pick and others which tend to show that the inferior parietal lobule could be considered as the center of verbal amnesia.

(3) **Optic Aphasia** (Freund).—An individual thus affected recognizes, objects placed before him but is unable to name them. When, however, he palpates, smells or tastes them, he immediately recalls the name. Physiologically this disorder can be explained either by interruption of connection between the visual and speech center or by an insufficient excitation of the cortex by the optic impression of the unnamed objects.

(4) **Congenital Aphasia** (Kussmaul).—It is not the result of a localized central lesion. The child hears and understands but cannot speak or at least cannot articulate as well as other children of his age. The pathogenesis is obscure.

General Remarks on Aphasia.—The preceding study of motor and sensory speech disturbances may lead to the idea that each form of aphasia is independent of others. In reality such an occurrence is an exception. In the majority of cases the involvement of one center affects other centers. Aphemia, for example, is frequently associated with agraphia, word-blindness also affects the faculty of writing, word-deafness the faculty of articulating speech. There are also cases in which motor aphasia is associated with sensory aphasia; all forms of speech are abolished. The aphasia is then **total**. The latter is frequently coincident with right hemiplegia. Pathologically however (Gowers, Bastian, Dejerine) not the entire speech area is found to be affected in

every case. Beduschi (*Encéphale*, 1910) has shown that total aphasia occurs almost always when Wernicke's zone is involved together with the gyrus supramarginalis. In the white substance of the latter lies an arcuate bundle of fibers, through which pass the association fibers connecting the visual and auditory areas with the motor center of speech.

The **mental state** of aphasic individuals deserves mention. It is usually affected. The enfeeblement is more marked in the sensory than in the motor form, and in the cortical more than in the sub-cortical type. The degree of mental impairment depends upon the extent of the lesion and varies from one individual to another.

Aphasia in left-handed individuals presents the same characteristics as in right-handed ones. Pathologically lesions identical with those described on the preceding pages have been found in the right hemisphere of left-handed individuals, as the most recent studies of Mingazzini (*Encéphale*, 1908), and of Monakow (*Grosshirn Pathologie*, 1905) proved it conclusively.

Latest View on Aphasia.—It was just mentioned that an isolated involvement of any one of the speech-centers is a great rarity. Indeed clinically we observe, for example, in the motor aphasia of Broca's type not only loss or disturbance of spoken language, but almost always some degree of alexia and agraphia; also some disturbance of the inner (intrinsic) speech. Pathologically only a lesion of the F^3 is held accountable. In the sensory type of aphasia (Wernicke) in which the posterior third of the T′ and the angular gyrus are supposed to be involved, not only there is a defect of the inner speech and inability to understand spoken and written words but there is also a marked defect of motor speech (paraphasia and jargon aphasia). There have been cases reported in which a diagnosis of one form of aphasia was made during life and at autopsy lesions corresponding to the other form or to both forms simultaneously were found. In view of the complexity of the subject Marie's claim for a reconsideration of the problem of aphasia becomes a matter of necessity and the following is the new view held by Marie and his followers.

Marie's View on Aphasia.—Basing himself on clinicopathological records of a large number of cases (*Semaine Médicale*, 1906), Marie rejects the following features of the old classical conception of aphasia, viz. the specificity of Broca's center, the distinction between motor and sensory aphasias, the existence of pure (sub-cortical) forms of aphasia and finally the existence of individual speech centers. He argues that there is only *one* aphasia, there is only one speech-center localized in the left temporo-parietal lobe which is at the same time the specialized intellectual center but not the center for sensory images. Word-deafness and word-blindness

are but defects of the special intelligence of speech. Intellectual deficit is the main characteristic feature. Broca's aphasia is a combination of Wernicke's aphasia and anarthria. The latter is caused by a lesion of a quadrilateral area surrounding the lenticular nucleus (**lenticular zone**) ;

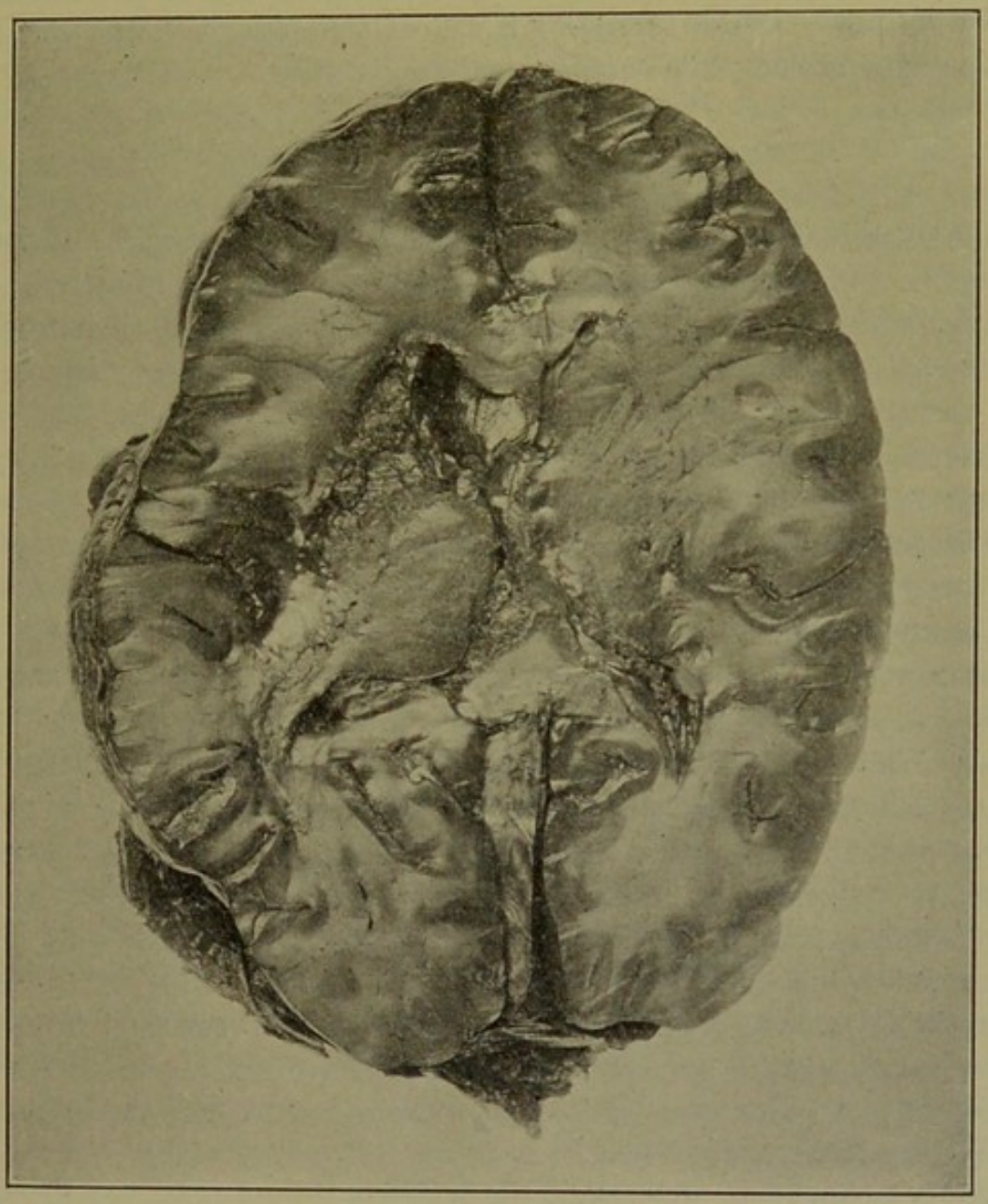


FIG. 71.—SOFTENING IN LEFT HEMISPHERE SHOWING COMPLETE DESTRUCTION OF THE LENTICULAR ZONE. (*Original.*)

its limits are: anteriorly a vertical plane level with the anterior sulcus of the insula, posteriorly a similar plane with the posterior sulcus of the insula, internally the lateral ventricle, externally the surface of the insula. Posteriorly the lenticular zone is in connection with Wernicke's zone (supra-marginal gyrus; angular gyrus, posterior portions of the T'

and T″). A lesion of Wernicke's zone produces aphasia, a lesion of the quadrilateral area produces anarthria. The F^3 which lies in front of the quadrilateral area has nothing to do with aphasia (Fig. 71).

Since Marie's original contribution various observers reported anatomical cases corroborating his views. On the other hand, Dejerine and others brought forward facts which are at variance with the new conception of aphasia. The subject is still debatable.

APRAXIA

Under this name is understood an inability to use the limbs for purposive movements in spite of absence of paralysis or of ataxia. The apraxic individual understands the command and possesses the knowledge of objects. Nevertheless he uses improperly objects presented to him and executes improperly orders given him.

Normally to fulfill an order it is necessary to understand it. The execution of the act necessitates the following conditions: (1) mental representation of the act. To accomplish the act it is necessary to execute a series of movements. Therefore the realization of the principal or final idea is associated with or dependent on secondary or intermediary ideas. The latter constitutes the **kinetic formula.** For example, to light a cigarette necessitates taking out a match, lighting the latter, put the cigarette in the mouth, etc. To accomplish the act consequently one must have a mental representation not only of the final act, but also of secondary acts. (2) The psychic conception of the act and the kinetic formula must be followed by kinetic images which means beginning of realization. (3) Finally the kinetic images are followed by motor phenomena which finally ends in execution of the act. Otherwise speaking, there are three elements in execution of an act: ideational element, transformation of secondary ideas into kinetic images and a motor element. When the first element is disturbed, there is some defect in the superior mental functions, viz. attention, memory and association of ideas. The patient is incapable to understand the details of the act. He has, therefore, **ideational apraxia**. When the second element suffers, the limbs cannot obey the will. The patient is unable to transform the plan of the act into motor images. This is **ideational-motor apraxia** or **kinetic apraxia** of Liepmann. When the third element is involved, the patient is incapable to execute correctly the movements. Evidently the process of innervation is inaccurate, the projection centers suffer. This is **motor apraxia** or cortical apraxia.

Clinically the following manifestations are observed (taking for a type

the ideational-motor apraxia: (1) Substituted movements. Example: patient opens the mouth when told to close the eyes. (2) Purposeless movements. Example: execution of an act having no relation to the command. (3) Absence of movements without regard to the order.

In examining for apraxia the following movements are to be investigated: (1) Walking, sitting down or getting up (autokinetic movements). (2) Simple movements: close the eyes, open the mouth, put out the tongue, raise an arm. (3) Show the right or left eye or ear, comb the hair. (4) Salute in a military fashion, make the movement of a kiss. (5) Make the movement of catching a fly. (6) Fill a glass with water, seal an envelope. This procedure devised by Laignel-Lavastine and Boudon (*Revue Neurol.*, 1909) is systematic and therefore very useful.

The study of apraxia has been put on a solid scientific basis by Liepman in 1900. He has brought forward the following important anatomo-clinical facts.

Ideational-motor apraxia is on the side opposite the lesion, but it may also be observed on the same side when the lesion is in the left hemisphere. The preponderant influence of the left hemisphere on the functions of speech and of all voluntary movements is carried through the corpus callosum. By means of the latter the left sensori-motorium transmits its influence on the right sensori-motorium. To obtain apraxia of the left hand in a right hemiplegic it is necessary to have a sub-cortical lesion affecting simultaneously the projection fibers for the right limbs also the fibers of the corpus callosum destined to be distributed in the right senso-motorium which controls the left limbs. A lesion of the left internal capsule will produce a right hemiplegia without left apraxia, while a lesion only of the corpus callosum will result in left apraxia without a right hemiplegia or apraxia.

Motor apraxia is always on the side opposite the lesion. Some authors believe that it is due to superficial changes of the cortex.

Ideational apraxia is associated with a suspension or a real dificit of mental faculties. An ideational apraxia occurring in a hemiplegic is an important indication of eventual dementia.

CHAPTER VIII

HEMIANOPSIA

In a separate chapter will be described ocular disturbances occurring in diseases of the entire nervous system. Special mention, however, deserves an ocular phenomenon which occurs in cerebral affections and which it is important to recognize. This is **hemianopsia.**

Under this term is understood a complete or incomplete blindness of one half of the visual fields. When the corresponding halves of both eyes are affected, the hemianopsia is **homonymous** and may be right or left, superior or inferior. As the latter two are comparatively little known, they will be omitted in our study. In the majority of cases the hemianopsia is lateral homonymous. When the blindness of the visual fields occurs in one half of one eye and in the opposite half of the other eye, the hemianopsia is then **heteronymous.** It is **nasal,** when the right half of the left and the left half of the right visual fields are blind. It is **temporal,** when the right half of the right and left half of the left visual fields are blind. Anatomically the occipital lobe and particularly the calcarine area are concerned in half vision. Embryological investigation of Flechsig show that the optic radiations which end in the calcarine area become medullated long before other fibers which are distributed in the occipital lobe.

In order to understand the mechanism of hemianopsia it is necessary to bear in mind the entire visual pathway. The latter consists of: (1) optic nerves, (2) chiasma,(3) optic tracts, pulvinar, external geniculate bodies and anterior quadrigeminal bodies, (4) optic radiations and cortex of the occipital lobes. (See Anatomy and Localizations).

The decussation of the optic nerves in the chiasma is not complete, so that each optic tract posteriorly to the chiasma contains **direct** fibers, which pass to the optic nerve of the same side and become distributed in the temporal side of the retina, also **crossed** fibers, which are distributed in the nasal side of the retina of the opposite side.

With these anatomical facts in view, it is easy to conceive the mechanism of hemianopsia. A lesion situated in the anterior or posterior angle of the chiasma will interrupt fibers distributed to the nasal halves of both retinæ and consequently will cause blindness of both temporal halves of the visual fields, viz. **bitemporal** hemianopsia. A lesion affecting

the right optic tract, for example, will involve the direct fibers going to the temporal side of the right retina and the crossed optic fibers going to the nasal side of the left retina; the symptom will be a homonymous left hemianopsia, otherwise speaking a blindness of the left visual field of both eyes. The same form of hemianopsia will be observed in lesions of the optic radiations and of the occipital lobe, especially in the calcarine fissure, cuneus and lingual lobule. Although the anterior quandrigeminal body and external geniculate body belong to the optic centers, there are, however, no exact data showing that a lesion of these bodies produces a homonymous hemianopsia. In order to emphasize the relation of the side of the lesion to that of the hemianopsia, I will call attention to the analogy which exists between this phenomenon and the motor and sensory disturbances in affections of one cerebral hemisphere. Each hemisphere is in control of the opposite side of the body. In hemianopsia therefore the seat of the lesion is on the side opposite to the blind visual field.

From a clinical standpoint two problems must be investigated. First is to determine the hemianopsia. Second is to localize it. In the first case the patient will frequently call attention to the lateral loss of vision. Each eye must be examined separately and in the majority of cases the hemianopsia is easily revealed. The next question is to determine in what portion of the long visual pathway lies the lesion. This can be ascertained from the reaction of the pupil.

As is well known, the pupillary fibers of the optic nerve go through the anterior quadrigeminal body. From the latter a system of fibers connect the former with the nucleus of the third nerve which controls the pupillary sphincter, which is located in the gray matter of the aqueduct of Sylvius. A lesion therefore in front of the quadrigeminal bodies will interrupt the connection between the pupillary fibers of the optic nerve with the nucleus of the sphincter of the pupil. In this case there will be a homonymous hemianopsia with no response of the pupil when a light is thrown into the latter on the blind side of the visual field. A lesion placed posteriorly to the quadrigeminal bodies will consequently not interfere with the pupillary reaction. The inaction of the pupil under the circumstances just described is known as **"hemianopsic pupillary reaction "**or **"Wernicke's pupil."**

Homonymous hemianopsia has this characteristic feature that the vision central is always preserved. The reason of it lies in the fact that the macula of each eye is in anatomical relation with both optic tracts and both cortical visual centers by means of a direct and crossed bundle of fibers. Even in some cases of double hemianopsia caused by bilateral

lesions in the occipital lobes central vision is reported to be normal. Such were the cases of Förster (*Arch. f. Ophthalm.*, 1890) and of Beevor and Collier (Brain, 1904).

Cortical homonymous hemianopsia presents this peculiarity that it is very frequently associated with hemiplegia and hemianæsthesia, because the destructive lesion in the occipital lobe and optic radiations which causes the hemianopsia frequently extends into and implicates the internal capsule. When the lesion affects also the cortex of the angular gyrus, in addition to hemianopsia word-blindness will be present.

To sum up the study of homonymous hemianopsia, one can say that it is caused by lesions of the visual pathway between the chiasma and the occipital cortex. As to the optic nerves in front of the chiasma, in view of their anatomical composition (see above) the hemianopsia may be **monocular,** when only a half of one optic nerve is involved.

Up to now only unilateral hemianopsia was considered. Hemianopsia may be **double.** The patient is then totally blind. Such cases are usually the result of bilateral cortical lesions. It is characterized by the integrity of the fundi and absence of Wernicke's pupillary reaction. Curiously enough the central vision is preserved or rapidly reëstablished, probably because of the relation of the macula lutea to both cortical visual zones (see above).

CHAPTER IX

TUMORS OF THE BRAIN

The brain is a frequent seat of growths in children and adults. They may originate in the cerebral substance, in the meninges or in the cranium itself. In view of the great resistance of the skull to expansion of intracranial neoplasms, the brain is always under pressure irrespective of the point of origin of the tumor. Disturbances of mechanical nature are therefore most prominent in tumors of the brain.

Pathology.—In studying the pathology two elements are to be considered: (1) the tumor itself and (2) the condition of the cerebral tissue.

(a) **Varieties of Tumors.**—In order of their frequency they are: gliomatous, sarcomatous, tubercular, syphilitic (gumma), endotheliomatous, vascular, cystic and parasitic (echinococcus and cysticercus), carcinomatous, cholesteatomatous, osteomatous.

Glioma.—It is a soft and very vascular tumor. It is never encapsulated, but continuous with the neighboring cerebral tissue: the brain substance is, so to speak, infiltrated with the soft gliomatous tissue. For this reason the exterior aspect of the cortex undergoes very little change; there is only a distinct softness of the brain. The most frequent seat of glioma is the white substance beneath the cortex.

Histologically it is essentially composed of neuroglia tissue. The latter presents its characteristic appearance, viz. cells with their large nuclei, isolated or in masses. When in addition to them there are also connective tissue elements, the tumor is called gliosarcoma. In rare cases (probably congenital) a hyperplasia of the cortical elements is seen besides abundant neuroglia and vascular tissue. It is called the **neuro-glioma.** Glioma is seen more frequently in middle life than in old age.

Sarcoma.—It is a soft tumor, but distinctly harder than glioma. It presents this characteristic feature, that there is a sharply defined line of demarcation between it and the brain tissue and it is therefore easily separable from the latter. It is frequently encapsulated. It is reddish and of spheric form. Histologically it consists essentially of round or spindle cells; it is not vascular.

In the majority of cases sarcomata originate in the dura-mater, periosteum or skull. They develop frequently at the base of the brain.

There is a variety of sarcomata characterized by a proliferation of connective tissue. They are called fibro-sarcomata. Their multiplicity and localization all over the central and peripheral nervous system, particularly in the cerebello-pontine region, also in the roots of the spinal cord and cauda equina, are the characteristic features of these tumors. Sarcoma occurs particularly in middle life. A very malignant form is **melanotic sarcoma** which is a secondary tumor.

Tubercular Tumor.—Solitary tubercles belong to the most frequent growths of the brain. Unlike glioma they have a remarkable tendency to become encapsulated. They are generally of the size of a cherry and of a spheric form. Their usual seat is in the most vascular areas, viz. in the fissure of Sylvius, at the base of the brain, in the vicinity of the pons and in the interpeduncular space. The most common seat is around the Pacchionian bodies (see Anatomy). They are found more frequently near the meninges than within the cerebral tissue. The cerebellum is a favorite seat of tubercles in children. There is frequently more than one tumor. On section no vessels will be seen in the center, which is in a caseous state. The granular tissue of which the tubercule is composed is formed at the expense of the perivascular sheaths and of the neuroglia tissue. The tubercle bacillus is very frequently found. Tubercular tumors may produce tuberculous meningitis.

Syphiloma (Gumma).—These tumors are frequently found in adults. Small in size (chestnut), irregular and nodular in shape, firm in some parts and soft in others, they are most frequently located at the base of the brain and quite often also in the cortex, especially in the anterior portions of the hemispheres. At the base they inevitably involve the cranial nerves or the large blood vessels, the obliteration of which naturally leads to a softening of cerebral tissue. The meninges are usually involved (inflammation, adhesion) so that it is difficult to tell where the growth originated: in the brain tissue or membranes. There are often several gummata. Very small gummata are sometimes found around the blood vessels; they resemble then miliary tubercles, but the latter are generally extremely small. On section they present irregular cheesy spots, between which is seen fibrous tissue. Like the tubercle, syphiloma is not vascular.

Endothelioma usually originates in the dura and is single. It occurs at any age.

Vascular Tumors.—**Angiomata** are rare, but **aneurisms** are not very rare. They are usually found on the bents or curves which the arteries form in their course. Syphilis is probably the cause of cerebral aneurisms. Statistics show that the basilar artery is the most frequently affected and the next in frequency is the middle cerebral.

Cystic.—Besides cystic formations developed in place of old hemorrhagic foci or areas of softening (see Apoplexy), there are also some which develop in connection with sarcomata or gliomata. When any of these tumors breaks down and the débris are carried off, a cavity surrounded by walls composed of the elements of the growth takes its place and thus a cyst is formed. These formations usually occupy the ventricular cavities and are also found in the cerebellum.

Occasionally the cerebral substance or the meninges are the seat of cysts containing parasites, viz. **cysticercus** and **echinococcus.** The cysticercus is found in the meninges and particularly in the area of the perforated spaces. The latter fact is the reason of the ocular phenomena observed during life. The echinococcus is observed especially in certain countries, Australia for example. It usually forms adhesions with the meninges and it may be eliminated through the natural openings of the cranium.

Carcinoma.—In the majority of cases it is secondary to carcinoma of other organs of the body. It is comparatively frequent in the ventricular cavities or in the walls of the ventricles. It is vascular and rarely encapsulated. It is usually multiple. It occurs mostly in aged people. **Cholesteatoma and Osteoma** are very rare. The former is easily recognizable by its brilliant appearance.

(b) **Effect of Tumors on Brain Tissue.**—Cerebral tumors have a direct effect upon the neighboring nervous tissue and a distant effect upon all parts of the brain. Direct pressure produces displacement of the brain toward the point of least resistance, which is usually the ventricles, flattening of the convolutions and destruction of nerve elements. The meninges are tense. The area of softening which is usually seen around the neoplasm is due to the destruction of the nerve tissue. The latter process is an irritative process, which eventually leads to an inflammation. Distant pressure is sometimes the cause of meningitis, which is observed in the course of brain tumors. The cerebro-spinal fluid is habitually increased, so that the entire brain is wet (**œdema**) and internal hydrocephalus may be produced. The latter condition is particularly marked in tumors situated in the vicinity of the openings connecting the ventricles, as, for example, near the middle lobe of the cerebellum or the quadrigeminal bodies. When pressure from the tumor or from the hydrocephalus is produced toward the base of the brain, the cranial nerves are involved.

The increased intracranial pressure produces an obstacle to the free cerebral circulation thus causing an increase of the intracranial venous pressure. The latter has a direct effect on the cerebro-spinal fluid thus

rising its pressure. As the increase of this fluid is not only in the cranium but also in the spinal canal, the arachnoid of the latter becomes distended and the posterior nerve-roots are thus over-extended. Degenerative changes of these roots and of the posterior columns are not infrequently met with in cerebral tumors (Fig. 72).

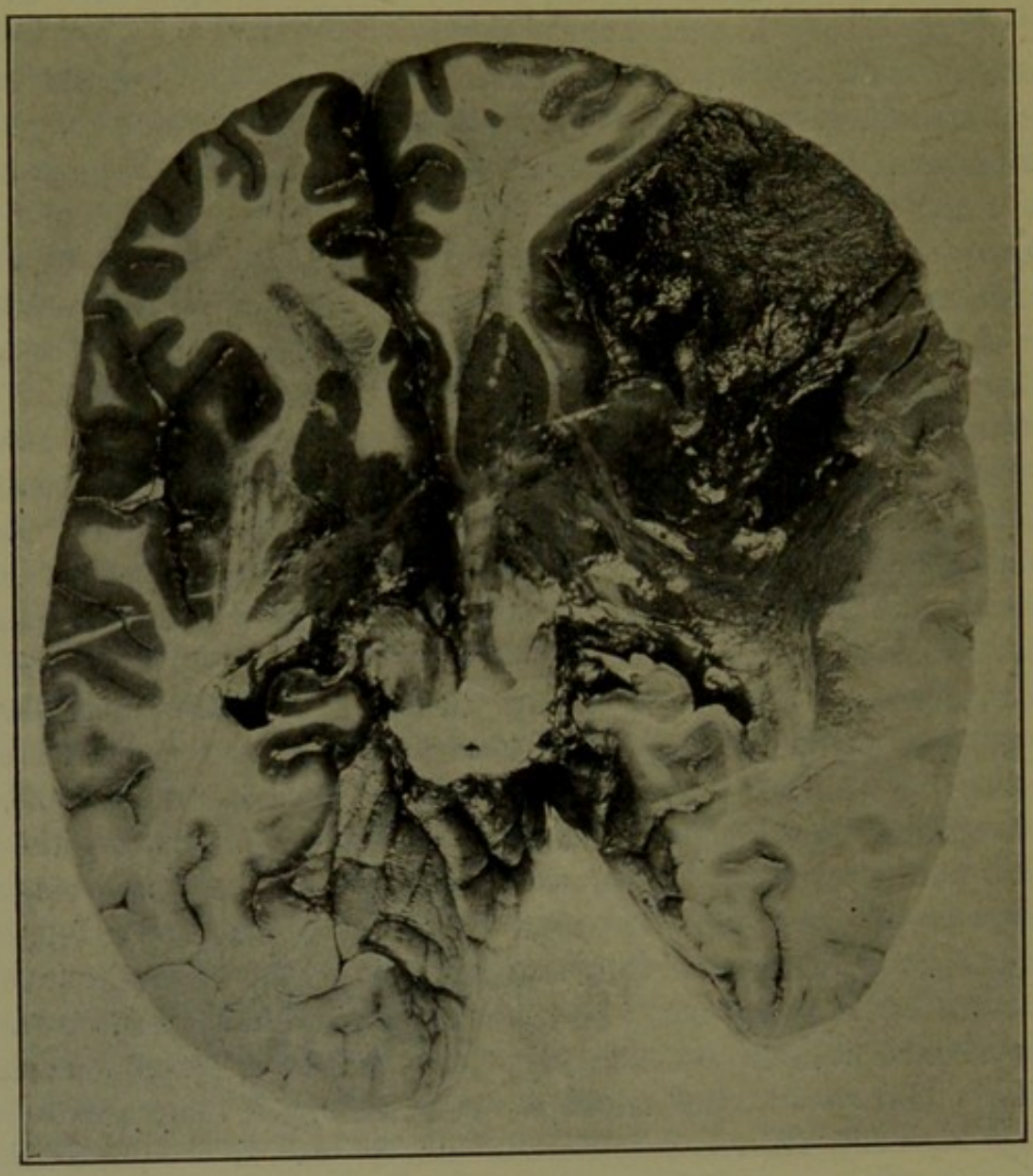


FIG. 72.—TREMOR (ROUND-CELL SARCOMA.) WEDGED IN BETWEEN THE L. FRONTAL AND TEMPORAL LOBES, PENETRATING THE LATERAL VENTRICLE. (*Original.*)

Symptoms.—They are (A) **general** and (B) **local.**

A. The **general** symptoms are common almost to all forms of tumors irrespective of their seat and nature. They are: headache, vomiting, vertigo, optic neuritis, general convulsions, insomnia, mental failure with depression.

Headache.—This is one of the earliest symptoms. It has no definite

seat and it rarely corresponds to the seat of the tumor. It is deep and persistent; it is usually continuous and sometimes excruciating. It may become aggravated upon slight pressure with the hand (which suggests an involvement of the skull or of membranes) or upon a slight motion of the head, upon coughing or forced respiration. It is usually progressive in intensity in the course of the disease and not relieved by ordinary drugs. In many cases it starts in the morning and then it is accompanied by vomiting. Sometimes it is worse at night and then syphilis should be thought of, although specific infection is not invariably the case. The cause of the headache lies probably in the irritation of the meninges due to increased intracranial pressure. **Vomiting** is characteristic by the facility with which it is brought on and by its occurrence irrespective of food. This is the so-called "cerebral vomiting." They usually coincide with periods of exacerbation of headache. **Vertigo** is constant, but particularly marked in cerebellar tumors.

Optic Neuritis.—Circulatory disturbance of the retina is a very frequent occurrence in cerebral growths (80 per cent.). It may result in **optic neuritis** or **choked disc.** The papilla is in a stage of œdematous infiltration with venous stasis very probably as the result of increased intracranial tension. Neuritis is rarely unilateral. One-sided optic neuritis is more frequently met with in cerebral than in cerebellar tumors and when it occurs it is frequently on the side of the tumor; it is therefore of localizing importance. A certain degree of optic neuritis may exist for weeks and months without appreciable impairment of sight. When the neuritis becomes progressive or choked disc is beginning to form, limitation of visual field will be noticed and gradual blindness will develop. The natural and ultimate consequence of optic neuritis is **white atrophy** and consequently amaurosis. In a small percentage of cases loss of vision occurs before atrophy of the optic nerve develops. Cushing, Bordley and Heuer have recently shown that alterations of color fields (inversion) frequently occur in intracranial tumors as an early manifestation.

The following peculiarities were noticed by them: (1) A tumor may advance slowly and be unaccompanied by cerebral œdema or other pressure symptoms; distortion of the color fields (dyschromatopsia) may be the earliest indication of the latter and observed on the side opposite to the lesion. (2) Unilateral dyschromatopsia accompanied by an incipient degree of choked disc may be on the side of the lesion and is therefore of localizing value; if dyschromatopsia is bilateral, the higher degree of it is on the side of the lesion. (3) Dyschromatopsia is a more delicate manifestation of intracranial pressure than papillœdema.

Convulsions.—Epileptiform generalized convulsions are not infrequent

in brain tumors. They are due to an irritation of the cerebral tissue. They usually appear unexpectedly, but never at the beginning of the disease. They have the character of genuine essential epilepsy, viz. generalized. According to Hirt's statistics this form of epilepsy occurs in 50 per cent. of cases of brain tumor. There are cases on record in which epilepsy was for a long time the only symptom of brain tumor. Consequently in a given case of epilepsy occurring late in life without an apparent cause, cerebral neoplasm should be thought of. When the convulsions are focal but not generalized, they have a localizing value (see below).

Insomnia.—Sleeplessness is quite frequent. It is probably due to the continuous severe headache. It can also be explained on the basis of circulatory disturbances in the brain. It is more frequent in adults than in children.

Mental Disturbances.—Hebetude, apathy, indifference, impairment of memory, depression or else irritability are all symptoms which may be encountered. Occasionally symptoms of true dementia with hallucinations or delusions may develop. The latter form is particularly present in tumors of the frontal lobe. Stupor is usually a late symptom. Somnolence is frequent. Whenever the patient is free from suffering, he goes to sleep In a boy of twelve, with a cerebellar neoplasm, I observed an unusual tendency to sleep, so that during the last eight days he slept continuously except for a few minutes during the day to take nourishment. In the last twenty-four hours he could not be aroused and gradually expired. Intense somnolence is also observed in tumors of pituitary body. In tumors of corpus callosum Raymond observed eccentricity of actions, impairment of memory and apparent integrity of intelligence.

B. **Local Symptoms. Tumors of the Antero-superior Portion of the Frontal Lobe.**—While mental disturbances may occur in tumors of any portion of the brain (see preceding chapter, also chapter on Localization of Center of Intelligence), it is nevertheless averred that they are more frequent, more precocious, more pronounced in tumors of the prefrontal area than in those of any other portion of the brain. Consequently when they appear early they have a localizing value. As to the character of the psychic disturbances it is impossible as yet to draw positive conclusions. The carefully collected records, however, show that the main symptoms are: change of character, inability to concentrate attention, incoherence in speech, deficiency of orientation and of discrimination. Jastrowitz (*Deut. med. Wochenshr.*, 1888) described a special mental manifestation which he called "moria." It consists of an emotional gaiety, of an unusually lively spirit. The author claims that he observed it frequently in prefrontal growths. (See also my article on the Function

of the Prefrontal Lobes in *J. Amer. Med. Ass.*, 1907.) In tumors of this area are sometimes also observed paralysis or localized convulsions on the side opposite to the lesion. These symptoms occur when the tumor is deep seated, so that it involves the short commissural fibers connecting the prefrontal lobe with the motor area or when the irritation of the prefrontal cortex extends backward to the cortex of the motor area. In such cases there will be changes in the deep and superficial reflexes (see chapter on Reflexes). Usually in prefrontal tumors there is only a paresis of the opposite side of the body. In cases of localized convulsions a paretic condition will follow on the affected side. G. Stewart observed in a few cases a fine intention tremor of the head on the side of frontal tumor. The same author calls attention to a loss or diminution of the abdominal reflex on the side opposite to the tumor.

Tumors of the Antero-inferior Portion of the Frontal Lobe.—When the second frontal convolution is involved the characteristic focal symptom is **agraphia.** When the third frontal convolution is affected, the symptoms will be motor **aphasia.** It is to be remembered that in right-handed people aphasia and agraphia will be caused by tumors of the above area in the left hemisphere, while in left-handed people the lesion will be in the right hemisphere (see chapter on Localizations). Tumors of the frontal lobe compressing the olfactory bulb or situated on the basal surface of the frontal lobe are associated with loss of smell in the nostril on the side of the tumor.

Tumors of the Motor Area.—In view of the motor function of this area (see Localizations) a tumor developing gradually and irritating it will give rise to motor symptoms in the form of tonic or clonic convulsions limited to one side of the body. They are known as focal or Jacksonian epilepsy (see this chapter) In this form of epilepsy consciousness is usually intact. The seat of the spasm (face, arm or leg) will depend upon the seat of the tumor in the three distinct portions of the motor area (see Localizations). It happens not infrequently that the convulsions commencing in one arm, in one leg, in one-half of the face or in the distal end of one limb (thumb, fingers, toes) spread rapidly to the rest of the body on the same side or to the entire body. Sometimes convulsions affecting one-half of the body are preceded by a paræsthesia in one limb, as, for example, a tingling sensation, numbness or pins and needles. These strictly localized phenomena are of utmost importance for diagnostic purposes: the order of extension of the spasm is invaluable for localizing the initial irritation and consequently the seat of the tumor. The presence of a sensory aura indicates the seat of the tumor in the neighborhood of the ascending parietal or the parietal convolutions.

Tumors of the motor area produce also paralytic symptoms, but they usually come on later in the course of the disease. They are generally **monoplegic,** but may become hemiplegic. Convulsions are usually associated with paralysis, but there are cases of brain tumor with a gradually developing paralysis without convulsions A progressive paralysis, viz. monoplegia slowly developing into hemiplegia, is almost pathognomonic of cerebral tumor. In such cases the tumor is situated in the sub-cortical white matter, viz. in the motor fibers uniting the cortical motor area with the internal capsule. When the tumor originating in the sub-cortical tissue continues to grow and reaches the cortex, the patient will begin the disease with paralytic symptoms and later develop convulsions. Hemiplegia may set in suddenly. It is then an indication of a hemorrhage into the tumor or in the surrounding tissue.

In tumors of the motor area with paralytic symptoms (hemiplegia or monoplegia), the usual signs of paralysis of cerebral origin are observed (see Hemiplegia). In cases of focal epilepsy without paralysis the reflexes of the affected side are usually increased and especially immediately after each attack. The paradoxical reflex (see chapter on Hemiplegia) is particularly persistent immediately after as well as between the individual attacks. This reflex therefore is valuable as it is indicative of a localized cortical irritation.

Tumors of the Ascending Parietal Convolution and of the Parietal Lobe.—The most conspicuous symptoms are disturbances of general sensations (**hemianæsthesia**) and particularly of muscular sense on the opposite side of the body. The impairment of sensations is more marked in the distal than in the proximal ends of the limbs. For details and for the study of **astereognosis** the reader is referred to the chapter on Localizations. A tumor seated in the posterior portion of the inferior parietal lobule (angular gyrus) of the left hemisphere will give rise to **word-blindness** (**alexia**). In case a parietal tumor will encroach upon the motor zone, in addition to the above symptoms, there will be also paralysis. In parietal tumors Jacksonian fits occur, but always commencing with a sensory aura. If the parietal tumor extends backward so that the occipital lobe becomes involved, there will be also hemianopsia.

Tumors of the Occipital Region.—The chief focal symptom of tumors in this area is lateral homonymous hemianopsia. For details see chapter on Localizations and Hemianopsia. Frequently there are also subjective visual disturbances, such as flashes of light, of colors. They are due to an irritative state of the cerebral tissue. When the tumor is on the lower surface of the occipital lobe so that pressure on the cerebellum will be present, cerebellar manifestations will follow. When a tumor of

the occipital lobe extends forward so that it encroaches on the angular gyrus, in addition to the hemianopsia there will also be alexia. Convulsions, general or focal (on the opposite side) occasionally occur. Paralysis on the opposite side also occurs when the tumor extends forward.

Tumors of the Temporal Lobe.—The function of hearing is associated with the temporal lobe (see Localizations). A tumor of the left first temporal lobe in right-handed people and of the right first temporal lobe in left-handed people gives rise to **word-deafness** (see chapter on Aphasia). In a specimen exhibited by me before the Philadelphia Neurological and Pathological Societies in April and May, 1906, the left first temporal convolution was extremely thin and narrow. The patient presented during life word-deafness: he heard sounds, but could not understand spoken words.

Tumors of the apex of the temporal lobe are believed to cause disturbances of **taste and smell.**

Hughling Jackson in 1876 described attacks consisting of a "dreamy state" preceded by peculiar sensations of taste or smell. During the attack the patient has a feeling of unreality and recalls events that happened long ago. At the same time he keeps on moving his mouth and jaws as if he chews. These seizures are called **"uncinate fits,"** and are observed in tumors of the tip of the temporo-sphenoidal lobe.

Tumors at the Base of the Brain.—In view of the presence of the cranial nerves at the base of the brain (see Anatomy), the symptomatology of basal tumors is somewhat complex. The characteristic feature of such conditions is **crossed** or **alternating** paralysis. It consists of hemiplegia affecting the side of the body opposite to the side of the lesion and of a palsy of one or more cranial nerves on the side of the lesion. It finds its explanation in the fact that a tumor lying on one side involves the motor and sensory fibers which have not yet decussated (see Anatomy), also the superficial origin of the cranial nerves which, of course, do not undergo decussation at the base. Should the tumor occupy the middle of the base, bilateral paralysis will necessarily ensue. It is superfluous to describe the symptoms of involvement of each cranial nerve. The deductions present no difficulty, if their anatomy and physiology are taken into consideration. For example, a tumor in one of the crura involves the third nerve; a tumor in the upper half of the pons involves the third and fifth nerves. In the first case there will be a crossed hemiplegia with external strabismus or ptosis on the side of the lesion; in the second case, also crossed hemiplegia with third nerve symptoms and sensory disturbances of the face on the side of the lesion. See also chapter on Bulbar Paralysis. Tumors of the very anterior portion of the basal

surface of the brain, viz. of the inferior surface of the frontal lobes, will give rise to early visual disturbances and to olfactory manifestations. They find their explanation in compression of the optic and olfactory nerves.

Tumors of the Quadrigeminal Bodies.—In view of the anatomical relation of corpora quadrigemina to the cerebellum and to the motor nerves of the eyes, the symptoms will be those observed in cerebellar

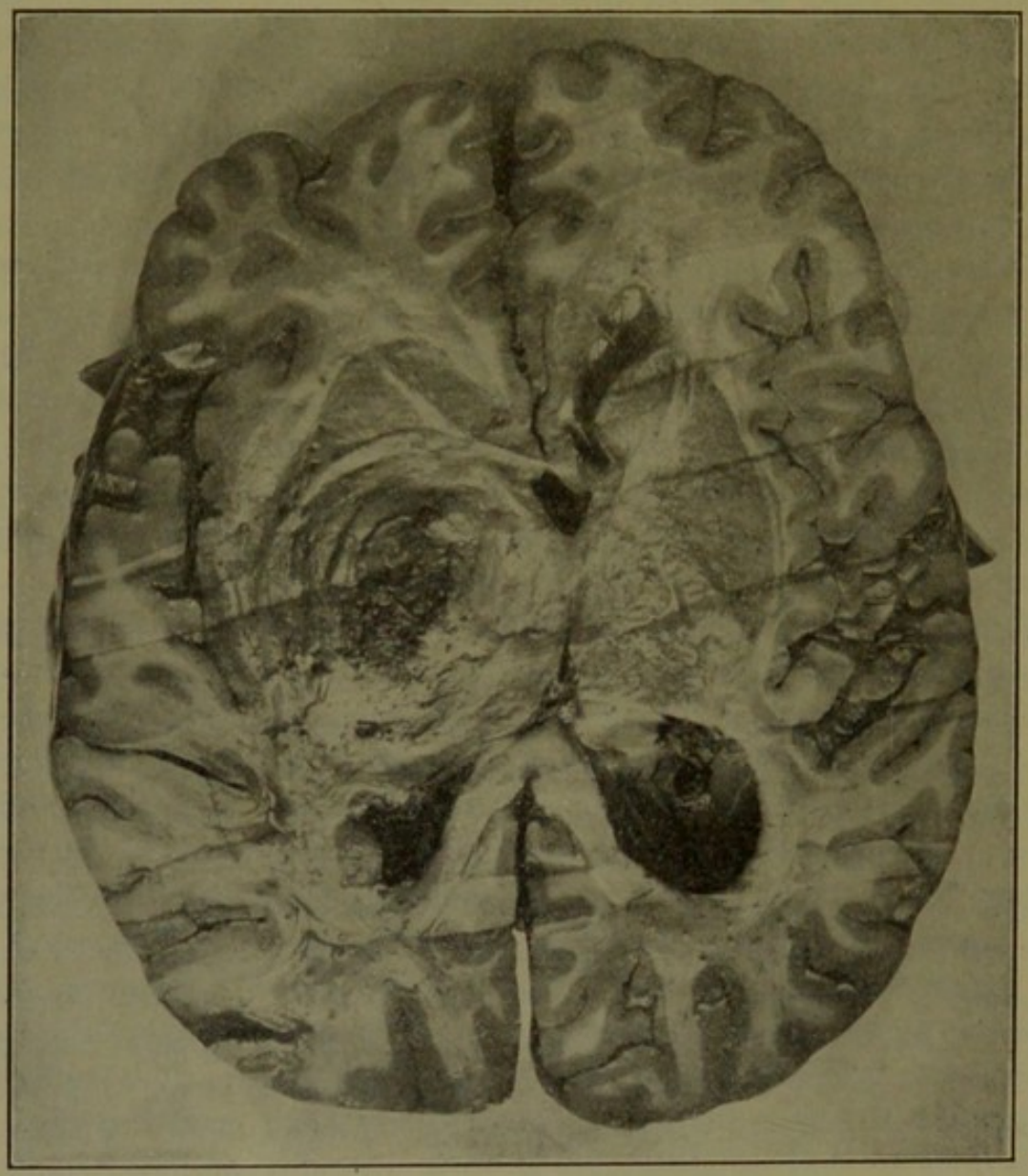


FIG. 73—TUMOR OF THE BASAL GANGLIA (MIXED-CELL SARCOMA). (*Original.*)

diseases and opthalmoplegia. The first are: titubation or staggering gait and incoördination in standing; the latter is bilateral and external. Nystagmus is also a frequent symptom (see details on pages 213, 214, 215).

Tumors of the Basal Ganglia and of the Corpus Callosum.—The function of **basal ganglia** is unsettled. In diseases of these ganglia the

symptoms present nothing pathognomonic. There are also cases on record in which tumors were not suspected and surprisingly found in this region. However, in view of the close proximity of the internal capsule (see Anatomy), pressure symptoms of the latter will frequently be present, namely hemiplegia, heminanæsthesia and hemianopsia, according to the segment of the capsule involved (see Diseases of Basal Ganglia and Fig. 73).

Tumors of Corpus Callosum. Symptoms.—The general symptoms observed ordinarily in tumors of the brain, viz. headache, vomiting, vertigo, optic neuritis and convulsions, are only exceptionally met with in tumors of corpus callosum. **Special symptoms: Mental apathy,** indifference are very early manifestations. The diminution of activity may progress to absolute immobility. Somnolence is another frequent symptom. Progressive inability of concentrating thoughts, lack of attention, increasing depression, progressive diminution, of mental faculties may gradually terminate in **dementia. Amnesia** may be observed. Confusion, delirium and delusions, with hallucinations have been observed.

Motor Paralysis and Contractures.—Hemiplegia is the most frequent symtom, but what is particularly characteristic is its **progressive development.** The face is usually spared. Flaccidity of the limbs is the most frequent, contractures are observed in the minority of cases. Bilaterality of paralysis is less frequent than unilaterality. Monoplegia, paraplegia, hemiplegia on one side and monoplegia on the opposite side may all be observed.

Tremor, Choreiform movements are seen sometimes. **Ataxia,** Disturbance, of equilibrium and of gait have been observed in some cases. The tendon **reflexes** are exaggerated in the majority of cases. The toe phenomenon is occasionally seen. **Sensations** are rarely involved. Disturbances of **speech** are frequent. They may simulate the speech of paretics; dysarthria, aphasia, staccato speech have all been observed. A muttering speech or speech of intonation (Brissaud) is sometimes present; the patient moves his lips, but does not make sounds. Apraxia is a very serious manifestation. Its relation to the corpus callosum has been established by Liepmann (see chapter on Apraxia, page 138). The **sphincters** are not rarely involved. **Cranial nerves** are usually not involved. Occasionally ocular disturbances are observed.

The clinical localizations of the symptoms are, according to Schupter and Duret, as follows:

(1) **Anterior portion of corpus callosum:** psychic disturbances, speech symptoms and motor disorders.

(2) **Middle portion:** stupor, titubation, paretic condition of the extremities.

(3) **Posterior portion:** disturbance of gait, paresis of the lower extremities, cerebellar symptoms.

Course and Duration.—The majority of writers believe that the onset is acute and the disease progresses rapidly, especially the mental symptoms.

The duration is usually of three or four months. There are however cases of longer duration. In such cases a careful investigation will almost always reveal a history of some ill-defined psychic manifestations, disturbances of affectivity and of character; some infectious process will become the exciting cause for the rapid evolution of the disease.

Pathogenesis.—The knowledge of apraxia (page 138) is a proof of the fact that the corpus callosum accentuates the well-known supremacy of the left hemisphere in the mechanism of thoughts. The left hemisphere accumulates certain impressions acquired by the right hemisphere. On other hand, it controls the acts executed by the right hemisphere; the latter acts, but the left hemisphere makes it act. There is a sort of double current traveling through the corpus callosum, viz. one from the right to left, the other from the left to right. In this double function one finds the explanation of both syndromes, viz. one is the mental state isolated by Raymond, the other is apraxia emphasized by Liepman.

Tumors of the Pituitary Body.—In diseases of the hypophysis cerebri two conditions may be observed: one is **acromegaly** and **gigantism** (see these chapters), the other is **adipositas** with or without genital hypoplasia. Hyperpituitarism is probably the cause of the former, hypopituitarism of the latter. The clinical symptoms of tumors of the hypophysis are, besides acromegaly and adipositas, also the following: bitemporal hemianopsia caused by pressure on the chiasma, headache, somnolence occurring paroxysmally, inability to concentrate thoughts and impairment of memory. Frequently, involvement of the third and sixth nerves is observed. Exophthalmos may occur in advanced stages when the tumor after destroying the sella turcica presses on the orbital cavity. Loss of smell may be present. The latter two symptoms occurred in one of my cases (*J. Am. Med. Ass.*, 1912). Hemiplegia or generalized or focal convulsions may also occur.

The adiposity together with sexual infantilism constitutes the so-called **Fröhlich's syndrome** or syndrome adiposo-génital. The patient thus affected keeps on laying on fat in spite of the exceedingly small amounts of food which he may consume. The genitalia are atrophied (testicles and

ovaries). Glycosuria and excessive sweating are not infrequently observed. They may be the result of the disturbed function of the hypophysis (Fig. 74).

Course. Termination. Prognosis.—The malignant tumors (carcinoma, sarcoma, etc.) run a rapid course. Solitary tubercle, glioma or osteo-

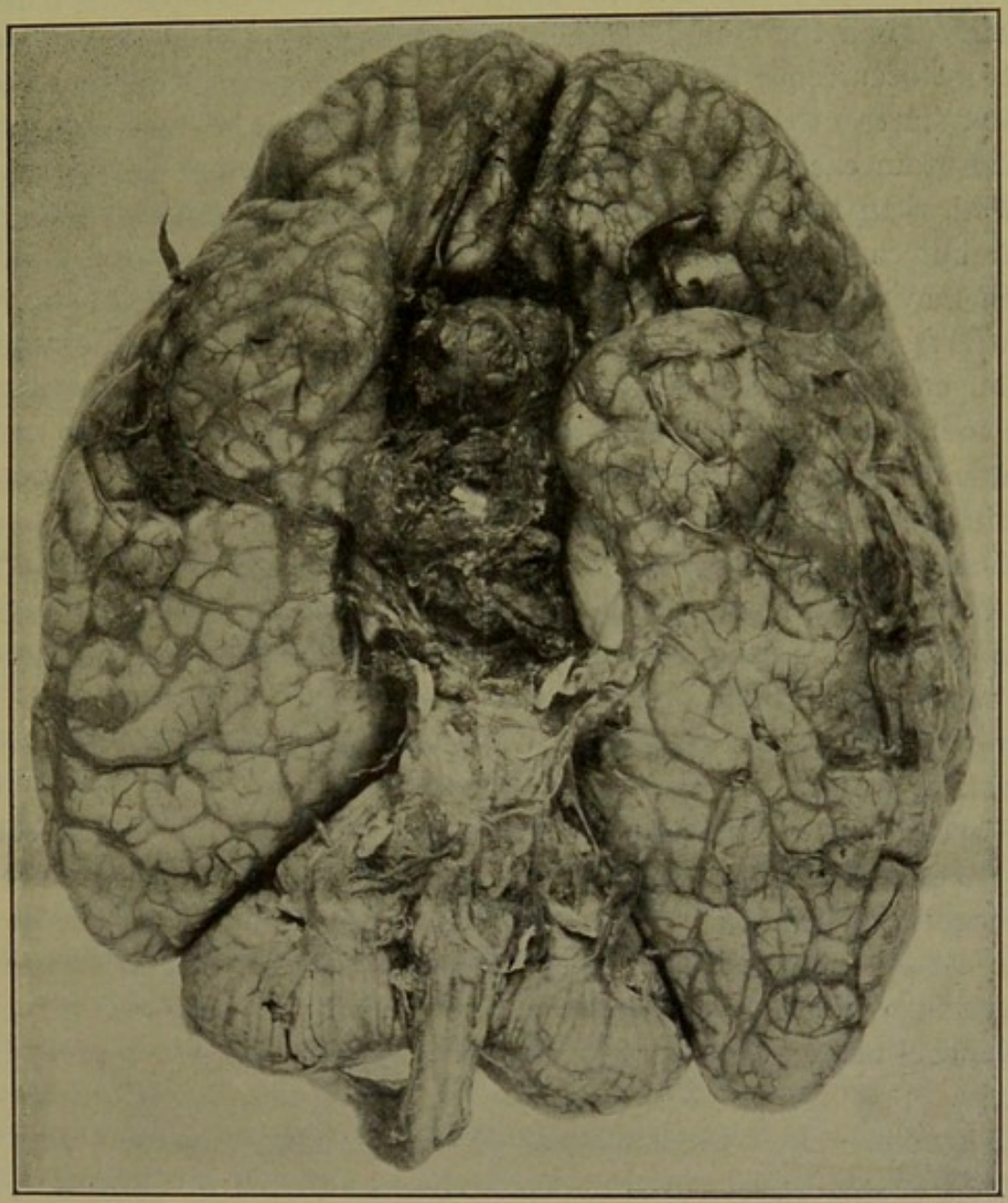


FIG. 74.—TUMOR OF HYPOPHYSIS, SHOWING TWO LOBES. Crura and Pons are Deformed (*Adipositas Cerebralis.*) (*Original.*)

sarcoma develop much more slowly than a sarcoma or carcinoma. The latter is fatal in two to three years and in children in two months. A cavernous angioma may persist for years. A tubercle may become stationary or undergo ossification or calcification. Young individuals bear tumors at the base of the brain better than adults owing to superior elasticity

and yielding capacity of bones and sutures of the skull. The development of cerebral tumors is as a rule inevitably progressive and death is the common termination. In some cases the evolution of a tumor may be very insidious and the symptoms very mild, so that diagnosis of hysteria, migraine, psychoneurosis, eye strain, dyspepsia is made. This may go on for years until an apoplectiform or an epileptiform attack occurs. Then the symptoms become pronounced and advance rapidly. Such an occurrence is possible when a tumor is located in the so-called "silent" area of the brain, under which term is understood any portion of the brain except the well-known regions, the function of which is well studied. In these cases a tumor may give rise to pressure symptoms alone but cannot be localized even if its presence is suspected. Sudden death may take place in the course of cerebral tumors. On the other hand arrest of development of symptoms is possible for a more or less prolonged period until an accident, an injury or some other cause will give an impetus to the silent tumor, which then begins to progress rapidly. Gummata when treated **energetically** run a different course: under this condition the symptoms may be greatly relieved.

Generally speaking, cerebral growths belong to the most serious affections. In a small group of cases in which strictly localized symptoms indicate the exact seat of the neoplasm, an operation may improve the condition considerably and even remove the symptoms. But even then recurrences are possible. The most favorable prognosis present gummata. Arrest of symptoms is observable in tubercular tumors, and according to some these neoplasms are capable of spontaneous retrogression. Tumors are liable to become softened, cheesy in center and reduced in size. They are likely to become encapsulated. In some cases spontaneous hemorrhages occur in the tumor thus checking its growth. The outlook in superficially situated tumors, such as in the meninges and cortex is more favorable than in deep-seated tumors.

Diagnosis.—Individual symptoms of brain tumors may be encountered in other affections, so that in order to make a diagnosis, the latter must be excluded.

Headache and vomiting occur in **migraine,** but the cerebral character of vomiting (see above) and the persistent character of the pain in the head will exclude the latter affection (see Migraine).

Abscess of the brain will be distinguished from tumor by rapid development of symptoms; the latter are very pronounced in abscess and gradually progressing in tumors. Optic neuritis is very rare in abscess, common in tumor. The etiological factors, as suppurative disease of

the ears or of neighboring tissues or cavities, are common in abscess (see also chapter on Abscess).

Chronic meningitis of alcoholic or syphilitic nature may be confounded with tumors. The identity of the optic nerve condition in both cases makes the diagnosis very difficult. This is particularly applicable to localized meningitis. When the meningitis is tubercular, the differentiation is easier, as optic neuritis is less frequent and the headache more severe than in brain tumors.

An error may be committed in making a diagnosis of **hydrocephalus.** The congenital character of the latter, the form of the head, the spastic palsy of two symmetrical extremities without convulsions will help to eliminate a cerebral neoplasm.

In order to differentiate from **cerebral hemorrhage or softening,** the etiological factors must be taken into consideration (see Apoplexy). Moreover absence of optic nerve changes is the rule in apoplexy.

The diagnosis between frontal tumor and **paresis** is sometimes difficult. Cases have been reported in which the mental phenomena of paresis were found in frontal tumors. In such cases the cerebro-spinal fluid may decide the diagnosis: lymphocytosis and increase of albumen are present in paresis, but very rarely in tumors.

In some tumors of the brain **epilepsy** may be the only symptom for a long time. The diagnosis is then very difficult. It is only when optic neuritis and headache make their appearance that the distinction can be made.

The most important rôle in cerebral tumors play the **localized** symptoms, when they develop gradually. It should not be forgotten that certain tumors may remain latent for a long time and be unaccompanied by the characteristic symptoms until a hemorrhage occurs into them. Then the paralytic or other focal symptoms will determine the diagnosis.

As to the **seat** of the tumor, the localizing symptoms already described for each individual area of the cortex, also for differentiation of cortical and subcortical growths, are to be taken into consideration.

The **nature** of the tumor can be determined with great difficulty. The patient's personal and family history should be investigated. The concomitant existence of cancerous, sarcomatous or other growths in other organs of the body, the existence of phthisis, a history of acquired syphilis, the positive or negative Wasserman reaction, all these informations are important factors in deciding the question of the variety of a given tumor. Improvement observed during the administration of certain drugs is valuable only to some extent in determining the nature of the tumor. Mercury and iodides, for example, have a beneficial effect

in syphilitic growths, but this is not absolute, as improvement has been also observed with the same drugs in gliomata or tuberculomata. The seat of the tumor has some bearing upon the determination of its nature. Thus tumors on the surface of the brain are frequently syphilitic, tuberculous or sarcomatous. In the subcortical tissue, mostly gliomatous. In the cerebellum, tuberculous. The duration is equally important: tuberculous and gliomatous tumors are long; syphilitic when not treated, also carcinomatous growths run a rapid course.

In recent years X-rays have been applied for diagnostic purposes in studies of localization of brain tumors. Although the results are not absolutely satisfactory in every case, they are nevertheless promising.

The determination of the nature of cerebral tumors presents particular difficulties in **parasitic cysts** of the brain (echinococcus). The affection is comparatively rare. According to the statistics of Cranwell and H. Vegas of Buenos-Ayres among 1.696 parasitic cysts of various organ only 36 were found in the brain, of which 27 were in children. The diagnosis of cerebral echinococcus is extremely difficult, as there are no pathognomonic manifestations. Parvu and Laubry (*Bull. de Soc. Méd., d. hôp.*, 1908) have observed that if the blood serum of individuals having the echinococcus in any other organ contain specific antibodies, the latter are absent in the cerebro-spinal fluid, but they will be present if the central nervous system is affected. Recently (*Press. Méd.*, 1911), Legry, Parvu and Baumgartner reported new proofs of the above observation. A positive deviation of complement in the cerebro-spinal fluid and negative in serum is pathognomonic of a parasitic cyst in the brain when a tumor is suspected and when no other cause could be accounted for.

Etiology.—The causes of brain tumors are, generally speaking, the same as in tumors of any other organ. **Trauma** has been considered for a long time as an exciting cause for development of intracranial growths. The symptoms of tumor may begin to appear shortly after the trauma or even years later. It is reasonable to presume that an injury plays the rôle of a provoking agent for a rapid development of a tumor which existed before the injury. It has been also proven that some tumors are of a congenital origin, as coexistence of tumors with cerebral malformations has been observed. There are good reasons to believe that **glioma,** which is met with mainly in young people, is of fœtal origin. Tubercular tumors are usually secondary to pulmonary tubercular affections. Syphilitic growths are chiefly acquired after an initial specific infection.

Treatment.—It should be the rule in every case of brain tumor to give the patient the specific treatment for a period of several weeks.

If the tumor is of specific character the patient may derive great benefit. In two of my very recent cases there was a remarkable retrogression of the headache, of vertigo and of the choked disc under the influence of large doses of potassium iodide (150 gr. t.i.d.) associated with mercurial

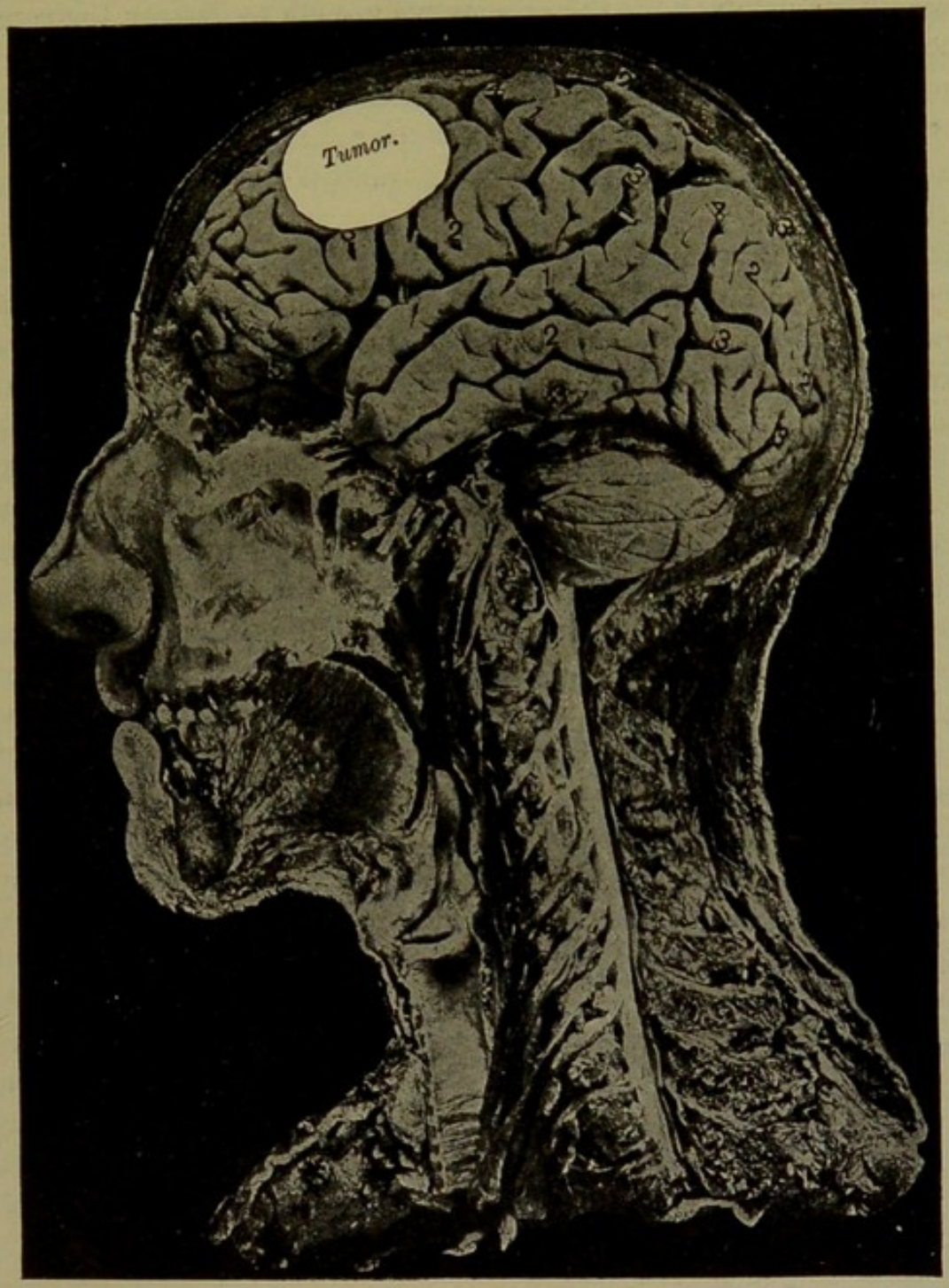


FIG. 75.—SITUATION OF A TUMOR, SHOWN FROM FRAZER'S GUIDE TO CEREBRAL OPERATIONS. The figure shows the relation of the convolutions to the head.

inunctions (℥j twice daily). On the other hand, some tumors, gliomata, for example, sometimes improve considerably under mercurials and iodides. The so-called **expectant** treatment, viz. the treatment with

antisyphilitic drugs for a period of weeks in the light of accumulated experience should be abandoned in spite of amelioration of some of the symptoms. Not only is it useless as far as the removal of the growth is concerned, but it may so considerable damage because waiting means an increasing opportunity for the progressive growth to destroy the cerebral tissue. Complete recovery or great amelioration of symptoms can be expected only from **surgical intervention.** The latter consists of the following procedures: (1) Lumbar puncture, (2) puncture of the corpus callosum, (3) palliative operation and (4) radical operation for removal of the tumor.

Lumbar puncture has given to some satisfactory and to others unsatisfactory results. Sicard has recently (*Presse. Méd.*, 1908) formulated the following rules which if followed strictly will give favorable results. For 48 hours the patient must be be kept in bed before the lumbar puncture is made; the head must not to raised during that time. The puncture is made in Trendelenburg's position (head lower than body), the patient placed on his side. The same position is to be kept for the following 12 or 24 hours, then the patient is placed horizontally for 48 hours. Lumbar puncture should never be made when headache, vertigo, etc., are aggravated by a horizontal position. I have obtained great amelioration of the distressing symptoms of cerebral growths in a number of cases by means of lumbar puncture. In three cases of tumor in the cerebello-pontine angle a similar procedure aided me in improving the patient's general condition and thus prepare him for the major operation. I have never met with untoward results.

Puncture of the corpus callosum has been advocated particularly by Anton and Bramann (*Münch. mediz. Wochenschr.*, 1908). From an experience of 40 cases they draw the conclusion that this procedure establishes a free communication between all the ventricles and the subdural space. It relieves promptly the headache, vertigo and vomiting, also the hyperemia and stasis of the papillæ. By preventing thus rapid blindness, it helps gaining time so as to prepare the patient for an eventual radical operation. During the puncture of the corpus callosum the ventricles should be probed so that any abnormal resistance in them should be destroyed.

Palliative operation, undertaken in cases of inoperable tumor, has for object **cerebral decompression.** It consists of removal of a portion of bone in the temporal or occipital areas and of opening the dura. Restoration or preservation of sight, diminution of optic neuritis, relief from, agonizing headache and vomiting, have all been observed after cerebral decompression. In sub-tentorial tumors the operation is performed in the

sub-occipital region, in all other cases the temporal region on one and later on the other side is the seat of choice.

In inoperable tumors, viz. when the neoplasm is so situated that it cannot be removed, or when localization is impossible or when the gravity of the condition is such that delay means loss of sight, decompression is urgent and at the earliest possible moment. While blindness is not prevented in every operated case, nevertheless the positive results obtained in some are sufficient to warrant the necessity for decompression. In Cushing's statistics of 130 cases, 59 had increased vision, 65 remained the same and 16 had decreased vision. Cushing's new idea of performing decompression on the side opposite to that occupied by the tumor seems to meet with favor.

That operative procedures may give excellent results can be also seen from the instructive statistics of Allen Starr, who says that out of 361 operated cerebral tumors 154 recovered. Oppenheim's statistics show 11.4 per cent. recoveries. Operations should be performed as early as possible, as the smaller the tumor the less damage will be done to the cerebral tissue. As to the variety of surgical procedures, osteoplastic operations have proven to be of more value and followed by better results than trephining. The operation should be performed as near as possible to the lesion, so that if subsequently it be decided to attempt the radical operation, there will be less damage to the brain tissue. As to the seat of the tumor, this is to be determined by a close neurological examination as to motor and sensory symptoms, reflexes, localized convulsive seizures, etc.

Horsley recommends in decompressive operations, irrigation of the subdural space with sublimate solutions (1 : 1000). This procedure has given him good results.

A few words in regard to the medical treatment. The latter is only palliative. Besides specific drugs, which may give favorable results in some cases, drugs must be given for alleviating pain, vomiting, vertigo or insomnia. Any of the coal-tar products (salicylates, phenacetin, antipyrin, aspirin, etc.), must be given before an operation is decided upon. Bromides, hyoscine, codein may relieve the vomiting, the pain, the vertigo and the convulsions. In some cases ergot is of great benefit, when other means fail. Veronal should be given for insomnia. The diet must be light and given in small quantity; the bowels should be kept freely open. In tubercular cases when relief is obtained from the distressing symptoms by the above means, cod-liver oil, iron and arsenic should be administered.

CHAPTER X

HYDROCEPHALUS

HYDROCEPHALUS is characterized by an excessive accumulation of fluid in the cranial cavity. In the majority of cases it occupies the ventricles (**internal hydrocephalus**), but it may also accumulate in the subdural space (**external hydrocephalus**).

VARIETIES OF HYDROCEPHALUS

I. (a) **Chronic Congenital.**—This form is associated with malformations of the brain. The hemispheres of the cerebrum and cerebellum may be entirely wanting (anencephaly), may present absence of certain lobes or of other portions (as corpus callosum, etc.), or they may be reduced in size (microcephaly or microgyria). (See chapter on Malformations.) With the congenital hydrocephalus are frequently associated symptoms of arrested development of various tissues or organs of the body (spina bifida, ectopia of organs, flat-foot, hare-lip, etc.).

(b) **Chronic Symptomatic Hydrocephalus.**—This form is secondary to other morbid processes. Cerebral (especially at the base) and mostly cerebellar tumors, through compression of the sinuses of the dura and of Galen veins, will interfere with the return circulation and produce ventricular dropsy. Quincke called attention to excessive exudation of cerebro-spinal fluid in the ventricles caused by serous meningitis. Finally hydrocephalus may be symptomatic of phlebitis of the sinuses.

II. **Acute.**—It is a rare affection. It is due to an infectious or toxic condition (gastro-intestinal diseases). The effusion is more frequently in the ventricles than in the subdural space. In the first case the inflammation affects the choroid plexus and the ependyma of the ventricles; in the second case, the pia-mater, and it is therefore the result of an acute serous meningitis.

III. **External hydrocephalus** is by far less frequent than internal. It is practically a **subdural cyst** and the result of a localized meningitis.

Pathology.—In the **ventricular** form the following condition is found. Among all the ventricles the lateral ones are chiefly affected. The amount of fluid is variable; two quarts is not a very rare occurrence. The fluid is clear, with a specific gravity of 1005 to 1010. It contains

a small quantity of albumen and a noticeable amount of chlorides (more than in the plasma of blood). The distention of the ventricles may be symmetrical on both sides or may be more pronounced on one side than on the other. When the orifices of Monro are dilated, the third ventricle and the aqueduct of Sylvius are distended, the dilatation will reach the fourth ventricle.

The lining membrance of the ventricles and the choroid plexuses are thickened. The cerebral tissue, being compressed, is reduced in thickness. The convolutions are flattened and the fissures disappear. In hydrocephalus occurring very early in life, myelinization of the nerve fibers is arrested. The pyramidal fibers through their entire course present in this case an atrophic condition. In hydrocephalus appearing later in life the pyramidal tract is in a state of secondary degeneration. The cranium is enlarged and globulous. The fontanelles are large, the sutures are separated.

FIG. 76.—HYDROCEPHALUS. (*Oppenheim.*)

Symptoms.—The most conspicuous **initial** symptom is the gradual increase of the size of the head. In some cases, however, **spasticity** of the extremities or **convulsions** may precede the abnormal development of the head.

The head is usually enlarged in its transverse diameter, the sutures are separated, the forehead is unusually prominent. The facies is striking: it is pale and thin, the subcutaneous veins are prominent, the eyes are protruding and directed downward. The patient is unable to hold the head erect, as the latter has a tendency to fall back. The hair on the head is sparse.

Nervous symptoms are mainly **motor.** Convulsions appear early; they are due to pressure upon the cortex produced by the dilated ventricles. The contractures of the extremities and weakness, which are often present, are incomplete. The tendon reflexes are increased and the toe phenomenon is present. The gait is difficult and uncertain.

General **sensibility** is rarely affected, but among the special senses the **vision** is very frequently and quite early affected in hydrocephalus because of pressure exerted by the dilated third ventricle upon the chiasma. Optic neuritis followed by optic atrophy is a common occurrence. Strabismus is also commonly observed. Mental development is frequently arrested. There may be idiocy or only feeble-mindedness.

All the faculties are retarded in early hydrocephalus; thus the speech is acquired late.

As the disease is usually progressive, the intracranial tension and pressure continue to increase. Headache is therefore a frequent symptom and the suffering sounds made by the patient are so characteristic that they received a special name, **"hydrocephalic cry."** It is a sort of a shallow scream which does not resemble any sound made by patients in other painful affections. In adults the fontanelles do not separate and therefore the enlargement of the head is not so conspicious as in children. The condition may then be confounded with cerebral tumor. Indeed the general symptoms of the latter are all present, viz. headache, vertigo, optic neuritis, convulsions. The diagnosis is difficult. However, early loss of upward movement of the eyes, early impairment of light reflex, progressive bilateral rigidity, are all almost pathognomonic of hydrocephalus in adults. The reason of the ocular phenomena is to be found in the downward pressure exerted by the distended third ventricle on the nucleus of the third nerve.

Prognosis.—The outlook is very serious. Death usually occurs at an early age and most frequently from some intercurrent disease. There are, however, cases which may recover, but they are very rare. Some writers reported a few cases in which the hydrocephalic fluid found a passage through the nasal fossæ and through the ears after a fracture of the skull. That hydrocephalus may become arrested in its development is a well known fact: the sutures become then ossified and the head ceases to grow large; the individual may live long.

Diagnosis. Chronic Hydrocephalus.—As the large size of the head is the most prominent symptom in hydrocephalus, the latter may be confounded with **rickets,** in which the head may be also voluminous. In the latter affections there is usually no intellectual disturbance; the fontanelles are not enlarged, the cranium is not uniformly enlarged; finally the usual stigmata of rickets (deformities of limbs, etc.) will render the diagnosis comparatively easy.

Acute Hydrocephalus.—Acute Cerebral Affections of early infancy may simulate hydrocephalus. It is therefore important in such cases to examine frequently the state of the fontanelles and of the sutures. **Tubercular meningitis** in infants may also resemble hydrocephalus. In making a diagnosis it should be remembered that tubercular meningitis occurs habitually in children that are weak or directly tubercular. Moreover the cerebro-spinal fluid in tubercular meningitis presents some special features, viz. increase of albumen and especially lymphocytosis and the tubercle bacilli. A lumbar puncture will therefore decide the

differential diagnosis. The greatest difficulty lies in differentiating **internal** from **external** hydrocephalus. However the enlargement of the head in ventricular forms is more rapid than in external forms. Besides, at the beginning of external hydrocephalus the symptoms are those of hemorrhagic meningitis or pachymeningitis, and the latter affections lead rapidly to death.

Etiology.—In **congenital** forms of hydrocephalus heredity plays a great rôle. There is almost always a history of some nervous or mental disorder or else some constitutional disease in the family, as tuberculosis, alcoholism and especially syphilis. Consanguinity of the parents has also been reported as an etiological factor. Fournier and Hutchinson call special attention to the effect of syphilis of the parents on the production of hydrocephalus in the offspring. During pregnancy physical injuries to the fœtus may lead to pathological changes and cause hydrocephalus, as, for example, compression, violence, etc.

Hydrocephalus, which makes its appearance **in infancy,** develops most frequently in the course of **gastro-intestinal diseases.** The mechanism of formation of the affection is various: it may be thrombosis of the venous sinuses or inflammation of the ventricular lining membrane and of choroid plexuses, with the result of abundant serous exudation. Rickets is not infrequently associated with hydrocephalus and therefore may be considered as a cause of the latter. Trauma of the head has also been mentioned as a cause of acquired hydrocephalus (Plehn, Quincke, Finkelnburg and Nonne). The etiology of the **symptomatic** forms of hydrocephalus depends upon the accompanying cerebral affection (see above).

Treatment. Chronic Hydrocephalus.—In view of the great rôle which syphilis plays in the etiology of this affection, a vigorous specific treatment must be instituted as early as possible. While a complete cure should not be expected, amelioration of symptoms may be obtained. Hygienic measures aided by a proper diet will improve the general health of the patient. In **acute hydrocephalus** the treatment must be directed against the cause. As gastro-intestinal diseases are a frequent cause, removal of the latter must be the principal aim: an appropriate diet with intestinal antiseptics, local applications, etc., must be administered.

Surgical intervention, still advocated by some, is a serious procedure. It consists either of puncturing the cerebral ventricles or of a lumbar puncture. Both methods are uncertain: observation shows that the patients either die from sudden decompression of the brain, when too much fluid is withdrawn, or from secondary inflammatory complications following the puncture. Moreover, the fluid reforms immediately after

the puncture; repeated punctures will therefore be necessary. On the other hand some very competent authors report favorable results from both procedures. Anton and Bramann (see Treatment of Tumors of Brain) obtained great amelioration of the general symptoms, viz. headache, vertigo, vomiting also of ocular disturbances by puncturing the corpus callosum and probing the lateral ventricles. Krause (*Zentralbl. f. Chir.*, 1908) drained directly the cavity of the ventricle into the subcutaneous tissue of the scalp by means of a silver tube inserted through the cortex into the ventricle and held in place. In some of his patients the tube was held in place for several months. Some favorable results were obtained. Equally good results were reported by Finkelnburg (*Münch. mediz. Wchnschr.*, 1910) who speaks of puncturing the motor area at a depth of 5 cm.

Lumbar puncture may also improve the general manifestations of hydrocephalus in a limited number of cases.

CHAPTER XI

DISEASES OF THE BASAL GANGLIA (OPTIC THALAMUS. CORPORA STRIATA. CORPORA QUADRIGEMINA)

I. THE SYNDROME OF THALAMUS OPTICUS

It was first brought out in 1903 by Dejerine and Egger.

Pathological Physiology.—The thalamic syndrome is the result of a lesion in the external and internal nuclei, or in the pulvinar of the thalamus; also in the fibers of the posterior portion of the internal capsule. The thalamus being essentially a **sensory** organ connecting the periphery with the cortical centers by means of cortico-thalamic and thalamo-cortical fibers, a lesion of it will naturally give place mainly to **hemianæsthesia.** The latter explains the **hemiataxia** which is so frequently found associated. The motor symptoms find their explanation in the involvement of the capsular fibers.

Symptoms.—They are: **hemianæsthesia, hemiataxia, hemiplegia, choreiform movements, athetosis, pain** on the anæsthetic side. Sometimes **mimicry** is affected.

Hemianæsthesia.—It is pronounced for deep sensations (muscular, tendinous, articular and osseous) and slight for superficial sensations (touch, pain and temperature).

The muscular sense, position of the limbs, the sense of active and passive movements, of resistance, of force, of weight are all completely abolished.

Hemiataxia.—It is slight and limited, but it does not interfere to a great extent with locomotion.

Hemiplegia.—It is slight. It is not a real paralysis but only a slight diminution of power. Contracture does not develop. It has a tendency to improve. The knee-jerks are usually exaggerated. Meynert, Schiff, Clarke and a few others observed in lesions of the thalamus: flexion of the arm on the opposite side, extension of the arm on the same side and head and eyes turned to the opposite side of the lesion.

Choreiform Movements and Athetosis are not marked. They are brought out especially on voluntary movements. Their pathogenesis is not yet definitely established. However athetosis appears to occur not infrequently in lesions of the thalamus.

Pain on the anæsthetic side is either spontaneous or brought on by the

least pressure. There may be distinct paroxysms of shooting pain. Instead of pain there may be numbness, burning or tingling.

In addition to the above symptoms there are sometimes hemianopsia, disturbances of the bladder and trophic disorders. The hemianopsia is of the homonymous type. It is probably due to the involvement of the fibers of the optic tract which originate in the pulvinar.

In a few cases of lesions of the thalamus the following phenomenon was observed: on voluntary movements the muscles of the face contract equally on both sides, but in the act of laughing or crying (emotional act) the motion of the affected side is considerably diminished or else abolished.

Course.—The onset is usually sudden. There is a loss of consciousness preceded by some vertigo. The motor disturbances improve rapidly, while the sensory persist and even increase.

Diagnosis.—A marked hemianæsthesia associated with a mild hemiplegia is the essential feature of thalamic syndrome. However there are other portions of the central nervous system which, being diseased, may present the above symptomatology, but in such cases there are usually **additional** manifestations which will aid in making a diagnosis (see Roussy's thesis).

II. CORPUS STRIATUM

Lesions confined strictly to the gray matter of striate bodies are very rare. However there are a few cases on record, in which the adjacent tissue was not involved. They are, exclusively, hemorrhages. The symptom-group of such an affection has been recently established with some precision by S. A. K. Wilson (Brain, V. XXXIV). He describes the affection under the name of **"progressive lenticular degeneration,"** which can be considered as the **syndrome of the corpus striatum.** Pathologically there is total integrity of the internal capsule and pyramidal paths, but there is a bilateral softening and degeneration of the lenticular nucleus involving more particularly the putamen and but slightly the globus pallidus; otherwise speaking, the affection is an **extra-pyramidal motor disease.**

Clinically it is characterized by a pronounced spasticity of the limbs and of the face without or with very little paralysis; by a bilateral tremor of all extremities which increases with voluntary movements; by dysphagia and dysarthria or anarthria; by spasmodic laughing or crying (only occasionally); by absence of the toe phenomenon and preservation of the abdominal reflexes; finally by transitory mental disturbances; impairment of the capacity of retaining impressions; childishness; docility. An-

other characteristic feature of the disease is its familial nature. Finally the disease is associated with cirrhosis of the liver.

The diagnosis should be made between this affection and **Pseudo-bulbar palsy** which in some respects resembles the former. In pseudo-bulbar palsy the palate and tongue are paralyzed. In the lenticular disease there is no true palsy of these two organs. In pseudo-bulbar palsy there is a double hemiplegia with the characteristic abnormal reflexes; they are all absent in the lenticular disease. Pseudo-bulbar palsy is a disease of the pyramidal tract.

The tremor of the lenticular disease resembles that of **Paralysis agitans,** but the latter is a disease of advanced age and chronic in nature, while the lenticular disease is a disease of youth.

Progressive lenticular degeneration occurs at the age of between 10 and 25, in males more frequently than in females. It is invariably fatal. Its duration is from two to seven years. The disease is probably acquired and apparently toxic in nature. That the toxin is elaborated in the liver, is a plausible inference. Syphilis and alcohol apparently play no rôle whatever.

Bilateral softening of the lenticular nucleus has been observed in cases of **gas poisoning.** Dana (*J. of Nerv. and Ment. Dis.*, 1908) published such cases. He observed during life a vasomotor (œdema) and gangrenous condition of the skin which he considered belongs to the syndrome of the corpus striatum. He says that this body has no specific and independent motor function. It has a supplementary motor function and especially in connection with articulation. It may have some control over the bladder. Gowers, Ormond, Homen and especially Mme. Vogt published cases in which there is the greatest resemblance with the syndrome as given by Wilson. The latter's description could be considered as a classical picture of the symptom-group of the corpus striatum.

III. LESION OF THE CORPORA QUADRIGEMINA

The anterior quadrigeminal bodies are connected with the optic tracts, and are therefore concerned in vision, the posterior bodies are connected with the cortex of the temporal lobe and are consequently concerned in hearing (see Anatomy.) The symptomatology will therefore differ according to what tubercles are involved. Diseases (hemorrhage, softening or tumor), confined exclusively to the quadrigeminal bodies are exceedingly rare. The adjacent portions are usually involved simultaneously. The symptoms cannot therefore be uniform. However the following symptoms have been observed almost constantly in tumors limited to the quadrigeminal bodies.

Eye Symptoms.—Ptosis, paralysis of bilateral associated movements of the eyes—upward and downward, also limitation of lateral movements of the eyes, finally paralysis of convergence—these are the most common ocular phenomena. Ophthalmoplegia is therefore characteristic. It is due to an involvement of the oculomotor nuclei situated beneath the quadrigeminal bodies. The pupils are usually unequal and dilated. Later in the course of the disease when secondary changes set in the optic nerves, the vision becomes defective, but the latter is not impaired in the beginning.

Ear Symptoms.—When the posterior quadrigeminal bodies are affected the hearing becomes deficient. Some observations point to the fact that a lesion of one posterior quadrigeminal body causes impairment of hearing on the opposite side.

Motor Symptoms.—Nothnagel observed in almost all cases a **gait** characteristic of an intoxicated person similar to that of cerebellar diseases. Loss of equilibrium in standing is another symptom. Motor paralysis is absent except in advanced stages when the tumor presses on the motor tracts. Nothnagel claims that when the cerebellar gait appears as the **first symptom** and especially in association with the above mentioned ocular phenomena, the diagnosis is certain. The knee-jerks are usually not involved except when the motor tract becomes infringed upon by the progress of the tumor. Then they become exaggerated and the toe phenomenon makes its appearance. They may be found also diminished or abolished like in cerebellar diseases.

Tremor of the arms, also of the head and legs has been observed in some cases.

CHAPTER XII

DISEASES OF THE MEMBRANES OF THE BRAIN

I. INFLAMMATION (MENINGITIS)

In the chapter on anatomy we have seen that the brain is practically covered with two membranes, viz. dura and pia-arachnoid. An inflammation of the pia and that of the arachnoid separately does not exist; both latter membranes are usually involved simultaneously. We will therefore consider an inflammation of the dura—**pachymeningitis,** and an inflammation of the pia-arachnoid—**leptomeningitis.**

A. **Pachymeningitis.**—The dura-mater consists of two layers. The outer layer, which is intimately adherent to the skull, is considered as its endosteum. The inner layer is smooth and in contact with the arachnoid. Either of these layers may become inflamed independently of each other. There are therefore external and internal pachymeningitis.

(a) **External Pachymeningitis** is mainly a surgical affection; it is always secondary to diseases of the skull and especially to injuries of the latter. When a fracture occurs, the blood detaches the outer layer of the dura. The accumulated blood may become absorbed when it is in a small quantity, but in unfavorable cases suppuration takes place. Caries of the bone, osteitis may also lead to an external pachymeningitis.

Pathology.—At first red and œdematous, the dura soon becomes thickened and adhesions form between it and the skull. This is frequently observed in lesions of the petrous bone. In some cases suppuration takes place and purulent collections are found between the dura and the skull.

Symptoms.—In severe traumatic cases there is usually coma, which sometimes may be deep and prolonged. In case the blood or pus compress any of the cortical centers, corresponding symptoms will be present (see Localizations). Hemiplegia, Jacksonian epilepsy are not infrequent.

Treatment.—The only rational treatment is surgical intervention, which must be instituted as promptly as possible.

(b) **Internal Pachymeningitis.**—It is frequently called hemorrhagic internal pachymeningitis or **hematoma of the dura.** The disease is very rare and met with in the extreme ages of life, viz. in very young children and aged individuals. It occurs not infrequently in the insane and especially in paretics. Alcoholism (chronic) is a very frequent etiological factor.

Rheumatic diathesis, infectious diseases, traumatism are other causes of pachymeningitis interna. Purpura, scurvy, rickets and tuberculosis are the causes of infancy.

Pathology.—The chief lesion lies between the arachnoid and the dura and consists essentially of a thickening of the latter and formation of layers of membranes on its inner surface to which they adhere. The membranes are very vascular. The new blood vessels of this new tissue have external and internal coats, but no media, a condition which makes them fragile. Rupture of the new blood vessels, which frequently undergo degeneration, is the origin of the blood found in pachymeningitis. The blood accumulates between the laminæ of the newly formed membranes. The amount of blood varies from a very small quantity to a very large collection. The hemorrhagic cyst thus formed may undergo transformations, among which the most important are serous, purulent (through secondary infection) and state of calcification. It goes without saying that the brain tissue will suffer from compression caused by the adherent cyst; inflammation with softening will be the result. The most frequent seat of the hematoma is the parietal lobe; then in order of frequency come the temporal, frontal and occipital.

Symptoms.—When the hematoma is very small, the pachymeningitis may pass unobserved, but most of the time the hematoma reaches a sufficient size to present symptoms of cerebral compression. In the majority of cases prodromal symptoms are present. They are: headache, vertigo and a paralytic condition, accompanied by convulsions on one side. Headache may be the only symptom for some time. It is very severe, localized on one side of the skull and usually not accompanied by fever. The pupils are as a rule contracted but more on the side of the lesion. Nystagmus may be present. Œdema of the papilla is frequently observed and it is due to the compression; it has the same value as in tumors of the brain with this difference that it appears late in the course of pachymeningitis. Gradually and sometimes rapidly coma makes its appearance; at first it is only slight, but gradually becomes more and more profound. Death supervenes usually in a short time. The paralysis may affect one or two limbs, also the face. The palsy is rarely complete. Its onset is slow but progressive.

In cases with a slow onset a gradually increasing mental hebetude with great somnolence are the most striking features. In a case of a physician of the Jefferson Hospital, who died from this affection, there was noticed an extraordinary tendency to sleep; he would fall asleep wherever he happened to be. I also noticed that he was unable to concentrate his attention. Gradually he developed unilateral convulsions,

an exaggerated knee-jerk on the same side, also the paradoxical reflex. An extensive hematoma of the dura was found on the opposite side.

Prognosis.—In all cases the outlook is very grave. Recovery is extremely rare. Remissions may occur in the course of the disease, but death is the usual termination. The **duration** is usually from a few weeks to several months.

Diagnosis.—Mental hebetude, somnolence, headache with localize palsies and convulsions may be met with in **cerebral syphilis.** The paroxysmal character of the headache and its aggravation at night, finally the history of a specific infection will differentiate brain syphilis from internal pachymeningitis.

It was mentioned above that the paralytic symptoms develop gradually in hematoma of the dura. This will differentiate the condition from cerebral hemorrhage, in which the apoplectic seizure and the hemiplegia appear suddenly.

Tubercular Meningitis may sometimes simulate internal pachymeningitis, but the less acute headache, constipation, retraction of the abdomen and the character of the cerebro-spinal fluid of the former will render the diagnosis less difficult. The greatest difficulty is experienced in making a diagnosis between hematoma of the dura and **cerebral tumors.** In such cases it may be of some use to remember that œdema of the papilla is a very frequent occurrence in tumors. It is less frequent in pachymeningitis and it appears quite late in the latter disease.

Treatment.—Counter-irritation, leeching in the mastoid regions, elevation of the head, ice to the head, avoidance of stimulants are all the means used in such cases. If there are strictly localized symptoms, evacuation of the hemorrhagic focus is advisable. In the case mentioned above the first operation gave an excellent result. The patient rallied and the cephalalgia disappeared.

B. **Leptomeningitis.**—It may be acute and chronic.

Acute Meningitis.—In studying the acute form the following varieties will be considered: (1) Non-tubercular, (2) tubercular and (3) epidemic cerebro-spinal meningitis.

I. NON-TUBERCULAR MENINGITIS.

The latest researches in the domain of microbiology (Fränkel, Weichselbaum, Netter, etc.) have definitely established the infectious nature of meningitis. Meningeal infection may originate from an **intra-cranial** or **extra-cranial** focus. In the first case a cerebral abscess will invade the meninges by direct contact or through the blood vessels. In the

second case a fracture of the skull will present a direct road for invasion of the microbes from exterior. The natural cavities located at the base of the cranium, viz. nasal, orbital, pharyngeal, auricular, etc., which are constantly exposed to infection, are in relation with the cranial cavity by means of a large number of blood vessels and nerves; the infection can be therefore easily transmitted. Various diseases of these cavities may easily lead to an infectious involvement of the meninges. Among them the most frequent is the infection of the ears. All suppurations of the latter are to be feared, especially those of the middle ear which spread to the mastoid cells. The infection is here frequently transmitted to the meninges through the lymphatic vessels or through the neighboring sinuses. An infection may originate on the face (abscess, erysipelas, a boil, etc.), in the hair, in the bones of the cranium (osteomyelitis, tuberculosis or syphilis). An infection of any part of the body may become the point of departure of meningitis. The latter may develop in the course of infectious diseases (pneumonia, typhoid fever, etc.) and present therefore a bronchial, pulmonary, intestinal, biliary, urinary, endocardial or puerperal origin. In such cases the microbes or their toxins pass into the general circulation and the meninges, which are exceptionally rich in blood vessels, become therefore easily the seat of secondary infection.

The largest number of cases of meningitis are due to the pneumococcus (Netter). It may develop secondarily to a pneumococcic infection or primarily. In the latter case it may be sporadic or epidemic. The gross characteristic feature of meningitis due to this microbe is the formation of pus, which is greenish and particularly thick.

Other microbes are met with less frequently. In order of frequency they are: Meningococcus of Weichselbaum, diplococcus of Talamon-Fränkel, streptococcus, typhoid bacillus, staphylococcus. There are also cases in which the meningitis is due to a mixed infection, as for example, pneumococcus with staphylococcus and others. It is impossible at present time to differentiate clinically with precision the forms of meningitis caused by various microbes. They all have many common features. The predisposing factors should not be overlooked in cases of meningitis. Traumatism, exposure to cold, intellectual exhaustion and alcoholism, are all conditions which favor an invasion of microörganisms.

Finally meningitis may occur independently of microörganisms. Such is the case of lead intoxication. Meningitis in chronic lead poisoning is an established fact.

Pathology.—The first period of an inflammatory condition of the pia-arachnoid is characterized by a congestion. The meninges are red, the

blood vessels are dilated not only in the membranes, but also in the adjacent portion of the cortex. Soon exudation takes place. The latter may be **serous,** or **purulent.** The fluid of the serous exudate differs from cerebro-spinal fluid in that it contains a larger quantity of albumen and of leucocytes. Quite frequently fibrinous masses form in the exudate; they lie over and between the convolutions, agglutinate the latter to each other and raise the arachnoid. Such are the findings in meningitis in its early stages. In an advanced stage pus takes the place of the serofibrinous exudate. Most frequently it is arranged in bands or else in islands. Sometimes it is so abundant that it covers the entire convexity of both hemispheres, extends to the base, envelops the medulla and involves the cranial nerves. Between the serous and purulent exsudates there are intermediary varieties, as the transition of one into the other is not rapid.

The pia-mater is infiltrated and œdematous. The inflammation usually spreads to the choroid plexus and to the ventricles. The choroid plexuses are swollen, thick and frequently covered with pus. The contents of the ventricles may be transparent, purulent or sero-purulent or else hemorrhagic. The brain tissue is involved to a more or less great degree according to the stage of meningitis. At an early period, when there is only a serous exudate, small hemorrhagic foci with abundant leucocytes will be noticed. In an advanced stage softening of cerebral tissue is observed. It is due to vascular changes in the affected area.

Symptoms.—The many etiological factors enumerated above will naturally lead to the idea that there may be a great variety of clinical pictures of acute meningitis. Nevertheless, irrespective of the nature of meningeal infection there exists a **general type** in which the symptoms are met with constantly and from which the special forms differ little. The symptoms of this type are as follows.

Two distinct phases are observed in the majority of cases, viz. **excitation** and **depression.** Very frequently the first period begins almost suddenly by an intense fever and a chill. In other cases the onset is insidious and preceded for a few days by headache and vomiting. In children the onset may be accompanied by convulsions.

Gradually the initial symptoms become aggravated. The **headache** which is continuous assumes an unusually severe character: it is burning, lancinating, increased upon the slightest motion, a noise in the vicinity of the patient or upon exposure to light. **Insomnia** is the usual consequence of the headache. The **fever** which is from the first pronounced (101°–103°) remains as such throughout the disease, although morning remissions may occur. In the **purulent** form of meningitis the tempera-

ture may reach 104° or 105°. In cases of fatal termination the temperature may ascend even to 106° or 108° toward the end. The fever is attended by a rapid and full **pulse.** Absence of parallelism of pulse and temperature, viz. elevation of temperature and slow pulse or *vice versa*, is observed quite frequently in all forms of meningitis but especially in the tubercular form. The **respiration** is accelerated and irregular: the thoracic and diaphragmatic movements are not synchronous during breathing. **Vomiting** is of a cerebral character, viz. it occurs without the slightest effort and unaccompanied by nausea. **Constipation** is unavoidably present. It is unusually rebellious to purgatives. The **abdomen** is **retracted.** The **facies** of the patient is quite characteristic: redness and pallor alternate, the eyes are glossy, photophobia is marked. The **skin** is warm and dry. Irritation of the skin with a nail is followed by a persistent redness (**Tâches cérébrales**).

Ocular phenomena are particularly important. At first strabismus with diplopia makes its appearance. The pupils are unequal and contracted but later become dilated. The eye-grounds show a dilatation of the retinal veins and lateral œdema of the papillæ.

Optic neuritis is a frequent, although not a constant symptom, especially in meningitis of the base of the brain. Other cranial nerves, particularly those of the ocular muscles and the facial are frequently affected in basal meningitis.

The symptoms of excitation which characterize the first period affect the **intellectual sphere,** the **motor** and **sensory** apparatus.

Delirium appears with the early symptoms. The patient is agitated, very talkative, restless. He talks incoherently, rapidly and loudly, screams, shouts. He fights, resists restraint. Hallucinations, especially visual, are frequent.

The **motor** symptoms are manifested in **convulsions, palsies** and **contractures.** The first are particularly frequent in children. They may be generalized or localized. Contractures are more frequent than convulsions. They are almost constant and have an important diagnostic value. They are very irregular and intermittent. The most frequent seat of contractures is in the muscles of the neck (posterior cervical muscles). The rigidity of the neck is the earliest and the most persistent phenomenon; it affects particularly the movements of flexion of the head. If the muscles of the back are involved, opisthotonos will be the result. The muscles of mastication (trismus), of the eye globes (strabismus), of the face, of the pharynx, of the larynx, of the tongue, of the abdomen and of the sphincters may be affected. In the limbs the contracture affects mostly the flexor muscles. The presence of contracture can be shown by **Kernig's sign.** It

consists of an inability to extend the leg over the thigh when the patient is sitting, or else when the patient is lying on his back with his thighs flexed over the trunk. This phenomenon is due to an irritation or inflammation of the spinal meninges and the roots. While it is present almost in every case of meningitis, nevertheless it was found in conditions other than meningitis, as for example, in typhoid fever; it is absent in tubercular meningitis. Brudzinski (*Arch. de méd. des enfants*, 1910) described a **neck reflex** which is constant in meningitis. It consists of flexion of the thighs and legs, when the head is firmly flexed forward.

Among other motor symptoms can be mentioned exaggerated deep and superficial reflexes. The plantar reflex appears in extension on one side and in flexion on the other. The knee-jerks are sometimes abolished. Paralysis of the extremities may appear quite early in meningitis. It may be only transitory and disappears when the patient commences to improve, or it may develop gradually and remain permanent. The paralysis may be hemiplegic or monoplegic; the face is usually also involved.

The **sensory** symptoms of the first phase of meningitis consist of general cutaneous hyperæsthesia and disturbances of the special senses (hearing and sight); the least noise is painful to the patient; photophobia is a common observation.

When the patient enters the **second phase** of the disease the above described symptoms gradually become less and less pronounced. Depression takes the place of the tumultuous manifestations of the first period. The delirium and agitation disappear. The convulsions and the contractures leave the patient and instead of them **paralysis** develops. It may be hemiplegic or monoplegic. The **sphincters** of the bladder and rectum also become paralyzed; incontinence is the consequence. The **fever** increases, the **pulse** is slow. Mental **hebetude** becomes more and more marked, general **anæsthesia** makes its appearance. Soon **bulbar** symptoms enter the scene. The **respiration** becomes superficial and of Cheyne-Stokes' type. The extremities and face are cold and death follows in deep coma.

Course. Duration. Termination.—Ordinarily the general type of acute non-tubercular meningitis has a rapid course. In the majority of cases it lasts about eight or ten days. In some cases death may occur in the first phase, during the convulsive period. While death is the usual termination, nevertheless there are cases of recovery. The latter may be incomplete or complete. In the first case some permanent lesion may be present as a sequel, as for example, paralysis, mental impairment, hydrocephalus, ocular palsies.

Clinical Forms.—There are certain forms of acute non-tubercular

meningitis which differ more or less from the classical type described above.

Serous Meningitis is characterized by considerably less marked symptoms. Headache and constipation are usually absent or very mild. Delirium is less persistent than in the purulent form. According to Quincke congestion of the retina is a frequent occurrence. The course is long and irregular with periods of amelioration and aggravation. Recovery is the usual outcome. The nature of serous meningitis deserves special mention. Bacteriological researches of the cerebro-spinal fluid have demonstrated either presence or absence of microörganisms. Typhoid bacillus, pneumococcus, staphylococcus, Pfeiffer's bacillus, have been found. Serous meningitis is therefore of an infectious origin. The same results have been obtained experimentally by injecting various cultures into the meninges. It is consequently legitimate to admit an etiological unity of all forms of acute meningitis and that bacterial infection of meninges may produce either a plain congestion with serum exsudate or a purulent meningitis. The result is identical with what is observed in infections of serous membranes of other organs. The fact that sometimes the microörganisms are not detected in the cerebro-spinal fluid does not speak against the infectious nature of serous meningitis, because in some cases their number is exceedingly small or they may have already disappeared from the fluid which is to them a poor medium. Under the name of **Méningism** Dupré in 1894 described slight meningeal manifestations while at autopsy no anatomical changes are found. They may develop either primarily (in hysteria, or in association with intestinal parasites) or secondarily in the course of infectious diseases. Probably all those cases of so-called meningismus are in reality curable cases of slight serous meningitis.

Circumscribed Serous Meningitis was first described by Ströbe in 1904. It is met with in cases in which a former meningitis produced adhesions and thus isolated an arachnoid area. It consists of an infiltration with fluid of the arachnoid tissue which is therefore distended. It is a sort of œdema of this tissue and the fluid is imprisoned in it. The fluid is clear and sterile and is under sufficient tension to compress the underlying nervous tissue and thus produce symptoms of a neoplasm. The most frequent seat of these pseudo-tumors is the spinal region. The latter was particularly studied by Schlesinger and Krause. In the cerebellar region they are more frequent than in the cerebrum. The nature of circumscribed serous meningitis is probably infectious in spite of the fact that the cerebro-spinal fluid is found to be aseptic. The diagnosis between this affection and tumor is difficult. Oppenheim mentions

alternating **remissions** and **aggravation** as a characteristic diagnostic sign. The treatment consists of excision of the wall of the entire pseudo-cyst and recovery is frequently complete.

Meningitis due to Pneumococcus.—It is the most important form of meningitis. It is encountered not only in pneumonia, but also in various affections, such as grippe, typhoid fever, endocarditis, puerperal fever. This frequency is due to the constant presence of the pneumococcus in the cranial cavities. For this reason traumatic meningitis is frequently caused by the pneumococcus. The same germ is found in meningitis complicating otitis. In the majority of cases this form of meningitis is purulent and the pus is situated between the pia and the convolutions. In the primary form the temperature rises very high, higher than in the epidemic cerebro-spinal form. Albuminuria is marked. In the secondary form it usually remains latent and then suddenly a rise of temperature with a violent delirium announces a meningeal complication. The cerebro-spinal fluid shows usually leucocytosis of the polynuclear variety; in some cases a lymphocytosis precedes polynucleosis. Sometimes no leucocytes can be found.

Typhoid Meningitis.—Developed in the course of typhoid fever it presents these peculiarities: absence of headache, vomiting and constipation.

The elevation of temperature is not high and the course is slow. It is more frequent in children. It usually develops in the second week of typhoid fever. The cerebro-spinal fluid possesses a very marked agglutinative power for the Eberth's bacillus contrary to ordinary typhoid fever in which this power is none. Leucocytes and microbes may be present even when the cerebro-spinal fluid is perfectly clear.

Meningitis in the course of typhoid fever may not be caused by typhoid bacillus, but by other microörganisms. On the other hand meningitis caused by the typhoid bacillus may develop without typhoid fever. It is then a purulent meningitis.

The prognosis in typhoid meningitis is various: it may be favorable or unfavorable. Netter claims that it is unfavorable when Kernig's sign is present.

Grippal Meningitis.—In the course of grippe meningeal symptoms may develop. They are not due exclusively to the Pfeiffer's microörganism, but to association of several germs. Meningitis with Pfeiffer's bacillus alone is met with in very young children.

Traumatic Meningitis.—It is caused by penetration of microörganisms either through the cranial cavities or through the fractured skull. The onset is characterized by elevation of temperature and rapidity of pulse. Lumbar puncture shows a bloody cerebro-spinal fluid. If centrifugation

is done immediately, the fluid above the blood will be colored red if meningitis is present, but yellow if meningitis is absent. The sediment will show a polynucleosis 90 per cent. and microörganisms.

The prognosis is not absolutely fatal.

Otitic Meningitis.—Inflammation of the ears may give rise to a generalized or to a localized meningitis. Frequently, but not always, acute otitis causes a generalized meningitis, while a localized abscess is the result of a chronic otitis. In children, meningitis may occur while the otitis is apparently mild. It is an inflammation of the inner ear that gives the largest percentage of meningitis. Lermoyez finds one case of meningitis among every eight cases of labyrinthian disease, while in cases of middle ear diseases the percentage is 1 to 600. Involvement of the labyrinth can be recognized by the following symptoms: noises, loss of hearing, vertigo, nausea; loss of mastoid perception of the tuning fork; vestibular nystagmus especially on temperature tests. Involvement of the meninges can be recognized by sudden elevation of temperature which is in contrast with a slow pulse, by facial or oculomotor palsy, by excruciating pain in the temporal region.

Various microörganisms have been found. The most frequent are: streptococcus and staphylococcus. As to the cerebro-spinal fluid, it may be clear, cloudy or purulent. The appearance of the fluid is not in proportion with the gravity of the meningitis, as perfectly clear fluid may contain virulent microbes. It has also been shown that in rapidly fatal otitic meningitis there may be only a congestion or œdema of the meninges and brain.

Syphilitic Meningitis.—Acute syphilitic meningitis is rare. It usually develops gradually. We find here the usual picture of acute meningitis. Palsies are very frequent and affect particularly the ocular muscles and especially those supplied by the third nerve. The palsies are usually transient. Another characteristic feature is the persistent headache and absence of fever.

Finally the evolution of the disease is irregular and variable. It is amenable to treatment and improvement follows rapidly, but it has a tendency to recur. A very important aid in diagnosis of syphilitic meningitis is found from a lumbar puncture. The cerebro-spinal fluid shows a marked lymphocytosis and what is especially valuable is a positive Wassermann reaction. Anatomically is seen a gelatinous exsudate in the meninges in which the spirochetæ are found. The blood vessels show an endo- and peri-arteritis.

Gonococcal Meningitis.—That this microörganism is capable to produce meningitis is now well established. The difficulty of recognizing is

the resemblance of the gonococcus to the meningococcus. In a case seen recently by me, the resemblance was striking. The absence of agglutination of the germ by antimeningococcus serum (**precipitin reaction** of Vincent) and failure of the latter to improve the patient decided the diagnosis. The patient had a severe attack of gonorrhea in the course of which he developed the meningeal symptoms. A lumbar puncture revealed a slightly cloudy fluid with abundant polymorphonuclear cells and the diplococci in pairs with their opposed surfaces flattened were found. In spite of repeated injections of serum the patient died.

Meningitis of old age presents some special features. Considering the onset and the course Schlesinger (*Neurol. Centralbl.*, No. 20, 1912) finds the following main forms of meningitis in aged:

(1) Meningitis with the classical symptoms.

(2) Latent or ambulatory type with vague disturbances assuming the picture of neuralgia.

(3) Cases with apoplectiform onset followed or not by hemiplegia.

(4) Meningitis under the picture of rapidly oncoming dementia.

As to the **symptomatology**—rigidity of the lumbar and dorsal spine appears early, while rigidity of the neck is rarely an early symptom; psychic disturbances are almost invariably present; Kernig's sign is present.

The disease has usually a chronic course. It lasts weeks.

The prognosis is favorable. Sometimes the recovery is only clinical but not anatomical; irritation of the meninges may persist. The cerebrospinal fluid in long standing cases is rich in albumen but poor in cells.

Meningitis in alcoholics is sometimes in a latent state, so that sudden death may occur very unexpectedly. Usually all the symptoms, and especially the delirium, are at their maximum.

Prognosis of Meningitis.—Generally speaking it is serious. With our present methods of treatment, especially sero-therapy, recoveries are not rare. Frequently traces of the old affection, such as palsies, deafness, blindness, etc.) remain indefinitely. Sometimes an apparent recovery may be only a remission.

Diagnosis.—When a group of symptoms as described above is observed in a diseased individual, the first thought should be that of meningitis. Nevertheless we must not lose sight of the fact that purely cortical affections of the brain and of the cranial nerves may present an identical clinical picture. Also there are cases on record which presented the same manifestations and at autopsies no trace of meningeal inflammation was discoverable. In another series of cases only œdema or slight hyperemia of the meninges were found post-mortem. The term **pseudomeningitis or**

méningisme was created by Dupré to designate such a condition (see Serous Meningitis). Investigations have shown that in this so-called **meningitis without a lesion,** the fluid of the œdema contained pathogenic microörganisms of the disease during which the meningeal symptoms developed. The latter are therefore due to the microbes or to their toxins. Thus the examination of the **cerebro-spinal fluid** becomes an absolute necessity.

Lumbar puncture inaugurated by Quincke in 1891 consists of penetrating into the spinal canal between thefourth and fifth lumbar vertebræ for the purpose of obtaining a small quantity of the cerebro-spinal fluid. The examination of the latter must be physical, chemical and bacteriological. The following are the characteristic features of this fluid in acute meningitis.

The color is cloudy or purulent and sometimes bloody. The presence of pus, microbes or tubercle bacillus is an indisputable indication of meningitis. A clear fluid, without changes in its contents, is most frequently (but not always) an indication of absence of meningeal involvement. In one of my cases of acute meningitis the cerebro-spinal fluid obtained from a lumbar puncture was perfectly normal while at autopsy the convexity of the brain was covered with pus.

The following findings permit to establish a diagnosis with great probability.

When the fluid is purulent, non-tubercular meningitis is almost certain.

When the fluid is bloody and the blood is of a pathological origin (not due to the puncture of the skin) it will remain bloody during the lumbar puncture from the beginning to end. After standing, the blood will form a sediment, and the upper layer will be a transparent fluid of yellow color. The latter is characteristic of intraspinal hemorrhage.

When the fluid is clear, meningeal involvement will be revealed by the following symptoms: if the fluid leaves the spinal canal under great pressure; if after standing several hours or less a thread-like formation is seen and finally if there is an increase of albumen (normally there are only traces of it). If the latter two are present, tubercular meningitis is almost invariably certain. Cryoscopical study shows that the freezing point is lowered and oscillates between 0.49° and 0.64° (normally it is 0.72°–0.75°). The most important information is obtained from a cytological and bacteriological examination. The polynuclear leucocytes predominate in acute non-tubercular meningitis, while in the tubercular form the lymphocytes are in abundance (normally only a few cells are found). This leucocytic formula is not absolute in every case; the condition may be found reversed. Besides, lymphocytosis may be observed in other affections,

such as tumors or abscess of the brain. Polynucleosis may be found in tubercular meningitis. In all cases cytological examination is not the only one to be relied upon. On the other hand the absence of the leucocytic formula is not a sufficient indication to reject the diagnosis of meningitis. Daniélopolu and Iancovescu (*C. R. Société de Biologie*, 1911) have described a reaction which is based on the following principle: "the cerebro-spinal fluid in normal condition interferes to a certain extent with the hemolytic action of taurocholate of sodium." In cases of meningitis this special characteristic is very much pronounced. The reaction was positive in all cases of meningitis, but negative in other diseases of the nervous system. It has therefore a diagnostic importance especially in cases with a negative or doubtful leucocytic formula.

A bacteriologic examination is necessary in all cases. It reveals especially during the first period of the malady various microörganisms: pneumococcus, diplococcus, streptococcus, etc. As the cerebro-spinal fluid is a poor culture-medium, it is necessary to make cultures and inoculations. The presence of microörganisms decides the nature of the meningitis. One must bear in mind that the search for tubercular bacilli is not always successful.

Wassermann test is another valuable addition to the above mentioned investigations of the cerebro-spinal fluid. When it is found positive, the syphilitic nature of the meningitis cannot be doubted. As to the spirochætæ I have never succeeded in locating them in this fluid.

Recently Vincent and Bellot (*Presse Méd.*, 1911) have devised a new test for diagnosing meningitis caused by meningococcus and meningitis by other microörganisms. The test which is called **"precipitin reaction"** consists of adding one or two drops of antimeningococcus serum to a tube of freshly obtained cerebro-spinal fluid which has been cleared by centrifugation for ten minutes; the test-tube is then placed in an incubator at 52° C. (125.6° F.) for a few hours. In case of meningococcus meningitis precipitation will be present. This reaction is very important as it determines the therapeutic conduct: when it is positive, antimeningococcus serum should be used.

The diagnosis of acute meningitis presents sometimes difficulties in differentiating from **cerebral abscess** following otitis media. The localized symptoms and the character of the headache will in the majority of cases decide the question (see Cerebral Abscess).

Tumors of the brain show usually localizing symptoms, of which focal epilepsy is the most frequent. Besides, there is no elevation of temperature and the course is very much more prolonged than in meningitis.

Tubercular Meningitis will be recognized by its subacute course,

less intense fever and delirium and longer duration; finally by the existence of some tubercular focus and by the cytological examination of the cerebro-spinal fluid.

Cerebro-spinal meningitis will be recognized by the presence of the meningococcus in the cerebro-spinal fluid and by Vincent's precipitin reaction.

Treatment.—The frequency of fatal termination of the disease shows the failure of therapeutic measures. The treatment is therefore only symptomatic: antipyretics, purgatives, application of ice to the head, sedatives, narcotics are the only means at our command. Mercurial treatment should be tried in syphilitic cases. Bleeding and counter-irritants should be avoided, as they weaken the organism. Bathing is useful. Warm baths have a more beneficial effect than cold: they decrease the nervousness, relieve pain and contractures, produce diuresis. Saline infusions may be of benefit. In a case of a child under my care remarkable immediate results were obtained from the latter procedure (*Therap. Gazette*, 1902).

Lumbar puncture with evacuation of a certain amount of cerebro-spinal fluid relieves considerably intracranial hypertension and therefore relieves the headache. In some cases a prolonged evacuation has given satisfactory results: the needle is left in the spinal canal for several hours, but this procedure is not without some danger. As to injections of anti-meningococcus serum this will be discussed later.

Haines (*Transactions of the Ninth Internat. Otological Congress*, 1912) has recently devised a new surgical procedure in treatment of meningitis of any type of the disease except the meningococcus variety. If a free and continuous drainage could be established for the cerebro-spinal fluid, the intracranial pressure in meningitis would subside and the exsudate be reabsorbed. As in meningitis the excess of cerebro-spinal fluid crowds the brain against the skull, no operation could be successful when performed on the convexity of the brain: as soon as the brain tissue is exposed, it would crowd into the opening and become lacerated. In such cases no drain introduced could functionate properly. Haines argues that the space between the two poles of the cerebellum and the medulla, the **cisterna magna,** is in free communication with all other subarachnoid spaces about the brain and cord and with the cerebral ventricles. This space is most accessible to surgical attack. Opening of this space is not followed by a hernia cerebelli. A drainage established here is free and continuous. The operation consists of an incision in the mid-line from the occipital protuberance to the spinous process of the axis. A trephine is applied about an inch above the margin of the foramen magnum in the mid-line. If the occipital sinus is found

single, it is tied up at its upper part. If double, incision of the dura is made between them. After the dura is incised and a certain amount of fluid allowed to escape, a drain (gutta-percha) is introduced. Haines contends that if this operation is performed early, a cure may be obtained.

This operation may prove of value also in basal fractures.

Preventive Measures should never be neglected in cases of lesions of an infectious nature. Our modern knowledge of the etiology of meningitis (see above) indicates the care to be taken of traumatic or septic affections.

II. TUBERCULAR MENINGITIS

This form of meningitis is the most frequent. It is due toan infection. of the meninges by Koch's bacillus which has a special predilection for the pia-mater. In the majority of cases it occurs in very young children, especially between two and ten. H. Koch's recent investigations (*Ztschr. f. Kinderheilk.*, v, No. 5) show that the maximum number of cases occurred in the first four years of life with the largest number in the second year. It rarely occurs after thirty years. The infection is always **secondary:** it develops in individuals having a tubercular focus in some organ or tissue even in a latent state. The most frequent source is pleuro-pulmonary tuberculosis; next frequent seat is found in the mediastinal and mesenteric glands, in the bones, in the articulations, in the intestines and in the genito-urinary tract. The manner with which the infection is produced is not settled. It is generally believed that the bacilli reach the meninges through the arterial or lymphatic systems.

In considering the direct cause of tubercular meningitis sight should not be lost of the **predisposing** factors. Tubercular heredity plays an important rôle. Trauma, unfavorable hygienic surroundings, disturbances of nutrition due to some prolonged infectious disease (especially measles and whooping-cough), prolonged physical exertion and mental strain, alcoholism—are all causes of tubercular meningitis in a predisposed individual. Tubercular meningitis may develop in meninges already infected by other microbes. Thus a purulent meningitis may be followed by a tubercular meningitis. Meningococcus meningitis may terminate by a tubercular meningitis (Lion and LeBlaye, *Bull. et Mém. Soc. méd. des hôp. de Paris*, 1910).

Pathology.—The characteristic alterations of tubercular meningitis are predominant at the **base** of the brain. They are found principally in the area of circle of Willis, between the chiasma and the cerebral peduncles, in front of the pons and around the medulla. The lesion consists of (1) **miliary tubercles** and (2) common **inflammatory products.** The latter presents a serofibrinous (rarely purulent) substance which may extend

along the arteries toward the convexity of the brain; it covers the pia and sends out prolongations so that adhesions are formed between the convolutions and a more or less thick layer surrounds the branches of the Sylvian artery, the chiasma and the origin of the cranial nerves. The pia itself is congested, œdematous and thickened.

The most characteristic formation is found in the **miliary tubercles.** Along the blood vessels, and particularly at the level of their bifurcations are seen fine grayish nodules. They occupy exclusively the pia. These granulations or tubercles may adhere to each other and form groups. Sometimes they are very abundant and disseminated over the entire brain; but habitually they are localized along the blood vessels. In order of frequency they are found: at the base of the brain, circle of Willis, fissure of Sylvius, convexity of the hemispheres, finally the cerebellum, pons and medulla. Histologically they consist of the usual tubercular elements and a very large number of tubercle bacilli.

Besides the inflammation of the meninges, the blood vessels and the cortex are also involved in tubercular meningitis. Periarteritis, endarteritis and thrombosis are the vascular changes usually found in this affection. The above mentioned œdema of the pia as well as the changes in the brain substance are due to them to a great extent. The extension of the meningeal process to the superficial layer of the cortex will produce **encephalitis** (see Encephalitis) with its characteristic condition, viz. congestion of the blood vessels and œdema. Softening and hemorrhagic foci occur in the deep substance of the brain; they are the result of obliteration of blood vessels. The pia is intensely adherent to the cortex.

Tubercular meningitis is frequently accompanied by abundant exudation in the ventricles (hydrocephalus). When the amount of fluid is considerable, the ventricles, being distended, compress the convolutions, so that complete cessation of cerebral functions may take place, viz. coma and death. The choroid plexuses are thickened, the ependyma is softened and torn and sometimes tubercular granulations are seen on it. Tuberculous meningitis may extend to the membranes of the spinal cord. We find here congestion and serous or sero-purulent exsudates, also tubercular granulations. The latter follow the course of the blood vessels. The lesions predominate on the posterior surface of the cord and in its lumbar portion. The tubercular infiltration may extend to the nerve-roots and spinal ganglia.

Finally it is a common observation to find tubercular foci in viscera or other tissues, and particularly in the lungs.

Symptoms.—Three periods are to be considered: (*a*) prodromal, (*b*) period of cerebral irritation, (*c*) period of depression.

Prodromal symptoms are rarely wanting, especially in children. The little patient, who usually suffers from some latent tubercular focus, becomes silent, sad or unusually irritable, discontented; he loses his appetite, has gastro-intestinal disturbances, especially constipation, complains of vague pains. The adult becomes languid and is unable to do mental work; at the same time is irritable and restless. Then **insomnia** makes its appearance and becomes persistent. This is soon followed by **headache** and occasional **vomiting.** The child is pale, loses in weight, is feverish. The **fever** is usually **moderate.** The pupils are unequal and dilated. The pulse is irregular. Photophobia is present. Light reflex is sluggish. The reflexes are usually exaggerated. Convulsions are present; they are mostly localized and especially in the face or in the limbs. Contractures are characteristic and appear early. Kernig's sign is not frequent at the beginning, but Brudzinski's sign (see above) is constant. The contracture may affect the muscles of the eyes and produce strabismus. Retraction of the abdomen is quite constant. Twitchings, tremors, involuntary movements are observed. This condition may last from several weeks to months. The transition to the **second period** is gradual. Little by little the above symptoms become accentuated. Particularly three symptoms become conspicuous, viz. **headache, vomiting** and **constipation.** The **temperature** is usually moderate (100°–102°) and of a remittent character; its chief characteristic is the irregularity: sometimes it may undergo a transient rise, at another time it falls much below normal. The **pulse** is generally unequal and irregular; its changes are **not parallel** with those of the temperature: the latter may be abnormal by its rise, the former by its fall. Toward the end the pulse loses its irregularity but gains in rapidity. Respiration is also irregular. Deglutition becomes difficult and the pharyngeal reflex is lost. All these manifestations are indicative of bulbar involvement and render the prognosis unfavorable. The patient's **facies** is typical by its immobility. He shows impatience, desire to be let alone. Gradually **delirium** enters the scene: it is usually mild, with slight agitation and interrupted occasionally by a "hydrocephalic cry." (See Hydrocephalus.)

Ocular Symptoms.—Photophobia, pupillary inequality, ophthalmoplegia, nystagmus and œdema of the fundus—are all constant symptoms. They are due to the basal extension of the pathological process. These cranial nerve symptoms are of a great diagnostic importance.

The duration of this period is from ten to fifteen days. Its symptoms are indicative of destruction or softening of brain tissue (see Pathology). The contractures gradually disappear and are replaced by relaxation. At this stage palsies of one or two limbs occur and they usually precede the

generalized relaxation. Aphasia is not rare. Convulsions are rare in this phase. The patient gradually becomes stuporous, somnolent, the corneal reflex disappears. The condition may last several days. Coma enters the scene, the sphincters become relaxed, respiration difficult and of Cheyne-Stokes type, the body cyanosed. The temperature after a rapid rise falls far below normal. Death is inevitable.

Course, Termination and Prognosis.—Ordinarily the disease runs a sub-acute course. In the majority of cases the two main periods of the disease last two or three weeks, but the prodromal phase may last from one to several weeks. On the other hand **remissions** present a characteristic feature of the disease, so that the duration may be prolonged. Death is the natural termination in most of the cases, if not in all. Rare cases of recovery have been reported, but they were probably of non-tubercular nature, or cases of **localized** tubercular meningitis. The prognosis must be based not only upon the general symptomatology, but also upon the state of the cerebro-spinal fluid; in the majority of cases the latter alone will decide the tubercular or non-tubercular nature of the meningitis. Tubercular meningitis in children and in adults differs from each in some respects. The prodromal period is frequently absent in children. Instead of constipation there is diarrhœa. Convulsions appear early. The fontanelle is bulging. Rigidity of the neck and contractures in general are not pronounced. In adults mental symptoms are conspicuous: delirium is frequent; it is accompanied by a confusional state and sometimes by hallucinations. Motor symptoms are also prominent.

Diagnosis.—In a number of cases the diagnosis presents great difficulties, as on one hand there are affections (infectious for example) in the course of which cerebral symptoms may simulate meningitis, and on the other hand tubercular meningitis may remain latent for a considerable time and present at the beginning only a few vague symptoms. **Hysteria** sometimes presents the clinical picture of meningitis, including the fever. Similar symptoms may be observed in the course of **rachitis,** of **gastro-enteritis** (in infants), of **influenza,** of **uremia,** of **hereditary syphilis.** Although the differential symptoms characteristic of each of these diseases will enable us to recognize them in the majority of cases, nevertheless the diagnosis is not always possible without the aid of a new procedure which has proven to be of great utility. I speak of **lumbar puncture** (see chapter on Acute Meningitis).

The following are the characteristics of the cerebro-spinal fluid of tubercular meningitis. Ordinarily the fluid is clear but at close observation, especially when it is looked at through the opening of the tube, it will appear slightly greenish. It runs out under high pressure.

After standing, a slight coagulum will form. The albumen content is very high (1–2 grm.). Sodium chloride in normal conditions is 7 grm., 43 per liter. In tubercular meningitis its contents is 6 grm., 36 (Nobécourt and Voisin).

Normally there are very few cellular elements in this fluid. While, as we have seen, in acute meningitis, the polymorphonuclear leucocytes are abundant, in tubercular meningitis the **lymphocytes** predominate. If polynuclear leucocytes are met together with the lymphocytes, the former almost always show degenerative changes (J. Hohn, *Berl. klin. Wchn.*, 1912). **Lymphocytosis** (160 per cubic millimeter) exists not only in the typical forms, but also in cases with a latent and insidious onset. Recent researches have shown that lymphocytosis is the expression of any chronic morbid process of the meninges. Thus it is observed also in tabes, cerebro-spinal syphilis, paresis, and multiple sclerosis. It has therefore no absolute diagnostic value, although it will decide with absolute certainty the question whether a meningeal involvement occurring in the course of other diseases is of a tubercular or non-tubercular nature.

Another interesting peculiarity of the cerebro-spinal fluid is the **permeability of the meninges** for certain drugs, for example for iodide of potash or methylene blue. Normally the pia-arachnoid is not permeable from outside. Although the same permeability was observed in uremia, nevertheless this phenomenon is absent in other forms of meningitis but present in the tubercular. When a patient is given for several days any of these two drugs, it will be revealed in the cerebro-spinal fluid.

As to the specific bacillus, it is not always possible to find it in the cerebro-spinal fluid, but when present, the diagnosis is established. It is absolutely necessary to centrifugate the fluid immediately after the lumbar puncture, because the fibrin in precipitation englobes in its meshes the bacilli.

In every case of suspected tubercular meningitis the cerebro-spinal fluid should be examined from every standpoint, chemical as well as cytological, especially in cases in which the bacillus cannot be found. The above mentioned characteristics of the fluid are not absolute.

Treatment.—There is no specific medication for tubercular meningitis. The treatment is purely symptomatic. Headache will be relieved by cold applications to the head; antipyretics will be used for fever, sedatives and narcotics for restlessness and insomnia, frequent warm baths for contractures, purgatives for constipation. Iodids, also the usual anti-tubercular remedies, as creosote, guaiacol, etc., may be useful. As to surgical means, lumbar puncture may relieve cerebral compression, but it cannot be considered as a curative measure. A permanent drainage of the cere-

bro-spinal fluid has been recommended, but it is a procedure associated with some risk.

For Haynes' continuous drainage of "Cisterna Magna," see page 184.

III. EPIDEMIC CEREBRO-SPINAL MENINGITIS

Etiology.—The epidemic character of the disease is very important, as it is sufficient to differentiate it from other forms of acute meningitis. Sometimes it assumes a sporadic form. That the disease is contagious is admitted generally. It has been observed that the disease had spread through individuals coming from localities in which epidemic meningitis existed.

According to the latest investigations the etiological factor of this disease is the meningococcus (diplococcus intracellularis) of Weichselbaum.

Cerebro-spinal meningitis may develop during the course of influenza and be due exclusively to Pfeiffer's bacillus, but this is not the genuine epidemic form. Sometimes other pathogenic microbes may be found in the cerebro-spinal fluid in assoication with the meningococcus, such as pneumococcus, streptococcus, etc., but these are probably cases of secondary infection.

Finally, cerebro-spinal meningitis may be **purulent and aseptic,** in which no bacilli can be found in the cerebro-spinal fluid. It may run a similar course as the epidemic form, but sero-therapy has an unfavorable effect on it; its effect on the leucocytic contents is not the same as in the epidemic form.

The meningococcus is considered pathognomonic of epidemic cerebro-spinal meningitis, as statistical studies of epidemics have shown them to be present in eighty per cent. Epidemic cerebro-spinal meningitis is observed at all ages but it is particularly frequent in young children. In Koplik's statistics (*Med. News*, 1904) in 70 per cent. it occurred below the age of four.

The disease is observed more in winter and spring than in other seasons. The epidemics usually do not last longer than three or four months and develop as a rule slowly. The infection is transmitted from the germ carrier through the naso-pharyngeal cavity to the meninges, very probably via the blood vessels, but this question has not been entirely settled.

Pathology.—The lesions of the meninges found here do not differ materially from those found in other forms of suppurative meningitis. The exsudate, which is found in every form of acute meningitis (see this chapter), is here purulent. Pus is particularly situated along the veins and predominates at the base of the brain at the level of the chiasma, in the region of the fissure of Sylvius and around the cranial nerves. The ventricles are dilated and filled with a cloudy fluid. The choroid

plexuses are thickened and filled with pus. The ependyma is in a state of inflammation. In the spinal cord the pus is especially abundant on its posterior surface and in the lumbar region. The purulent exsudate develops sometimes with an extraordinary rapidity: in some cases twenty-four and even five hours after the appearance of the first symptoms.

The dura-mater of the brain is usually not affected, but is frequently congested in the cord. The brain and cord are usually intact, but they may also undergo softening in certain areas. Degenerative changes in the cortical cells, also in the cells of anterior cornua of the cord, degeneration of nerve fibers are not infrequently observed. The spinal roots and ganglia participate in the pathological process. The **cerebro-spinal fluid** obtained by lumbar puncture presents the following peculiarities:

(1) In **acute stage.** The fluid is cloudy. The sediment, obtained either by standing for several hours or by centrifugation, contains numerous polynuclear cells which are distinctly in a state of degeneration; they are deformed and stain poorly. Mononuclear cells and lymphocytes are in a small number (4 per cent.). Meningococci are found in the degenerated polynuclear cells. Albumen is increased, sodium chlorid diminished. Vincent's precipitin reaction (see page 183) is an important diagnostic element.

(2) In **the period of amelioration,** especially when antimeningococcus serum was used, a rapid improvement in the appearance and contents of the fluid is seen. It becomes clear and transparent. Lymphocytosis takes place of polynucleosis, the meningococci disappear.

The blood presents a marked leucocytosis reaching sometimes 50,000. Meningococcus may be found in cultures of blood.

As to alterations in other organs, they are usually those found in the course of other infectious diseases: exsudates in serous cavities (pleura, pericardium, articulations), hypertrophy of the spleen, etc.

Symptoms.—In the majority of cases the onset is sudden and the symptoms appear in the midst of perfect health. In some cases the onset is less abrupt and is preceded by a few prodromal manifestations, as headache and general malaise. At all events, the clinical picture at the beginning of the disease is almost identical in all forms of meningitis.

Headache is never absent and is unusually violent and tenacious. It radiates to the neck and along the **spine.** The latter becomes extremely tender to touch, so that the slightest movement of the body gives the patient excruciating pain. The entire body may become painful, but particularly the joints. **Vomiting** is always present and is of cerebral type (see Tumors of Brain). **Chills** are a constant initial symptom. These three symptoms are accompanied by a rise of **temperature**, as high as 102°. Soon

contractures make their appearance, they affect particularly the muscles of the neck. They are the most conspicuous symptom of the entire clinical picture. The **rigidity** of the neck is by far more pronounced than in other forms of meningitis. The head may be drawn back almost to a right angle. When the muscles of the back are affected, the body may assume the form of opisthotonos. Any attempt to move the body increases the rigidity. **Kernig's sign** (see Acute Meningitis) is almost always present. Brudzinski's sign (see page 177) is constant. **Convulsions** are frequent in children; they are usually generalized. Sometimes they are followed by hemiplegia or palsies of cranial nerves. Tremors are sometimes observed. The knee-jerks are frequently diminished or abolished. The toe phenomenon is frequently present. There is a general **hyperesthesia,** affecting the skin, musculature and joints. Vasomotor disturbances are present: alternating redness and pallor of the face are common. Tâches cérébrales are observed. The **eye** symptoms consist of dilatation or contraction and inequality of the pupils. Strabismus and diplopia are sometimes present. Suppuration of the conjunctivæ and of the middle ear may occur sometimes; the meningococcus and the pneumococcus have been found in the pus.

Herpes on the face and on the lips is quite frequent. Erythema of the skin is seen on the elbows, knees and buttocks. Purpuric eruptions are seen in very grave cases. In certain epidemics they are frequent, so that the term "spotted fever" is applied to them. In purpuric cases epistaxis is frequent. The **pulse** and **respiration** are very irregular: they vary from one individual to another. The **temperature** remains elevated during the entire course of the affection.

Delirium and **agitation** are generally present in the first period of the disease, especially in adults.

When the patient enters the **second phase** of the disease, the contractures and convulsions are substituted by **paralysis.** It may be hemi- or monoplegia, or else paraplegia. This occurs in prolonged cases or during convalescence. In cases with fatal termination the agitation is replaced by **depression :** the respiration and pulse become feeble, the delirium gives place to **coma.** In the fulminant form the entire course of the disease may last but a few hours.

Clinical Forms.—Cerebro-spinal meningitis does not follow the same course in every case. The **acute** form has a sudden onset with chills, violent headache, high fever and vomiting. The characteristic symptoms develop rapidly, convulsions make their appearance and the patient usually dies in a few days. In the so-called **fulminant** form the patient becomes comatose in the beginning and expires at the end of but a few hours. The

protracted form is characterized by remissions and phases of amelioration and aggravation; it may last weeks and months. In case of recovery there are always sequelæ: paralysis, psychic disturbances, blindness or deafness and in infants chronic hydrocephalus. Cases have been also reported in which the patients presented mild symptoms and were not bed-ridden; the malaise, Kernig's sign, rigidity of the neck and lumbar punctures made the diagnosis unmistakable; this is the so-called **ambulatory form.**

During epidemics an **abortive** form of cerebro-spinal meningitis is sometimes seen. The patient feels fatigued, has some fever; headache and rigidity of the neck develop. All these symptoms disappear in a few days. During an epidemic all manifestations of an infectious character, no matter how mild they may be, should be looked upon with suspicion. In such cases the meningococcus should be investigated not only in the cerebro-spinal fluid but also in the naso-pharyngeal cavity.

Closely connected with epidemic cerebro-spinal meningitis is the **posterior basic meningitis** of the English writers. It was first described by Gee and Barlow in 1878 and the organism isolated by Still in 1898. The organism resembles greatly Wechselbaum's meningococcus. The difference lies in some cultural characteristics. The most striking symptoms are according to the English writers: early head retraction, early blindness and subacute course of the disease. Recovery is frequent, but blindness may remain permanent. The distinction between posterior basic meningitis and the epidemic cerebro-spinal meningitis as well as the precise difference between Still's and Wechselbaum's meningococci are not definitely established.

Prognosis.—It depends upon the form of the affection (see above) and upon the severity of the epidemic. Generally speaking it is grave, in spite of sero-therapy. It is especially serious in infants. The future of the patient is of a great concern in view of the complications accompanying cerebro-spinal meningitis (sequelæ).

Sequelæ.—The most important sequelæ are those showing involvement of the nervous system. Encephalitis and myelitis are the most frequent. **Paralysis** (hemiplegia, monoplegia or paraplegia), ataxia, neuritis, are very common complications. Cranial nerve palsies are quite frequent. **Hydrocephaly** is not rare. It is the result of ependymitis and inflammation of the choroid plexuses. **Otitis interna** is the most frequent: deafness is the result and it is usually bilateral. **Ocular complications** are frequent. They are: mydriasis, inequality of pupils, palsy of ocular muscles, iritis, papillitis and optic atrophy. **Psychic sequelæ** are not rare. They are: change of disposition, irritability, diminution of intelligence.

Diagnosis.—As cerebro-spinal symptoms may develop in the course of

infectious diseases, the diagnosis sometimes presents difficulties. There are forms of cerebro-spinal meningitis caused by Pfeiffer's bacillus (influenza) or Eberth's bacillus (typhoid fever). The character of the cerebro-spinal fluid, viz. its bacteriology, chemistry and cytology (see above), the character of the onset, the course of the affection in addition to the existence of an epidemic, will enable to make a correct diagnosis. For differential diagnosis with other forms of meningitis see the preceding chapters.

Tetanus presents trismus at the beginning, but this is rare in cerebro-spinal meningitis. Moreover in the latter affection the contractures are much more easily reducible than in the former.

Hysteria may sometimes simulate the disease, especially by its rigidity of the neck, but absence of fever and the presence of special stigmata in addition to the lack of other symptoms will make the diagnosis of hysteria easy.

Uremia will be differentiated by the absence of fever, herpes and other symptoms.

Treatment.—The general principles are those of other forms of meningitis. There are no specific medications. The symptomatic treatment is the only one that can be applied. Sedatives (bromides, chloral, morphin, coal tar products) are useful to combat insomnia and pain. If the heart is not very active and the blood pressure not very high, ergot given hypodermatically may be of some benefit. An adult should receive a hypodermic syringeful (30 m.); a child five years old, one-fourth of this dose (7 1/2 m.); a child of ten, a half (15 m.). Ergot contracts the blood vessels, relieves congestion, quiets cerebral excitement. Hot baths of ten to twenty minutes' duration repeated every three or four hours according to patient's general condition and kept up during the entire course of the disease have been recommended. Some improvement has been also obtained from saline infusions. Some satisfactory results are being reported from repeated lumbar punctures. But all these measures are only palliative.

Sero-therapy appears to be the only means of curing epidemic cerebro-spinal meningitis. Flexner and Jobling in this country have prepared an antimeningococcus serum which appears to give excellent results. In Germany Kolle and Wasserman, in France Dopter, and in Austria Markl have also devised sera which give equally good results. The earlier the serum is used the better results can be expected. A lumbar puncture is made as soon as the disease is suspected, about 30 c.c. of the cerebro-spinal fluid is allowed to escape and an equal amount of serum injected through the same needle. If after one dose improvement is noticeable no further injection is necessary. In the majority of cases improvement

is noticed after the first injection. Headache, delirium, insomnia, fever, are the first to show amelioration. Rigidity of the neck and other contractures persist longer. The cerebro-spinal fluid shows progressive improvement: from cloudy it becomes clear: the polynuclear cells are less altered, the lymphocytes become predominant; albumen decreases; the precipitin-reaction (Vincent) becomes negative. In cases in which the first injection is not followed by improvement, a larger dose of serum should be given daily for four days. After the four injections, wait two or three days, and if no improvement, resume the injections. Flexner's statistics since the use of his serum are striking. An analysis of his 523 cases shows a mortality of 25.4 per cent. In Dopter's cases (339) the mortality was 11.80 per cent. The German statistics show a mortality of 18.35 per cent. Considering the tremendous mortality (80 per cent.) before the introduction of the serum, the latter is a great therapeutic acquisition. The duration of the disease at present is but eight to ten days. There is also a marked diminution of the sequelæ. The greatest success is obtained when the injections are made at the earliest possible moment. In a series of 328 cases Flexner obtained 85 per cent. recoveries when the injection was made from first to the third day, 78 per cent. when it was made from the fourth to the seventh day and 61 per cent. when it was made after the seventh day. Sero-therapy does not cure every case of meningitis. It fails in the fulminant cases, in cases of associated meningitis, especially in tubercular form, finally in delayed cases. In grave cases Flexner advises two injections a day. The same is to be done when the cerebro-spinal fluid remains cloudy and contains meningococci. As to the dose, 10–15 c.c. are sufficeint for a child of two; 30 c.c. in an older child, 30–40 c.c. in an adult. Sometimes the dose must be increased. Flexner in 21 cases had to use 200–300 c.c. per dose. Each injection should be preceded by an evacuation of an equal or rather greater amount of cerebro-spinal fluid. The lumbar puncture should be made in the fourth space. The patient is placed on his side and the dorsum is curved as much as possible. As soon as the injection is made, the patient is placed on his back, the head is lowered, and he is kept in this position at least two hours.

Relapses may occur. Of 1,294 patients treated by Flexner, 56 showed relapses after a greater or less interval of time. Prompt resumption of the serum injections often, but not invariably sufficed to control the reinfections. Of the 56 patients, 40 recovered, the rest died. Relapses therefore respond less well to the serum than the primary infections (*J. of Experim. Med.*, May, 1913). The same author observed that complications and sequelæ are favorably influenced by the serum.

The least influenced is deafness, because injury to the internal ear takes place very early and sometimes before the diagnosis of meningitis has been made. Arthropathies have been shown to be amenable to direct injections of the serum. The tendency to hydrocephalus in the young has been diminished and the intraventricular injection of the serum in several cases controlled the infection and inflammation of cerebral ventricles and reestablished communication between the latter and the subdural space of the spinal cord. Recovery has been rendered complete.

Sophian (*J. Am. Med. Ass.*, 1913) has recently formulated special rules for a safer and more efficient method of administration of antimeningococcus serum. From an extensive experience he ascertained that the state of the blood pressure is an excellent guide for both the safe withdrawal of cerebro-spinal fluid and injection of serum. As soon as the removal of fluid commences, there is a drop in the blood pressure. Considering an average blood pressure of 110 mm. of mercury, further withdrawal of fluid should stop if the drop of blood pressure is 10 mm. in adults and 5 mm. in children. The serum is injected by gravity method better than by syringe. The entrance of serum in the sub-arachnoid space is usually followed by a drop of blood pressure and continues to drop. After it had dropped 20 or 30 mm. of mercury, the further dropping is much faster if more serum is injected. Sophian finds that a total drop of 20 mm. of mercury in an adult with average blood pressure of 110 to 120 mm. of mercury is a safe indication to stop further injection of serum. The average dose of serum in adults, he finds, is 20 to 25 c.c. and it is rarely necessary to inject more than 40 c.c. of serum. The symptoms of drop in blood pressure are: stupor, irregular respiration, slow and irregular pulse, dilatation of the pupils, incontinence of urine and feces. In case these symptoms become pronounced, as much fluid as possible should be removed and artificial respiration begun. Intramuscular injections of epinephrin and atropin should be administered.

The injections of serum controlled by blood pressure have given Sophian more satisfactory results than with the old method.

The immediate object of antimeningococcus serum is to dissolve and inhibit the multiplication of meningococci. If the exsudate in the spinal canal is thick, the diplococci may not be accessible. To render them accessible D. I. Hirsch suggested recently (*J. Amer. Med. Assn.*, 1913) irrigation of the spinal canal before injecting the antimeningococcus serum. He advises the preliminary irrigation in every case of cerebro-spinal meningitis. He removes from 30 to 40 c.c. of fluid by lumbar puncture; then with a small syringe, using gentle pressure, he introduces 30 c.c. of normal salt solution 100° F. into the canal; he allows this to

return; he repeats this three times in those cases in which the fluid is cloudy, and in purulent cases repeats until the fluid returns clear. Then only he injects the serum. He reports two cases in which the results of this treatment were highly satisfactory.

Serum Accidents.—Sometimes unpleasant symptoms follow injections of serum. They are: skin eruptions of the type of urticaria; rise of temperature; disturbance of respiration, convulsions, and in a few cases a comatose state. All these complications are fortunately rare.

Prophylaxy of Cerebro-spinal Meningitis.—Isolation of patients and of the germ carriers when found and disinfection of the naso-pharyngeal cavity and of all the surroundings are the best means to prevent the spreading of the disease.

IV. CHRONIC MENINGITIS

As a **secondary** affection chronic meningitis is not rare. It is quite frequent in syphilis of the nervous system, in disseminated cerebro-spinal sclerosis, in tumors and in focal lesions of the brain, also as sequelæ of acute meningitis. It is the essential lesion of paresis.

There is also a **primary** form of chronic meningitis (leptomeningitis). It is met with in **chronic alcoholism** and **traumata.** Similar to pachymeningitis (see this chapter), it affects preferably the convexity of the brain. The pia-mater is thick and adherent and it is difficult to separate it from the cortex. At the base it occurs very exceptionally. If it does, it is mostly of syphilitic nature. The ventricular ependyma often participates in the morbid process, particularly in the fourth ventricle: it is much thickened and the fluid is increased.

Symptoms.—They are vague, when the localization is on the convexity (in alcoholism). Diffuse headache, which is deep and very severe, also shooting pain along the course of cranial nerves, paralysis, especially hemiplegic form, disturbance of speech, papillary œdema, sometimes delirium, finally intellectual enfeeblement, constitute the whole symptomatology. When the base is involved, there are also palsies of cranial nerves. The chief characteristic of chronic meningitis is the fact that all these symptoms are not encountered in combination as is the case in acute meningitis, but only one or two of them are present at one time and become fixed and persistent. The course of the disease is very slow and death is the rule.

Diagnosis.—The affection may be confounded with cerebral tumors and cerebro-spinal syphilis. See these chapters.

Treatment.—It can be only symptomatic.

Aseptic Cerebro-spinal Meningitis.—Remlinger (*C. R. de Soc. de Biol.*, 1911) investigated this form of meningitis in a large number of soldiers in the army. Clinically there is no essential difference between it and the epidemic form, except that the prognosis is good. Lumbar puncture has a favorable effect on the disease. Antimeningococcus serum is harmful. The cerebro-spinal fluid is cloudy. In the sediment obtained from centrifugation are seen very much altered polynuclear cells. No microörganisms can be found. Vincent's precipito-reaction (page 183) is negative. Cultures also remain sterile. The disease has no epdemic character: the cases remain isolated. The meningococcus cannot be found in naso-pharynx or in any other part of the body.

CHAPTER XIII

THROMBOSIS OF THE INTRACRANIAL SINUSES

For the anatomical relations of the sinuses see the chapter on the Circulation in the Anatomy of the Brain.

Pathology.—The thrombus presents a grayish or reddish clot adherent to the walls of the sinus. It may affect only a part of the sinus or occupy its entire length and even extend into the tributary veins. Usually the clot does not entirely fill the lumen of the sinus. The resulting venous stasis produces a hyperemia and extravasation. Œdema is considerable. The cerebral substance becomes softened. The ventricles are distended.

The phlebitis, which caused the thrombosis, may suppurate. In such cases the thrombus is purulent and metastases are then often seen in various organs; cerebral abscess or purulent meningitis may also follow.

Etiology.—(a) **Primary thrombosis** of the sinuses, also called **"marantic thrombosis,"** occurs in individuals with a low vitality, as in tuberculosis, in cancer, in chlorosis, in protracted diarrhœas or in any disease of long standing, during which malnutrition and exhaustion are conspicuous. It occurs most frequently in children and in the aged. The cause of thrombotic formation lies in a weakness of the heart, produced by the above diseases, and therefore in retardation of circulation and increase of the coagulability of the blood. The longitudinal sinus is most frequently involved.

(b) **Secondary thrombosis** (inflammatory, infective) is due to a lesion in the neighborhood of the dura. The inflammation of the sinus may be the result of the direct contact with the diseased area or may occur through the veins which carry the infection to the sinus. Thus thrombophlebitis follows disease of the skull or face or of the ear. Caries of the petrous bone is frequently the cause. In the latter case the sinuses affected are: lateral, superior and inferior petrosal. Diseases of the orbital and the nasal cavities, of the pharynx, of the mouth, periostitis of the maxillary bones, erysipelas of the face, are all causes of thrombophlebitis. As to the microörganisms in the thrombus, nothing definite is known: streptococcus appears to be the most frequent.

Symptoms.—They are **general** and **special.** The **general** symptoms are: mental hebetude, somnolence, headache associated with vomiting, rigidity of the neck or of the extremities, sometimes convulsions. Optic neuritis is frequently present, especially in cases of chlorosis. In secondary or infective thrombosis there are also rigors followed by profuse perspira-

tion and elevation of temperature; œdema in the vicinity of the sinus; pain in the abdomen, diarrhœa may be present in some cases; great restlessness with delirium may be present in others. The **special** symptoms depend upon the sinus involved.

1. **Longitudinal sinus:** Distention of the temporal veins; cyanosis of the face; tension of the large fontanelle in children; epistaxis.

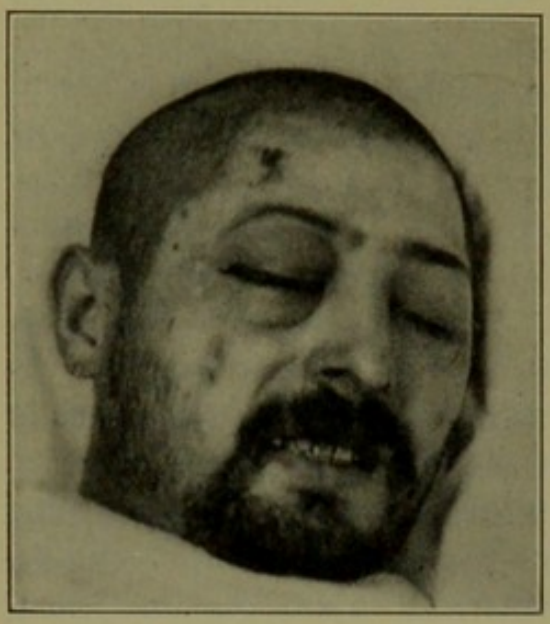


FIG. 77.—THROMBOSIS OF RIGHT CAVERNOUS SINUS. ŒDEMA OF RIGHT SIDE OF THE FACE. EXOPHTHALMOS OF THE RIGHT EYE.

2. **Transverse sinus:** The internal and external jugular veins are empty. The mastoid region is œdematous and painful.

3. **Cavernous sinus:** Stasis and œdema in the ophthalmic vein. Œdema of the orbit and of the eyelids. Congestion and œdema of the retinal blood vessels. Exophthalmos. Impairment of vision. Paralysis of the third and sixth nerves, also of first branch of the fifth nerve. The accompanying photograph is taken from a patient seen with Dr. Gittelson at the Mount Sinai Hospital, who suffered from a thrombosis of the right cavernous sinus. Autopsy verified the diagnosis.

Eye symptoms in sinus thrombosis have sometimes a diagnostic value. The following are the most important ocular manifestations.

(a) **Marantic form** of thrombosis. Here the eye disturbances are not as frequent as in the infective form.

Venous stasis and hyperemia in the fundi, optic neuritis, fixed pupil (stauungspapilla)—are all occasionally met with. More frequently are met disturbances in ocular movements, particularly conjugate deviation of the eyes. In such cases both sinuses, longitudinal and transverse, are simultaneously involved. The third nerve is oftener affected than others. Exophthalmos is rare, and if it does occur, there is an involvement of the cavernous sinus and of the ophthalmic vein.

(b) **Infective Thrombosis.**—Apart from otitic and traumatic cases, eye symptoms are very frequent in septic thrombosis. According to Uhthoff—hyperemia of the papillæ, optic neuritis, fixed pupils, optic atrophy—occur in 20 per cent. of cases. The involvement of cavernous sinus in septic sinus thrombosis occurs in 80 per cent. This explains the frequency of eye disturbances. Exophthalmos is the most frequent

symptom. Uhthoff observed it in 72 per cent. of cases. When the sphenoid bone is affected with secondary septic thrombosis of the cavernous sinus, the exophthalmos is bilateral. Palsy of ocular muscles is almost a rule when the cavernous sinus is involved. In sinus thrombosis of **otitic origin** visual disturbances and changes in the fundi are of great diagnostic value. The latter are frequently bilateral like in other intracranial affections. Immobility of pupils develops quite rapidly. Jansen found fixed pupils in 30–40 per cent. of cases. Optic atrophy according to Uhthoff is found in 24 per cent. of cases. Exophthalmos is by far less frequent in thrombosis of otitic origin than in other septic forms of thrombosis. Palsy of ocular muscles occurs when the cavernous sinus is involved.

Duration. Termination. Prognosis.—The secondary thrombo-phlebitis has a different course from the primary (marantic). The sudden onset, the rigor, the violent headache and the very high temperature are indicative of a purulent state. Its duration is usually a few days. It may be complicated by an abscess in the temporo-sphenoidal lobe. The marantic form is more insidious in onset and longer in duration. Death is the usual termination.

Diagnosis.—Appearance of cerebral symptoms in an individual suffering from any of the diseases mentioned in etiology should arouse suspicion in regard to a phlebitis and obstruction of the sinuses. However the symptoms may simulate meningitis, but the **special** signs characteristic of each sinus-obstruction will enable to reveal thrombosis.

The following features will enable one to distinguish between a marantic and an infective thrombosis. The usual seat of the first is the single sinuses, such as the longitudinal; the seat of the second is in those that are bilateral. The first has a tendency to become organized and absorbed; the second, to suppuration. In the course of the first cerebral hemorrhages occur; in the second hemorrhages are rare. The first has a tendency to softening which is not the case in the second. Septic metastatic infections are frequent in the second, rare in the first. Purulent meningitis, cerebral abscess, are common in the second, but absent in the first (Macewen).

Treatment.—Prophylactic measures are the most important. The most rigorous attention should be given to diseases of the ear and others to prevent secondary infection. The medical treatment of thrombosis will be purely symptomatic. In the marantic form tonifying measures are necessary. The original disease must be treated most energetically. Saline infusions copiously given have given me satisfactory results. The surgical treatment is advisable only in the secondary form of thrombo. phlebitis. Opening and curetting of the affected sinus may sometimes be of benefit.

CHAPTER XIV

CIRCULATORY DISTURBANCES OF THE BRAIN

ANEMIA AND HYPEREMIA

Anemia (Generalized)

Pathology.—The meninges and the cortical substance are pale. Sometimes œdema with effusion between the convolutions is observed.

Etiology.—It occurs most frequently in the two extreme ages of life. **Hemorrhages** more or less abundant; rapid **evacuation** of pleuritic or ascitic fluid; **cardiac diseases,** diseases of the blood; chlorosis, pernicious anemia, leukemia; inanition; **intracranial** pressure (tumors, hydrocephalus); **embolism** or **thrombosis** of cerebral arteries; **lead** or **nicotin** intoxications —these are the usual causes of cerebral anemia in adults. Finally vascular constriction leading to anemia may be caused by intense **emotion** and by certain **drugs,** viz. ergot, belladonna, chloroform, etc.

Symptoms.—They are different in the **acute** and **chronic** forms.

Acute.—In anemia of a sudden onset the patient becomes dizzy. This is accompanied by a noise in the ear and disturbance of vision. Vomiting is quite frequent. Convulsive movements are not rare. The heart is slow. Arterial pressure is increased. An imperative somnolence sets in early and coma may follow; pupils then become dilated, the reflexes are lost, the respiration is difficult. The blood pressure falls, the heart becomes accelerated. Death occurs soon. If the cerebral circulation is reëstablished, the above symptoms rapidly disappear. In milder forms convulsions and vomiting are absent. In exceptional cases optic atrophy has been observed.

Chronic.—This variety is met with mainly in chlorotic patients. The psychic symptoms are the most conspicuous. Headache, deficient memory, difficulty of mental application, amnesia, irritability or else marked depression are frequently observed. Insomnia, vertigo, occasionally hallucinations and delirium are also met with in chronic cerebral anemia.

In **young children** Marshall Hall observed a form of cerebral anemia to which he gave the name "hydrocephaloid" from its resemblance to hydrocephalus. It occurs mostly in exhausting diarrhœas. This infantile anemia has a period of agitation followed by a period of depression. The little patient is pale and apathetic; the pupillary light reflex is lost; the pulse and respiration are irregular; death occurs in coma.

In **old individuals** cerebral anemia is the result of an atheromatous condition of the blood vessels. Headache, vertigo, confusion are the main symptoms.

Prognosis.—It is favorable in mild forms. Convulsions are a grave omen. Speaking generally, the prognosis depends upon the cause.

Diagnosis.—As the symptoms of cerebral anemia and hyperemia are similar, the diagnosis presents in this respect great difficulties. Potain's rule will enable to differentiate; in hyperemia **lowering** of the head will produce vertigo; in anemia **raising** of the head will cause it.

Treatment.—In the **acute** form the horizontal position of the head is the first indication. The increase of blood in the head can be aided by placing a binder on the lower extremities. Cardiac stimulants, especially alcohol, should be resorted to. Artificial respiration and saline infusion are beneficial. In the **chronic form** the treatment will be that of vascular diseases or of anemia.

Hyperemia

Pathology.—The blood vessels and the sinuses are overfilled with blood. The ventricles and sub-arachnoid spaces present an increase of cerebro-spinal fluid. The cortex and white matter are reddish. In persistent passive hyperemia changes in the cortical cells and especially in Purkinje's cells have been observed.

Etiology.—Hyperemia may be **active** and **passive.**

Active cerebral congestion may be due to any cause which produces vaso-dilatation of cerebral vessels. Infectious diseases, insolation, alcohol and other intoxications, organic cerebral affections are the usual causes of active cerebral congestion. Sudden contraction of the peripheral vessels, the transition of sleep to the waking state, a physical or mental effort, sensory excitement which leads to a rise of arterial pressure, are also accompanied by cerebral hyperemia.

Passive congestion occurs mostly in cardiac diseases during the period of asystoly; in chronic pulmonary diseases, in emphysema. Finally tumors or any mechanical obstruction compressing the jugular veins or the abdominal aorta will also produce a passive congestion of the brain.

Symptoms.—In **active** congestion the initial symptoms may be **acute** and **subacute.** In the acute form the onset is very sudden and loss of consciousness may occur. Fortunately the symptoms usually improve rapidly, but a transient aphasia or hemiplegia may follow. Sometimes epileptiform convulsions with or without loss of consciousness may accompany an acute attack. In some cases delirium may be the initial symptom.

In the **subacute** form the patient feels a rush of heat to the head; sees a peculiar flickering before his eyes; has a throbbing in his temples, has headache, is apathetic and somnolent. The pulse is small, the facies is cyanosed, the conjunctivæ are injected.

The **passive** cerebral congestion is essentially chronic. Headache, persistent insomnia, vertigo, depression, finally turgid face—are the usual symptoms.

Diagnosis.—The transient character of the paralytic attacks of the grave form of acute cerebral congestion will enable to differentiate from similar attacks in apoplexy, in which the history of the case and the course decide the diagnosis. The same can be said of the epileptiform seizures. From cerebral anemia it will be differentiated by Potain's rule (see Anemia).

Prognosis.—It is usually favorable, if the condition is not due to a cardiac affection. If the heart is diseased, there is always danger.

Treatment.—Removal of the cause is the first indication; consequently prophylactic measures for preventing excesses or other causes leading to congestion are the most important.

In an attack the following means should be adopted. Bleeding, leeching, blistering, purging are well-known measures. The head must be raised. Ice applied to the head gives relief. Hot foot baths are valuable. Internally bromides are advisable, but opiates and chloral should be avoided. In passive congestion removal of tumors or of other mechanical obstructions is directly indicated.

CHAPTER XV

FRACTURES OF THE SKULL

The study of this subject is very important. As it frequently happens, the intracranial contents are almost invariably involved in fractures of the skull. The knowledge of the seat of the injury, of its character, of its possibilities, of the degree of involvement of the intracranial tissue and of the seat of the latter will enable one to make a prompt decision as to immediate or remote surgical intervention.

Fractures may be **incomplete** or **complete.** In the incomplete variety there may be either a fracture of the **outer** or of the **inner table** of the skull. The former is observed chiefly in regions where the two tables are separated, like the frontal sinus. Isolated fractures of the inner table are rare. Complete fractures affecting both tables simultaneously are not rare. In this case the fracture may be only a fissure, linear or else present separate fragments.

Another important variety is the **radiating** one. This form is the most frequent in fractures of the base of the skull. The radiation towards the base is done by the shortest possible route. A fall on the frontal region will produce a fracture which will reach the anterior portion of the base. In a fall on the temporal region the fracture will reach the middle portion of the base. A fracture in the occipital region will reach the posterior portion of the base. The most frequent of all is the fracture of the middle portion of the base. The reason of it lies first in the frequency of falls on the temporal region and second in the thinness of the cranial wall at this level. The fracture reaches the middle cerebral fossa and ends either in the sphenoid bone or in the petrous bone. In the latter case very important elements may be involved, viz. the external auditory meatus, the internal ear, Fallopian canal, internal auditory meatus. The fracture may spread either forward on the same side and involve the chiasma or reach the other side, or it may extend posteriorly to the cerebellar region. An important variety of fractures is the one **par contre-coup.** Here the lesion lies in another portion of the skull than in the one that was injured. Its localization is frequently either the occipital region or the petrous bone, but most frequently the orbital region.

Fractures of the skull are frequently followed by involvement of the membranes, brain itself and cranial nerves at the base. The dura is either

detached or torn. The blood vessels, the sinuses, may also be torn, hence extra- or intra-dural **hematoma.** If the arachnoid suffers, the cerebro-spinal fluid will find its way through the opening in the skull, or through the nasal fossæ or else through the external auditory meatus. A tear in the pia will produce a hemorrhage. Contusion of the brain is constant. The cranial nerves at the base are frequently involved.

Symptoms.—When the fracture is open, besides the fragments or depression of the bones we may notice cerebro-spinal fluid or brain tissue oozing through the opening. When the fracture is closed, the chief symptoms are: swelling of the scalp, depression of the bone and pain. As to recognition of depressions, one must always bear in mind a natural congenital anomaly of the skull or else a deformity caused by a previous injury; in such a case there will be no swelling of the scalp. Hematoma of the scalp may sometimes give the impression of a fracture.

In fractures radiating towards the base, the following symptom is most pathognomonic: **bleeding from the nose, ear and mouth.** Nasal bleeding may of course originate in the nose itself in case the latter is fractured. In such a case the diagnosis will be made from pain and swelling in the nasal region, also crepitation of the nasal bones. Bleeding from the mouth is the result of fracture of the sphenoid or of the roof of the pharynx. The bleeding from the ear is the result of fracture of the petrous bone (see Anatomy above). A bleeding from the ear may be due to a tear in the tympanic membrane, but then the bleeding is slight and very brief. Besides, in the latter case the disturbance of hearing is much less marked than in fractures of the petrous bone. Moreover in basal fractures we find facial palsy and total deafness. Sometimes the cerebro-spinal fluid finds its way through the ear, more rarely through the nose. In basal fractures ecchymoses of the conjunctivæ, the eyelids, the mastoid and the neck may occur. The first is the most frequent. It is due to infiltration of the cellular tissue of the orbit with blood originating in a fractured focus of the anterior portion of the base.

A very important diagnostic point lies in the presence of **blood in the cerebro-spinal fluid,** and in the **high tension** under which it appears upon lumbar puncture. In order to avoid errors, it is advisable to collect the fluid in three successive tubes. If only the first tube is colored,then the blood comes from a blood vessel during the lumbar puncture. If the coloration is the same in all the three tubes, the blood is in the cerebro-spinal fluid. The blood is due to its entrance from the torn blood vessels into the sub-arachnoidal spaces.

In addition to these physical signs there are also **functional disturbances.**

First of all the **shock** more or less marked immediately after the injury.

Then the cerebral manifestations which depend upon the area involved and which may appear either immediately or some time later. Thus we may have epileptiform convulsions, disturbances of sensations and paralytic phenomena, such as monoplegia, hemiplegia or paraplegia (see Cerebral Localizations). **Paralysis of cranial nerves** will give valuable data for localization. The most frequent occurrence in fractures of the base is paralysis of the seventh and eighth nerves.

When the **facial palsy** follows immediately after the accident, it is an indication that the nerve is completely torn within the Fallopian canal. If the facial palsy appears several days after the fracture, it is due either to compression by the extravasated blood, or to the newly forming callus or else to a secondary periostitis. In these cases the facial palsy is curable. The palsy is of the peripheral type.

The **auditory nerve** may be totally injured. The involved cochlear branch produces impairment of hearing or deafness, while the injured vestibular branch causes vertigo, loss of equilibrium and tinnitus.

Paralysis of nerves supplying the **ocular muscles** is not rare in fractures of the skull especially at the base. The most frequent is palsy of the sixth nerve. It follows fracture of the apex of the petrous bone. The symptoms are: internal strabismus with diplopia. Paralysis of the third nerve is exceptional.

Prognosis.—Fractures of skull are always a serious condition, not only on account of immediate but also of remote consequences. Speaking generally fractures of the vertex are less serious than those of the base. The outlook depends upon the intensity of nervous phenomena, vascular lesions, pressure of cerebro-spinal fluid during a lumbar puncture, infectious symptoms of meningo-encephalitis, sinus phlebitis. The most important factor which determines the gravity of any given case is the hemorrhage: the more abundant the latter is, the more serious is the outlook. When the fracture runs a favorable course, it usually ends in formation of a callus or when the separation of the fragments is considerable, a newly formed fibrous membrane takes the place of the callus.

Treatment.—It is the domain of the surgeon to take special care of the wound, to avoid secondary infection by means of most rigorous aseptic and antiseptic measures. In case of localized symptoms, such as paralysis, convulsions or sensory disturbances, an operative intervention is indicated. In cases of depression of the skull even without immediate symptoms, an operation should be undertaken, because cerebral symptoms of paralytic or convulsive nature may develop later. As to the nature of the operation, large osteoplastic flaps give the most satisfactory field for examination of the underlying soft tissue and for a more complete removal of the accumu-

lated blood. In cases in which localized manifestations are on the same side as the fracture, it is advisable first to operate at the seat of the fracture. If the results are not satisfactory, operate on the opposite side of the brain.

In fractures of the base which is not accessible, Cushing obtained encouraging results from decompressive operations in the subtemporal region (Ann. of Surgery, 1908).

Recently **lumbar puncture** has been recommended in cases of fractures at the base of the skull. The diagnostic value of lumbar puncture has been mentioned above. Very favorable results have been reported in traumatic cases treated by puncture of the spinal canal and extraction of certain amounts of the cerebro-spinal fluid (Lenhartz, Meslier, Muret, Quénu, Fowler and others). The advantage, of this procedure are: (1) relieve intracranial pressure and through it the cerebral phenomena. (2) remove partly the microörganisms which are the cause of secondary meningeal manifestations; blood is an excellent medium for development of germs.

In cases of injury to the head without an evident fracture of the skull, expectant treatment is most advisable. The patient is kept quietly in bed and watched very closely. At the appearance of localized or other disturbances, the therapeutic conduct is promptly to be decided upon.

CHAPTER XVI

CONCUSSION OF THE BRAIN

THE subject of concussion of the brain is of practical importance. The common belief that only in injuries accompanied by loss of consciousness there is concussion of the brain, is erroneous. There are many traumatic cases in which there is no genuine loss of consciousness, but only a vertigo, or a transient mental hebetude, or else a hazy recollection of the trauma—all cases in which symptoms of a grave nature may develop immediately or some time after the accident. An appreciation of these milder forms of cerebral concussion is of importance from a therapeutic standpoint, as if they are ignored or overlooked, serious consequences may follow.

Pathogenesis.—The nature of concussion is still debatable. As autopsies are exceedingly rare, a pathogenesis cannot be established with any degree of precision. The following views are being held:

(1) There is no material lesion in the nervous system and the disturbances are purely dynamic or else they are due to some perturbation in the cerebral circulation.

(2) There are some material lesions: they may be seen either macro- or microscopically in the medulla and affecting particularly the cardiac and respiratory centers.

(3) To others the reason of death lies in anemia of the respiratory center. The anemia is the result of a compression of the blood vessels produced by œdema of the brain. The œdema is caused by a paralytic vaso-dilatation brought on by the trauma.

Concussion or shaking, jarring of the brain which will be discussed here as occurring without a gross injury of the brain may result from a direct injury, such as a fall on the head or a blow, and from an indirect injury, such as striking any other part of the body during a sudden fall, landing suddenly on the feet, falling on the buttocks, etc. In all these accidents there may be a profound loss of consciousness which may develop immediately or else the injury may be followed by a very slight and very brief unconsciousness, also by no loss of consciousness. In the latter cases almost invariably symptoms will develop within a few hours or even a few days.

Symptoms.—In the severe cases after the patient recovers consciousness, he will present for some time a mental hebetude, a vague expression of the face. He will complain of pain in the head especially in the occi-

pital region, of noises in the ears. He will suffer from insomnia; he is irritable and at the same time apathetic. He is mentally dull, cannot concentrate his thoughts. He shows exhaustion upon the least exertion. On the least attempt to do anything, he gets covered with perspiration. If he falls asleep, he awakens often. The pulse presents this characteristic feature, that it is easily compressible and increases in rapidity while the patient is under examination. Temporary glycosuria has been observed by some writers.

In the mild cases, without much or any disturbance of consciousness the immediate effect of concussion will be pain in the head, some dizziness, sometimes vomiting and a general sense of feeling weak. The pulse presents the same peculiarity as in the severe cases. Insomnia, restlessness, photophobia, loss of appetite, constipation are present. The mentality may suffer, but this will be considered later.

Prognosis.—The outlook is variable. Even in the severest forms recovery may follow. On the other hand I have seen mild cases which lasted months and years and recovery remained incomplete. In the majority of cases, especially when there is no history of syphilis or alcoholism, when there are no complications, such as fracture of the skull, also when the treatment is instituted at the earliest possible moment, recovery may be complete. An incomplete recovery consists of a continuous asthenic condition, loss of weight, pains in the head and change of disposition. Irritability, dfficulty of adapting oneself to surroundings may remain for years.

In a certain group of cases mental phenomena of a graver nature may develop. Among the latter he following disturbances have been observed: (1) Transitory or permanent defects of intelligence especially of memory, disturbance of ability to retain impressions, retrograde amnesia. (2) Delirium ending either in recovery or associated with profound mental alterations ending in Dementia. (3) Hallucinosis. I observed this manifestation in five patients. There were no delusions. The hallucinations, mostly visual, lasted for months. The patients finally recovered. (4) Korsakoff's psychosis has been observed in individuals free from alcoholism by a number of writers. (5) Finally dementia may be the ultimate outcome of concussion of brain.

In children, when concussion occurs at an early age, arrest of mental development may occur. I have had the opportunity to study several children, whom I happened to know before the injuries occurred, and a decided change in their mentality and intellectual power took place. Their progress in mental development was unquestionably arrested.

Treatment.—The most essential principle of treatment is **absolute**

rest in bed which must be instituted as early as possible after the injury. This should be carried out not only in the severe cases, but also in the cases of the mildest nature. The various manifestations should be treated symptomatically. Sedatives, purgatives, light diet and particularly avoidance of stimulation, of excitement, of noise—is all that is necessary. Confinement to bed must be as long as possible. It is reiterated here, that neglect of recognizing the importance of earliest treatment may render the patients permanent invalids.

CHAPTER XVII

DISEASES OF THE CEREBELLUM

The cerebellum contains two important portions: cortex and central gray nuclei. In the cortex we distinguish: hemispheres and vermis. These two parts are physiologically independent of each other. The hemispheres are in connection with the cortex of the brain and basal

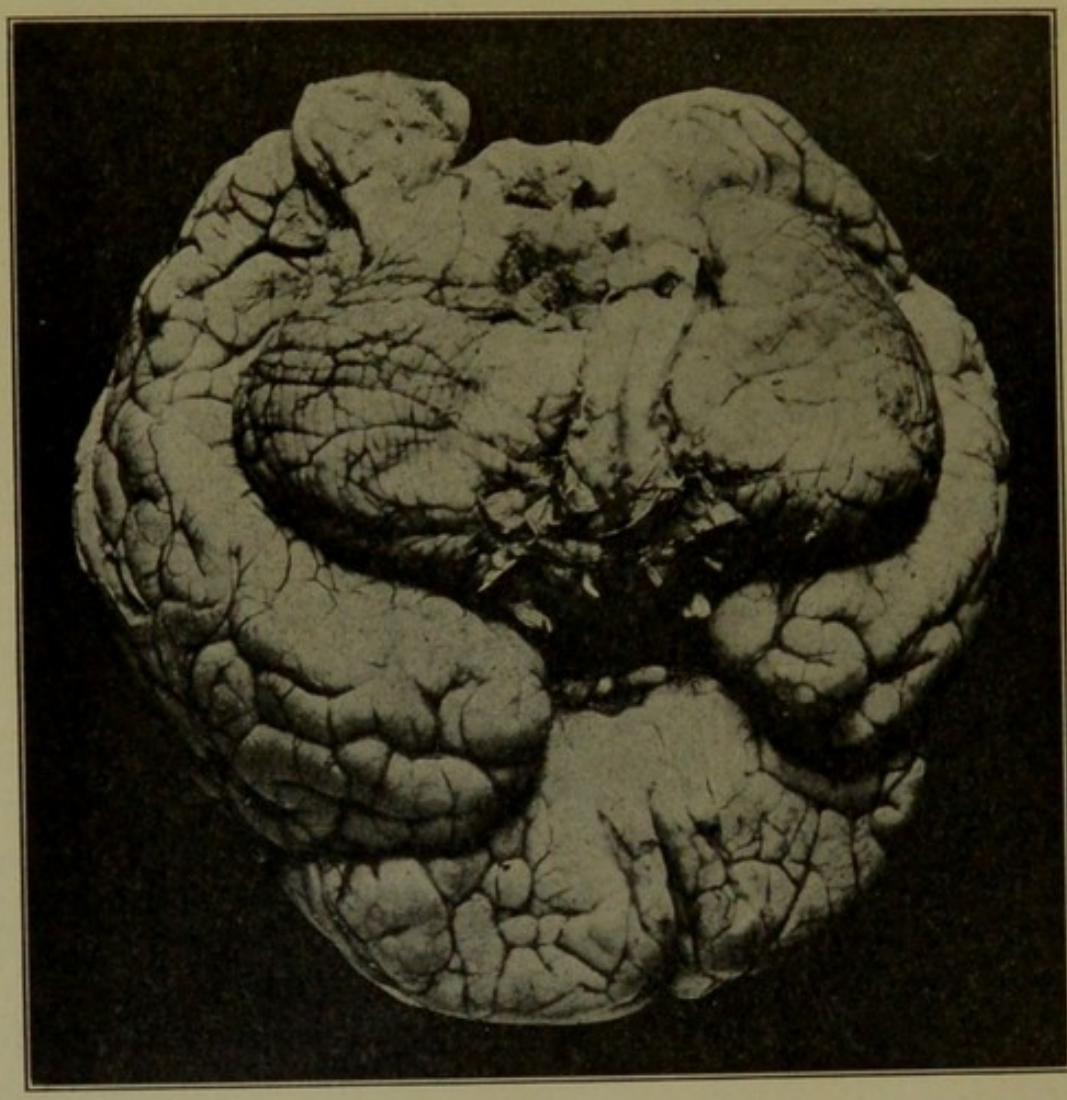


Fig. 78.—Cerebellar Tumor Involving Left Hemisphere and Vermis. (*Original.*)

ganglia. The vermis—with pons, medulla and spinal cord. The hemispheres are concerned in voluntary movements, the vermis is concerned in coördination through the vestibular nerve with the nucleus of which it is in intimate connection.

The function of the cerebellum is to control the equilibrium and regulate movements. When the cerebellum is removed, motion and equilibrium are not entirely abolished because of supplementary action of the brain and of the labyrinth.

Unlike the cerebrum, the cerebellum has its influence on the muscles of the same side of the body.

Lesions of the cerebellum may have a slow and progressive course, such as tumors, and an acute onset, such as hemorrhage or softening.

Tumors.—Among the tumors of the nervous system those of the cerebellum are the most frequent, and as to their nature, the **tubercular** form is most common. All the varieties of tumors met in the brain may also affect the cerebellum. Tubercle of the cerebellum is rarely solitary and never primary; it is always accompanied by pulmonary lesions.

. The point of departure of cerebellar tumors may be in the peduncles or in the meninges besides the cerebellar tissue itself. It may be located in one of the hemispheres or, as it frequently happens, in the vermis. Cerebellar tumors may be also located in the angle between the pons and cerebellum. They may arise then, either in the cerebellum itself or in the cranial nerves. As to the effect of a tumor on the neighboring tissue, see the Pathology of Tumors of the Brain.

Symptoms.—The **general** symptoms common to tumors of the brain (see this chapter) are met with here also. However they present some particularities deserving special mention. **Headache** is quite frequently occipital and very tenacious; it radiates to the neck. Percussion over the area corresponding to the seat of the tumor provokes pain. **Vomiting** occurs quite early. **Vertigo** is also a precocious sign and persists through the entire course of the disease. It is present even when the patient is at rest: sitting or lying down. Stewart and Holmes observed that when vertigo is present, objects and patient himself rotate from the side of the lesion to the opposite side.

Among the symptoms **especially characteristic** of cerebellar tumors, **titubation** occupies the first place. It resembles to a great extent the station and gait of an intoxicated person and consists of oscillations of the body to the right and left alternately, with a tendency to fall backward or forward. The movements are therefore zigzag-like. This condition is observed in tumors of the middle lobe. When the patient walks, he widely separates his legs. Standing is difficult because of continuous oscillations. Standing on the leg corresponding to the lesion is less steady than on the sound side. When the patient shows a tendency to walk toward one side and holds the trunk and head inclined to the same side, the lesion is on this side. This **cerebellar ataxia** may affect not

only the lower extremities (which is usually the case), but also the upper. When the patient attempts to point to an object, irregular incoördinate movements appear. Sometimes they are tremulous, but not like in insular sclerosis, because they do not increase at the end of the act; they rather disappear.

Another disturbance of association of movements observed frequently in cerebellar diseases has been described by Babinski (1899) under the name of **Cerebellar Asynergy.** When a patient thus affected attempts to walk, the trunk does not follow the legs in their forward movements (Fig. 65). When lying on his back the patient attempts to sit up, he

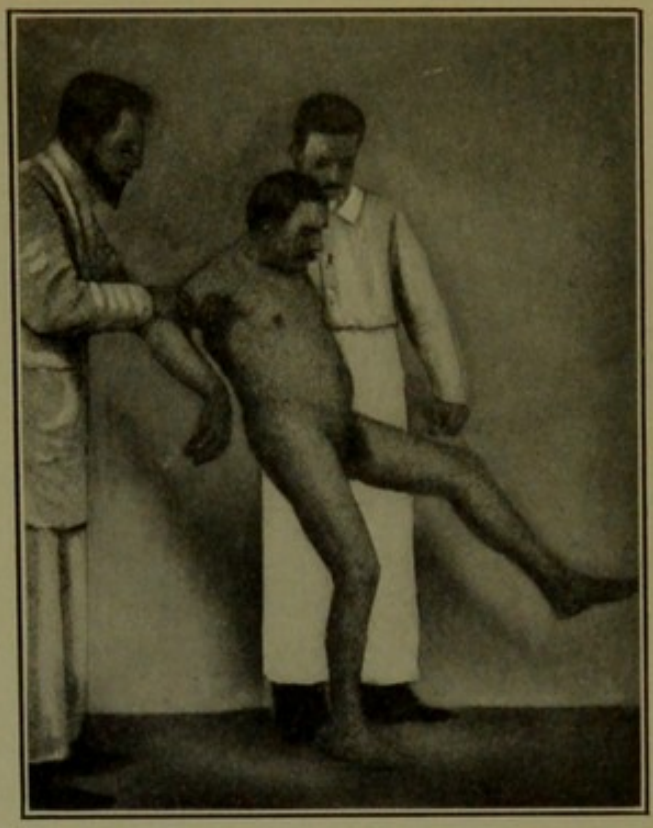


FIG. 79.—CEREBELLAR ASYNERGIA. (*Bouchard and Brissaud.*)

raises his legs and flexes the thighs on the pelvis. When seated on a chair the patient wishes to raise his leg, he flexes first the thigh on the pelvis and then only he elevates his leg; the latter movement is produced very abruptly; in order to put the foot back on the ground the leg first flexes over the thigh, then abruptly the latter becomes extended and the foot reaches the floor.

The disturbed associated movements just described may be confined to one side and they are then spoken of as **Hemiasynergy.** The same author also called attention to the following cerebellar phenomena:

(1) **Difficulty of Regulating Movements.**—When, for example, the patient attempts to walk, he will raise his foot higher than normally

which means excessive flexion of the thigh over the pelvis. When he wishes to put his finger on the nose, the former will overstep the point of destination and reach the cheek.

(2) **Adiadochokinesia.**—It consists of inability to execute rapid pronation and supination of the hand. If it is observed on one side it usually corresponds to the seat of the lesion.

(3) **Cerebellar Catalepsy.**—It consists of as pecial ability to maintain a fixed position longer than normally. When a patient lying on his back with his thighs flexed over the pelvis, and the legs over the thighs, raises his limbs, the latter will at first oscillate, but in a few moments the trunk and the limbs will become fixed. This fixation may remain several minutes and is not followed by fatigue.

Luciani called attention to **hypotonia** and **asthenia** of the limbs on the side of the lesion as characteristic of cerebellar tumors. General weakness (asthenia) is characteristic of crebellar diseases. It is probably due to inability to balance, to the loss of equilibrium of various portions of the body.

The **position of the head** deserves special mention. In 1908 (*J. Amer. Med. Ass'n*) I called attention to a head phenomenon which I found of localizing value in a series of cases some of which came to autopsy. It consists of an increase of vertigo or headache or of both when the head is turned to the side of the seat of the cerebellar lesion.

The **tendon reflexes** are usually altered. All varieties may be present, from diminution and loss to exaggeration. A unilateral change does not always correspond to the seat of the lesion. **Sensations** are usually not involved.

Ocular Symptoms.—They are present in the majority of cases and consist of a more or less pronounced loss of vision. The eye-grounds show hyperemia, œdema, **choked disc, optic atrophy.** The pupils are frequently unequal. **Nystagmus** is almost a constant symptom. It is more marked in lateral than in vertical movements of the eyes. It is usually slow and of wide range toward the side of the lesion but of smaller range and more rapid toward the normal side. Palsies of cranial nerves are observed in tumors of cerebello-pontine angle. Skew deviation of the eyes is sometimes observed. It consists of one eye looking downward and inward, the other outward and upward.

Articulation of speech is not infrequently affected in cerebellar tumors. It is slow and explosive, but not quite as explosive as in insular sclerosis.

Cerebellar tumor frequently causes an increase of cerebro-spinal fluid (**hydrocephalus**). This is probably the cause of the mental disturb-

ances, apathy and hebetude observed during the last period of the disease.

The symptoms of compression of the neighboring tissue are various. Hemiplegia, hemiparesis or crossed hemiplegia may be observed. The hemiplegia is not spastic, the face is not involved and hypotonia of the muscles is present on the affected side. The toe phenomenon and ankle-clonus may be absent. The cranial nerves, particularly the third and sixth, may become involved.

Course. Duration. Prognosis.—Headache and vertigo are usually the first symptoms. They may persist for several months before the titubation makes its appearance. Rarely amaurosis is wanting. Asthenia also appears early. Gradually convulsions develop. The duration of the disease is indefinite. It may last from several months to several years, although usually it runs a rapid course. The termination is fatal in cases which are not operated upon, although a few cases of recovery of tuberculoma of the cerebellum have been reported. Rapid termination occurs in cases of compression of the medulla.

Diagnosis.—In a certain number of cases the diagnosis of a cerebellar tumor presents great difficulties. This is particularly seen in the initial stage, when only general symptoms are present, as it is well known the latter are common also to cerebral growths. When in addition to the latter bulbar symptoms appear, the presumption is in favor of a cerebellar involvement. In cases in which in addition to the general signs even one of the special symptoms should make its appearance, the diagnosis will readily be made. When the cerebellar involvement is decided upon, the question of its exact seat must be solved, as operative procedures depend upon it. When the tumor is unilateral, the characteristic symptoms will be present on the same side. A tumor of the median lobe will give bilateral symptoms. Particular stress must be laid upon hemiasynergy, upon the unilateral occipital headache with tenderness on percussion of the occiput, upon the position of the head, upon nystagmus and other ocular symptoms, upon the state of the reflexes on one or the other side, upon the state of the reflexes on one or the other side, upon involvement of one or several cranial nerves on one side. All these informations are of great utility, as the symptoms usually correspond to the seat of the tumor.

As to other diseases with which a cerebellar affection may be confounded, **tabes** particularly must be mentioned. Ataxia and loss of reflexes may be present in both, but the zigzag movements of a cerebellar is not present in a tabetic; the raising of the feet high and dropping them with force on the ground are characteristic only of the tabetic. Moreover other symptoms will help to decide the diagnosis.

In **Ménière's disease** there are: vomiting, vertigo and even titubation, but absence of headache and of ocular symptoms will render the diagnosis comparatively easy.

Treatment.—In cases in which syphilis is suspected, mercurials and iodides should be tried. Relief and removal of the distressing symptoms for a long time may follow from specific treatment in cases of cerebellar gummata. In two of my cases the ataxia, vomiting and headache disappeared completely under iodides. Even in tubercular tumors of the cerebellum iodides will be of great value, as I could ascertain it in one of my cases, in which all the symptoms except the blindness disappeared. In the largest majority of cases surgical treatment alone is applicable.

In the chapter on Tumors of the Brain operative procedures are fully discussed with especial reference to **decompression;** the latter will at least decrease the intracranial pressure and thus relieve pain, making therefore life more tolerable. But where particularly lies the benefit of operation is the prevention of blindness and restoration of sight. In one of my recent cases the patient was relieved from his headache, choked disc disappeared, sight was restored and this condition lasted almost two years when he died from pneumonia. A large part of the skull of the occipital region should be removed. In doubtful cases as to the localization, it has been advised to trephine first the right temporal region, thus relieving general intracranial pressure. If the cerebellar symptoms remain unaltered, another operation over the occipital bone should be attempted ater (subtentorial operation).

TUMORS OF PONTO-CEREBELLAR ANGLE

These tumors originate either in the meninges or more frequently in the cranial nerves of the base of the brain, particularly in the eighth nerve, but sometimes also in the fifth, seventh, ninth, tenth, and eleventh nerves. They are usually solid, resistant and small. They contain fibrous elements (fibroma, fibro-sarcoma, neuro-fibroma). In one of my cases there was a cyst (*Old Dom. J. of Med. and Surgery*, 1910). They are benign tumors and their extirpation if successful, usually leads to recovery. They occupy the space between the lateral border of the pons and the cerebellar hemisphere. This space corresponds to the posterior surface of the petrous bone at the level of the auditory meatus, into which enter the seventh and eighth nerves.

Symptoms.—General symptoms of intracranial pressure are present, viz. **Headache, vomiting, stasis of papillæ.** The latter is an early disturbance. It is frequently followed by optic atrophy. Headache is

frequently localized in the occipital region. Pressure of the cerebellum produces the characteristic **cerebellar symptom group** (see above). Vertigo deserves special mention. Steward and Holmes assert that objects seem to move from the side of the lesion to the normal side, but the subjective sense of rotation of the patient himself is in the opposite direction. In tumors of the cerebellum the reverse condition of the latter is observed. Symptoms referable to **compression of the nerve trunks** at the base are the most characteristic feature. The **auditory** nerve particularly suffers and this is manifested by noises in the ear and soon by deafness on the affected side, while the ear itself is intact. Next in frequency are the **facial and trigeminal** nerves. The involvement of the former is manifested by a hemispasm and more frequently by paresis of the face. Hyperæsthesia, neuralgia or else anæsthesia, also corneal anæsthesia with loss of the corneal reflex are signs of involvement of the **fifth** nerve. More rarely the sixth, ninth, and tenth nerves are affected. Corneal areflexia, hypoacousia and paresis of associated movements of the eyes are according to Oppenheim characteristic of ponto-cerebellar tumors.

Operation is the only **treatment.** Among various methods that of Krause is the most accepted. He operates on the occipital region and exposes the cerebellar hemispheres. The vicinity of the cerebellum and of the medulla renders the operation very serious. The mortality indeed is very great. In so-called successful cases, the most distressing symptoms disappear, but the auditory manifestations especially are very little, if any, influenced by the operation. Vision may be only improved, except when there is already optic atrophy. In spite of the gravity operations are justifiable. Some very successful cases have been reported. Recurrences of the tumors are not known.

ABSCESS OF THE CEREBELLUM

Etiology.—Apart from metastatic abscesses, the most frequent cause is suppuration of the temporal bone. The latter is a common occurrence in chronic otitis media. Occasionally an acute otitis media may be the cause of a cerebellar abscess. The otitis itself may develop in the course of grippe, of pharnygitis, of exanthematous infectious diseases. Speaking generally, any cause leading to caries of the temporal bone, and its petrous portion particularly, will produce a cerebellar abscess.

Pathology.—When the abscess is seated within the cerebellar substance, the surrounding nervous tissue may be reduced to a minimum. As the abscess is usually secondary to caries of the neighboring bone (particularly temporal), the meninges will be found diseased. Phlebitis

of the sinuses, especially of the lateral, is a frequent finding and thrombosis is therefore equally frequent. The surrounding cerebellar tissue undergoes softening and destruction. The pus is thick, greenish and fetid. It contains the usual microbes of suppuration, viz. streptococcus, staphylococcus or pneumococcus.

Symptoms.—In **acute** stage the otorrhea and the deafness, which have been present for a more or less long period, become suddenly complicated by grave cerebral symptoms: coma with general symptoms of infection. The characteristic cerebellar symptoms cannot be revealed.

In the **chronic** stage the special cerebellar symptoms, described in "Tumors of the Cerebellum," are easily observed. In order to avoid repetition the reader is referred to that chapter. A few special manifestations are worth mentioning. Rigidity of the neck and retraction of the head, slow nystagmus, pronounced vertigo upon the least movement of the head, frequent occurrence of optic neuritis—are all not infrequently observed in abscess of the lateral lobe of the cerebellum.

An abscess may compress the neighboring portions of the nervous system. A crossed paralysis, viz. involvement of any of the cranial nerves on one side and of the extremities on the opposite side, is indicative of pressure at the base of the brain.

An abscess may remain silent for a long time, when it is not seated in the middle lobe and strictly limited. Most of the time it spreads to the neighboring tissue and produces great damage in the nervous tissue.

Prognosis.—It is identical with that of cerebellar tumors in the chronic stage, but it is very serious in the acute stage.

Diagnosis.—In the chronic stage it will be extremely difficult to differentiate the disease from tumor. Otorrhea or a diseased condition of the mastoid are suggestive rather of abscess than of tumor. In the acute stage the diagnosis presents sometimes insurmountable difficulties, as apart from the ear symptoms which may be overlooked, there is very little to make a diagnosis. The general condition is that of meningitis.

Treatment.—Operative intervention is the only treatment and it should be done as early as possible. A large opening should be made in the skull and the pus evacuated, if the abscess is on the surface of the cerebellum; a puncture should be made in the cerebellum, if the abscess is within the latter.

CEREBELLAR HEREDO-ATAXIA

Under this name P. Marie described a form of cerebellar incoördination **hereditary** in character and occurring in several members of the same family.

Pathology.—The characteristic condition of this affection is **atrophy of the cerebellum,** which is due to arrested development. The autopsy records show that in some cases in addition to the cerebellar atrophy there was also atrophy of the medulla and of the spinal cord. In Marie's first case there was also sclerosis of Goll's, Gower's and direct cerebellar tracts, finally marked diminution of the middle cerebellar peduncles. In some cases the changes were found to affect only the gray matter of the cerebellum. The cerebellum is rarely alone affected.

Symptoms.—The striking feature is the disturbance of coördination. The lower extremities are affected long before the upper. The usual cerebellar titubation with asynergia, asthenia, scoliosis, ocular disorders, exaggerated reflexes and integrity of sensory functions—otherwise speaking, all the symptoms found in cerebellar diseases (see above) are also found in Marie's disease.

The **characteristic** features lie in the **evolution** of the symptoms. The onset is gradual. In some cases neurasthenic symptoms precede the disturbance of equilibrium. The patient complains of headache, pain in the back and general fatigue. The first signs appear early in life, generally between fifteen and twenty-five years of age. Disturbance of equilibrium, which is soon followed by a typical cerebellar ataxia, are the initial symptoms. They are soon followed by a disturbance of **speech** and **voice:** the speech is irregular, each word is accentuated and precipitated; the voice is monotonous and guttural. When the patient speaks, there is noticeable an exaggerated **contraction** of the muscles of the face. In the course of the malady incoördination of the **upper extremities** usually develops. Their movements become uncertain; a fine intention tremor is quite frequently present, so that delicate acts, such as writing, threading a needle, etc., are almost impossible.

The **psychic** faculties are usually altered. Impairment of intelligence, of memory, irritability and indifference are the main disturbances observed.

The disease is invariably progressive, although it may remain stationary for a long time. In its lastp eriod there is absolute physical impotence: the patient is confined to bed and usually dies from some intercurrent disease.

Diagnosis.—The history, the onset, the gradual development of the symptoms, the family character of the disease will enable one to differentiate it from tumors, hemorrhages or abscesses of the cerebellum; also from cerebral diplegia. Some difficulty is found sometimes in distinguishing it from **Friedreich's ataxia.** In favor of heredo-cerebellar ataxia will be the family character, the age at which it occurs, the slow development,

cerebellar gait, asynergia, incoördination of the upper extremities, ataxia in writing, nystagmus, the peculiar facial mimicry, intention tremor, finally increased knee-jerks with ankle-clonus. The condition of the knee-jerks is particularly important, as their abolition is characteristic of Friedreich's ataxia. The latter affection may present sometimes, especially at the beginning, normal reflexes; on the other hand, in Marie's disease, when the lesion reaches the cord, the reflexes may be abolished. In such cases the other symptoms will aid in making a diagnosis.

Etiology.—A neurotic family history is not infrequently obtained. Alcoholism, tuberculosis in parents are reported by some observers. Consanguinity and syphilis have been noted in a limited number of cases. Some writers mention also infectious diseases and traumatism. The female sex is apparently more frequently affected than the male. Members of the same family become frequently affected at the same age.

Treatment.—Antisyphilitic treatment may be tried, but in view of the character of the lesion (see Pathology) no results can be expected from medications.

HEMORRHAGE AND SOFTENING OF THE CEREBELLUM

Hemorrhages are quite rare. In 100 cases of cerebellar diseases described by Krauss there was one case of hemorrhage. In 187 cases of apoplexy Starr found only four cases in the cerebellum. In the majority of cases they have been found in the hemispheres. Minute hemorrhages are less frequently observed than large ones. When a hemorrhage occurs, the blood is apt easily to break through the cerebellum and invade the fourth ventricle. The superior and inferior cerebellar peduncles are rarely the seat of hemorrhages, but the middle peduncle may be invaded by a hemorrhage originating in the pons.

Softening of the cerebellum is extremely rare for the following anatomical reasons: (1) the angle formed by the basilar and cerebellar arteries interferes with the formation of embolism in the latter; (2) thrombosis is very rare in the cerebellum.

Symptoms (of Hemorrhage).—The prodromal symptoms and the onset itself are identical to those of hemorrhage in the cerebrum. Pain in the occipital region, persistent vomiting are very early symptoms. If the patient does not die immediately after the loss of consciousness, he remains comatose. When consciousness is regained, the striking differential sign will be the absence of hemiplegia, which on the contrary is so constant in apoplexy of cerebral origin. However, Mann (*Monatschr. für Psychiatrie u. Neurologie*, 1902) described cerebellar hemiplegia on the side of the lesion which

presents the following characteristics: it affects all the muscles of the both limbs, while in cerebral hemiplegia only certain muscular groups are affected, such as flexors of the leg and of the foot, extensors of the hand; moreover, there is no contracture; the reflexes are exaggerated; there is no rigidity, no ankle-clonus, no toe phenomenon.

After the patient recovered from the immediate effects of the attack, there is noticed an extreme general weakness (not a paralysis), so that the patient cannot even remain seated. Soon the other symptoms of cerebellar diseases make their appearance, viz. cerebellar ataxia, etc. (see Cerebellar Tumors). If paralysis develops, it is almost never at the beginning, but later, and it is due to pressure upon the pons and medulla.

In **softening** the onset will not be sudden, but progressive. Later the symptoms will be identical with those of hemorrhage.

In view of a considerable anastomosis in the cerebellar *arterial* supply, softening is usually confined to a small area.

CHAPTER XVIII

DISEASES OF THE MEDULLA, PONS AND THE FOURTH VENTRICLE

THESE three portions of the nervous system contain extremely important elements. The sensory and motor pathways, the nuclei of ten of the cranial nerves, centers for some of the vegetative functions—are all located here. In diseases therefore of this area the symptomatology is complex. There are, however, a few groups of symptoms which constitute special forms of diseases characteristic of involvement of this area of the nervous system.

A. ACUTE SUPERIOR POLIOENCEPHALITIS (HEMORRHAGIC)

Pathology.—The lesion consists of an inflammation with hemorrhages in the gray matter of the aqueduct of Sylvius. The latter is therefore found in a state of softening. Microscopically are seen dilated blood vessels, the perivascular spaces are filled with blood, a leucocytic infiltration is marked. Sometimes this pathological process extends forward into the floor of the third ventricle and backward into the fourth ventricle. The nucleus of the third nerve, also the fibers emerging from it, are therefore the main seat of the lesion.

Etiology.—The most frequent cause is **chronic alcoholism.** Other intoxications, as carbonic acid, sulphuric acid, infectious diseases, alimentary intoxications (fish, meat, etc.) are sometimes followed by superior polioencephalitis.

Symptoms.—The onset is acute. In the course of chronic alcoholism or an infectious disease the patient is suddenly taken with **headache** and **vertigo.** Soon somnolence and delirium make their appearance. Rapidly **palsies of the eye muscles** develop.

Most of the time both eyes are involved, but the degree of palsy is unequal. Sometimes an associated paralysis is observed; either both external recti or both internal recti are affected. Ptosis is not frequent. Wernicke (whose name is attached to the disease) claims that the sphincter of the iris is never involved. I have records of two personal cases with palsy of this sphincter.

Optic neuritis, nystagmus may occur.

In addition to the ocular disturbances, which are the characteristic feature of the disease, other symptoms are not infrequently observed. Ataxia, unilateral paralysis of the extremities, disturbance of speech, exaggeration or abolition of the tendon reflexes, disturbances of deglutition and of mastication may be met with.

As the inflammation may become diffuse and be ascending or descending or both, cerebral as well as spinal symptoms may be associated. Polioencephalitis, superior and inferior (see further), and poliomyelitis are not infrequently combined (**polioencephalomyelitis**). The extension of the inflammatory process from the gray to the white matter of the pons and medulla (which is not rare) will explain the above-mentioned additional clinical phenomena.

Course, Duration and Prognosis.—The evolution of the symptoms is rapid. In the majority of cases the duration is from eight to fourteen days, although in one case it lasted sixteen weeks. Recovery was reported in a few cases. In one of my cases the palsy of the third and fourth nerves has been in existence two and one-half years. The patient is still living. The onset in this particular case was apoplectiform in nature with loss of consciousness. The palsy affected all the muscles supplied by the third and fourth nerves. In another case, a child of ten months, there was also loss of consciousness with involvement of the third, fourth and sixth nerves simultaneously. The child is now three years old, enjoying good health and the nerve palsy has greatly improved. In both cases the paralysis is bilateral, but unequally distributed to various muscles of the eye globes. In the case of the child the condition was preceded by an attack of cough for a period of four or five days, evidently of infectious nature. In the first case the woman was addicted to the use of alcohol. She was thirty-five years of age. Both patients present a nystagmus on lateral movements. The tendon reflexes were exaggerated in the woman.

Treatment.—The general symptoms may be relieved by the usual medications, as for example the headache by opium, morphia or coal-tar products. Bleeding, purgatives and diaphoretics may be tried. Little reliance should be placed upon drugs. Iodides may do some good.

B. CHRONIC SUPERIOR POLIOENCEPHALITIS (PROGRESSIVE NUCLEAR OPHTHALMOPLEGIA)

The disease is characterized by a slow but progressive paralysis of the muscles of the eye.

Pathology.—Atrophy of the motor nuclei of the eye is the character-

istic lesion. Chromatolysis with formation of vacuoles, also marked pigmentation of the cells of the nuclei, are the first stage of the affection. Later on the cells disappear and the nuclei are in a state of atrophy.

In some cases the nuclei of all the motor nerves of the eye are affected, in others only the nucleus of the third nerve is diseased. Secondarily the roots emanating from the nuclei, the nerves themselves and the muscles innervated by the latter undergo degeneration.

Etiology.—Infections, intoxications, syphilis may be the causes of the malady. It is occasionally observed in the course of organic diseases of the nervous system: **disseminated sclerosis, tabes, paresis.**

Symptoms.—The onset is insidious. While there is no constancy in the order of involvement of individual ocular muscles, nevertheless **ptosis** and **diplopia** are the first symptoms in the majority of cases. As at the beginning, the palsy is not complete, the movements of the eye globe in certain directions are yet possible, but only after an effort. The facies of the patient is quite characteristic. As the upper eyelids are lowered, he holds his head thrown backward in order to be able to see objects; the forehead is wrinkled as the frontal muscles are trying to raise the palsied eyelids. The movements of the eye depend upon the muscles involved. In an advanced stage of the disease the eyes become immobile, and in order to see on the right or on the left, the head must be turned. External ophthalmoplegia is frequently associated with internal ophthalmoplegia. In such cases the pupil ceases to react to light or accommodation.

Course. Duration. Prognosis.—When the lesion remains confined to the ocular nuclei, the disease may last an indefinite number of years. The disease may begin unilaterally and remain confined to the same side. When it has a descending course and becomes complicated by an inferior polioencephalitis (see further, death may ensue in a very short time. At all events a nuclear ophthalmoplegia is an incurable and grave affection.

Diagnosis.—From the **acute** form it will be distinguished by the absence of general symptoms (see above), by the slow course. Opthalmoplegia may be due to a lesion at the **base** of the brain, but in the latter case other cranial nerves are usually involved. In the course of infectious diseases or intoxications external ophthalmoplegia may be observed, but usually there are also palsies of the limbs, of pharynx, larynx, of face. These are cases of **multiple neuritis** and bear usually a favorable prognosis: improvement is almost invariable.

In **Myasthenia gravis** (see this chapter) there is also a weakness of the masticator muscles and the general asthenia is very striking.

Paralysis of the muscles of the eye may be the result of a disease of the orbit. In such cases the diagnosis is easy.

Treatment.—There is practically no medication to rely upon. Mercury and iodides should be tried even in cases without a history of syphilis. Electricity may also be applied.

C. ACUTE INFERIOR POLIOENCEPHALITIS (ACUTE BULBAR PALSY)

Pathology.—The lesion is identical to that of acute superior polioencephalitis (see above). The difference lies only in the seat. Hemorrhages and softening are the immediate causes. Here all other nuclei, except those of the third, fourth and sixth cranial nerves, are involved of only some of them. A not infrequent occurrence is the association of both forms of polioencephalitis.

Etiology.—Infectious diseases and alimentary intoxications are reported to be the causes of the disease, which is, however, quite rare.

Symptoms.—The onset is sudden or rapid and characterized by general symptoms, such as headache, chills, fever and pain in the neck. Loss of consciousness is usually present in hemorrhages, but not in softening. Soon the patient develops difficulty in swallowing and articulation; otherwise speaking, the picture is that of labio-glosso-laryngeal paralysis (see next chapter). The tongue, lips, palate are paralyzed. The food is regurgitated through the nostrils and the patient is threatened with suffocation. The pulse and respiration are irregular. Trismus is according to Joffroy a characteristic symptom. A comatose state sets in rapidly and death is the usual termination.

Termination. Prognosis.—The disease usually lasts from two to six days. Some exceptional cases of recovery have been reported.

Treatment.—Revulsion and counterirritation on the neck, also purgation, are the only means for the disease, which is almost invariably fatal.

D. CHRONIC INFERIOR POLIOENCEPHALITIS (CHRONIC BULBAR PALSY) (LABIO-GLOSSO-LARYNGEAL PARALYSIS)

The disease is characterized by a paralysis of the muscles of the lips, tongue, pharnyx and larynx.

Pathology.—The lesion consists of a primary and progressive degeneration of the nuclei of origin of the cranial nerves, situated in the lower half of the medulla, viz. those of the seventh, ninth, tenth, eleventh and twelfth pairs. The alterations are most marked in the nucleus of the hypoglossus

(twelfth). They consist of diminution or disappearance of the chromatophilic substance of the cells, of appearance of pigment within the cells, of a displacement or disappearance of the nucleus or nucleolus, finally of atrophy of all the prolongations of the cells. The nerve-roots emanating from the nuclei therefore appear very thin. The degree of involvement is not equal in all the cells of the same nucleus and in various nuclei.

As to the participation of the white matter of the medulla in the pathologic process, all the writers are not agreed. Some claim (Raymond, Leyden, Dejerine) that the labio-glosso-laryngeal paralysis of Duchenne is not an autonomous disease, but is almost always followed by an involvement of the pyramidal bundles to constitute amyotrophic lateral sclerosis. In the ascending course of the latter disease bulbar symptoms almost invariably appear, but there is also a form in which the disease begins with labio-glosso-laryngeal palsy (see Amyotrophic Lateral Sclerosis).

The muscles in which the affected cranial nerves are distributed also undergo atrophy, viz. those of the tongue, lips, pharynx and larynx.

Etiology.—Syphilis, fatigue, Bright's disease have been reported in the histories of some patients, but there are no positive data as to the true etiological factors of this affection.

Bulbar paralysis may occur in the course of amyotrophic lateral sclerosis, tabes, multiple sclerosis and syringomyelia.

Symptoms.—The onset is insidious and slow. There is usually a brief prodromal stage during which the patient complains of pain in the neck and a numbness of the pharynx. Gradually the lips, larynx and the tongue become paralyzed. The latter particularly is affected **first** in the majority of cases. At the beginning there is only a weakness in the movements of the tongue, but it keeps on increasing until complete immobility is established. The letters that require the coöperation of the **tongue** aer imperfectly pronounced. The speech is therefore impaired (**dysarthria**). The tongue is flat, diminished in size and presents fine fibrillary contractions, also reactions of degeneration. In an advanced stage the atrophy of the tongue is very marked; it then presents a depression and on palpation it is very soft. The speech is impossible (**anarthria**). **Mastication** and **deglutition** are difficult.

The **lips** follow the tongue. It is the orbicularis muscle that is first affected, but soon other muscles suffer. The atrophy and paralysis of the lips interfere with the pronunciation of labial letters, with the act of blowing, whistling and laughing. Reactions of degeneration appear early. The lips being immobile, the mouth remains open and the saliva is continuously dribbling. The facies is quite characteristic at this period.

While the expression of the eyes shows total integrity of intelligence, the condition of the mouth gives the impression of a stupid and crying face. This contrast is typical of bulbar palsy.

Paralysis of the **palate** is manifested by a change in the tone of the voice; the latter is nasal. The food is then regurgitated through the nose.

When the **pharynx** is paralyzed, the food is likely to fall into the larynx, as the deglutition is very difficult. The patient is threatened with suffocation. Artificial feeding is then necessary. The paralysis of the **larynx** increases the danger: the glottis being open, the food easily falls into the larynx.

The laryngeal condition, consisting of paralysis of the vocal cords and of all the muscles innervated by the recurrent nerve, produces disturbances not only of the voice, but also of **phonation.** The patient is unable to emit even a sound (**aphonia**).

The gradual but progressive involvement of the medulla leads to cardiac and pulmonary disorders. The **pulse** is small, irregular and feeble; attacks of **syncope** are quite frequent. The least effort brings on **dyspnea.** The patient is unable to expectorate, to breathe properly: mucus accumulates in the bronchial tubes. An ordinary bronchitis becomes thus very serious. Broncho-pneumonia develops easily.

Among other symptoms the condition of the reflexes should be mentioned. Those of the extremities are very frequently exaggerated. This fact shows the close relation of bulbar palsy to amyotrophic lateral sclerosis.

Course. Duration. Prognosis.—The disease is essentially progressive. Death may occur from three causes: **inanition** (because of the inability of swallowing), **syncope** or **broncho-pneumonia.** The latter may be of infectious or gangrenous nature when food enters the larynx. The usual duration of the disease is from a few months to a couple of years.

Diagnosis.—The essentially chronic and progresssive course after an insidious and slow onset, the successive involvement of the tongue, lips, palate and masticatory muscles, atrophy of the muscles with fibrillary contractions and reaction of degeneration are sufficiently characteristic symptoms for the diagnosis of bulbar palsy.

Palsy of the **palate** following diphtheria is accompanied by difficulty of swallowing and a nasal intonation of the voice. These cases will be recognized by absence of paralysis of the tongue and lips.

Hemorrhage and **softening** of the medulla are sudden in onset and the symptoms are pronounced at the beginning.

In **Pseudo-bulbar** palsy there is always a history of one or two attacks of apoplexy. The syndrome of labio-glosso-laryngeal paralysis is usually established after the second attack, but it has not the chronic character of the pure bulbar palsy. Besides, there is no muscular atrophy or fibrillary twitching. The spasmodic laughing and crying, impairment of intelligence, hemiplegic condition of the extremities are all typical of the pseudo-bulbar palsy (see this chapter).

Asthenic bulbar paralysis (myasthenia gravis) is recognized by the predominance of the paralysis in the muscles of mastication and in the levator palpebræ (ptosis). The extreme exhaustion manifested in the muscles upon the least exertion, the absence of atrophy and the special electrical reactions are characteristic of myasthenia gravis (see this chapter).

Treatment.—Counter-irritants, cautery on the back of the neck, also galvanism at the same level or direct electrization of the affected muscles; antisyphilitic remedies, belladonna or atropin for diminishing the salivation, general hygienic measures are all the therapeutic means we have at our command for a palliative treatment. Unfortunately the disease is inevitably fatal. When difficulty of deglutition supervenes, artificial feeding is necessary. In case of imminent asphyxia, tracheotomy should be performed.

E. PSEUDO-BULBAR PALSY

This name is given to the syndrome of glosso-labio-laryngeal paralysis described in the preceding chapter, which is not due to a lesion in the medulla but in the brain.

Pathology.—In order to understand the pathological mechanism of this symptom-group it is necessary to recall the following anatomical facts.

The muscles of the tongue, face, lips, pharynx and larynx are innervated by two systems of neurones. One, lower or peripheral, connects the muscles with the nuclei of the medulla. The other connects the bulbar nuclei with the cortex, and especially with the Rolandic operculum (see Anatomy); the connecting fibers lie in the geniculate bundle of the internal capsule. A lesion of the first neurones will give place to the genuine bulbar palsy (labio-glosso-laryngeal) of the preceding chapter. A lesion affecting only the second (upper or cortico-bulbar) system of neurones will result in a loss of transmission of stimulation from the brain to the lower neurone, and we will have a pseudo-bulbar palsy. In the latter case the lesion must be **bilateral,** because each hemisphere innervates the

muscles of both sides. A unilateral lesion of the cortico-bulbar pathway will therefore fail to produce the complete picture of labio-glosso-pharyngeal paralysis.

As to the nature of the lesion, it may be a hemorrhage, softening, cysts or patches of sclerosis (in disseminated sclerosis). According to Brissaud the lesion may occur bilaterally in the third frontal convolution, in the basal ganglia, in the cortex of one hemisphere and in the basal ganglia of the other. It may also occur in bilateral involvement of the motor segment of the internal capsule. In the majority of cases the lesion, which is either hemorrhage or softening, is extensive and involves the central ganglia and the internal capsule.

Etiology.—Any condition which is apt to lead to a morbid state of the cerebral blood vessels and consequently to hemorrhage, embolism and thrombosis is the cause of the disease. Syphilis, arteriosclerosis, cardiac diseases are therefore the chief factors.

Infantile Pseudo-bulbar Palsy has been also observed by Oppenheim and others in connection with diplegia (see this chapter). It is due to an arrested development or malformation of the lower parts of the central convolutions.

Symptoms.—In the majority of cases there is a hsitory at first of one attack of hemiplegia with some slight disturbance of phonation or deglutition. Soon a second apoplectiform seizure takes place and with it a complete picture of bulbar palsy is established.

All the symptoms of a true bulbar palsy will be observed here. A pseudo-bulbar individual will therefore present: immobility of the facies, stupid expression of the latter, a continuously open mouth, dribbling of the saliva, paralysis of the muscles of the cheeks, lips, tongue, palate, of mastication, of the vocal cords, abolition of the pharyngeal reflex; finally a nasal intonation of the voice, dysarthria with or without aphonia, dysphagia or complete inability to swallow, difficulty of respiration with attacks of dyspnea (see chapter on Bulbar Palsy).

In addition to these symptoms there are a few **special signs characteristic** of the pseudo-bulbar form. They are: absence of atrophy and of fibrillary tremor in the paralyzed muscles, preservation of normal electrical reactions and of the reflexes in the region innervated by the bulbar nerves. To this may be added a unilateral or bilateral hemiplegia, frequent involvement of the optic nerves (neuritis or atrophy), finally **mental symptoms.** The latter consist of a marked impairment of memory, of apathy, confusion and of dementia. Spasmodic **attacks of laughing or crying** are almost typical of pseudobulbar paralysis. The latter phenomenon is generally explained by an interruption of inhibition which

is normally trasmitted to the bulbar nucleus of the facial nerve through the cortico-bulbar fibers.

Course, Duration, Prognosis.—The disease is essentially progressive, but slow in its course. It may be interrupted or aggravated by slight apoplectiform seizures. The outlook is grave, as the termination is almost invariably fatal. It may last many years before death ensues.

Diagnosis.—The sharply defined symptoms enumerated above will enable to differentiate this form from the **true bulbar palsy** (see also the latter).

The **acute bulbar palsy** will be recognized mainly by the absence of mental symptoms and hemiplegia.

Asthenic Bulbar Paralysis (myasthenia gravis) presents a series of characteristic symptoms, such as extreme exhaustion upon the least effort, ptosis and palsy of other ocular muscles, inability to hold up the head, a special myasthenic reaction to electricity, etc.

Treatment.—Antisyphilitic medications may be tried even in cases without a clear specific history. As to the disturbance of respiration and deglutition, the same precautions should be taken as in the true bulbar palsy.

F. ASTHENIC BULBAR PARALYSIS (MYASTHENIA GRAVIS OF ERB AND GOLDFLAM)

Pathology.—Various changes in the central nervous system have been reported by a number of observers, but denied by others, so that in the state of our present knowledge there is nothing definite concerning the pathology of the affection. As in many cases, the autopsy findings have been absolutely negative, the disease is called: **bulbar palsy sine materia.**

It should be mentioned, however, that degenerative changes in the thymus (Weigert, Hansemann, Goldflam), tumors in the lungs, mediastinum and thymus were found in some cases. Degenerative changes have also been observed in the thyroid and in the pituitary body. Ependymitis of the aqeuduct of Sylvius has been reported. The muscles are very frequently altered: infiltration of cells between the fibers, hyaline degeneration. Buzzard (Brain, 1905) gave the name "Lymphorrhagia" to the infiltrated groups of lymphocytes scattered between the cells of the nuclei in the medulla and he considers them characteristic of myasthenia gravis.

To explain the pathogenesis of the affection several theories have been advanced. Among them the most important is the pluriglandular view. According to it the altered internal secretions of various glands play an essential rôle in the myasthenic syndrome: profound changes

in the glands have been found in a number of cases (see above). The pluriglandular apparatus has an effect on the bulbo-spinal motor system which is involved in myasthenia gravis. The disturbance of this apparatus has also a trophic effect on the muscles involved in the disease. Massolongo (*Riforma Medica*, 1912) believes that whatever the rôle of the glands with internal secretions may be, the clinical picture of the affection requires also a special inherent predisposition of an organic and morphological character of the bulbo-spinal centers. There is an inherent weakness of the gray motor centers of the mesencephalon and the spinal cord which produces a muscular exhaustion following an exertion.

Etiology.—The causes of the malady are as little known as its pathology. The prevalent opinion is in favor of a toxic nature: in a number of cases the disease developed after infectious diseases, also in instances of auto-intoxication.

Women are more frequently affected than men: children very rarely.

Symptoms.—After a brief prodromal period, consisting of headache and occipital pain, also of vertigo, in the majority of cases the symptoms begin with **ptosis.** The patient is compelled to contract the frontal muscles to assist the levator palpebræ in raising the eyelids. Very soon appears external ophthalmoplegia, so that strabismus and diplopia are among the earliest symptoms. When the facial nerve is paralyzed, both portions of it (upper and lower) are involved. This fact, together with the ptosis, gives the **facies** a peculiar aspect: the face is immobile, without wrinkles and somnolent, the head is raised when an object is looked upon.

The muscles of **mastication,** the **tongue, lips** and the **larynx** are in a paretic condition. Mastication, deglutition, phonation are therefore affected. Gradually the muscles of the neck become involved. The patient cannot then hold up his head, which has a tendency to fall forward or backward. This is quite characteristic of the disease. In a still more advanced stage the muscles of the trunk, of the abdomen and of the extremities become similarly affected. The patient is unable to sit up or stand up and has difficulty in breathing; dypnœa is marked on the least exertion. In the shoulder muscles the weakness is very marked. The patient is unable to hold up his arms.

In spite of the paralysis or paresis of the muscles of a more or less long duration, muscular atrophy is extremely rare, but a very important feature of the affected muscles is their special response to electrical stimulation. This is the so-called **myasthenic reaction.** It is an exhaustion reaction and consists of a gradual diminution and finally of a loss of

contraction, when a faradic electrical current is applied to the muscles. If after a brief rest the current is reapplied, the same phenmenon will be observed. This reaction is not observed from application of a galvanic current.

Sensations, reflexes and the function of the sphincters remain intact. Subjective aching in connection with exhaustion is frequently present. The reflexes become exhausted by frequent tests. Mentality is also preserved. One of the most characteristic features of the disease is the remarkable **variability** in the paralytic phenomena. Thus, for example, at a certain time of the day, especially in the morning, the patient is able to move about and exercise his muscles, but he soon gets exhausted. He may begin to speak, but gradually the voice gets weaker and finally complete aphonia sets in. He may attempt to whistle or blow out a candle; at first the acts are normal, but a second or third attempt are almost impossible.

Course, Duration Prognosis.—In the majority of cases the onset is with the muscles of the eyes and the disease has a descending course, but there are also observations showing that it may begin in the extremities and ascend. Speaking generally, the evolution of the symptoms is slow and progressive, although it may assume an acute form. Remissions are not infrequent and then the symptoms may also totally disappear. The disease may last ten or fifteen years. The prognosis is grave, as respiratory disturbances are not infrequent. Disturbances of deglutition are equally to be feared. Sudden death may ensue. The cases of so-called recovery are only long remissions.

Diagnosis.—The characteristic symptoms described above, viz. the onset, gradual descending development of symptoms, characteristic faces, myasthenic reaction, finally the variability in the paralytic phenomena are all sufficient facts to enable us to make a diagnosis.

From the chronic form of bulbar paralysis it will be distinguished by the absence of atrophy, fibrillary contractions and reactions of degeneration, and by the presence of the exhaustion.

Treatment.—It consists mainly of taking special care of the act of deglutition. The patient should be advised to eat very slowly. As soon as difficulty arises, artificial feeding must be resorted to. Absolute rest is indispensable in view of the extreme exhaustion upon the slightest effort. The presence of the myasthenic reaction in the affected muscles is a direct warning that electricity is a dangerous procedure; it must therefore **never be applied.** Adrenalin, extract of thymus and thyroidin have given some favorable results in some cases (Raymond, Buzzard). Sézary (*Semaine. Méd.*, 1913) suggests that when adrenalin is given in ade-

quate doses, benefit will be derived. He has seen cases in which subcutaneous injections of the whole extract or ingestion of fresh adrenals succeeded when the powder and epinephrin had failed completely. His experience demonstrates that certain slow changes in the adrenals, alone or with changes in other ductless glands, may induce a myasthenic syndrome. This justifies adrenal organotherapy, supplementing the adrenal with hypophysis treatment.

Among other remedies, Iodides, arsenic, phosphorus, iron and strychina should be tried.

G. HEMORRHAGE AND SOFTENING OF THE MEDULLA

Pathology.—Hemorrhages of the medulla are rare and are produced by the same causes as usual cerebral hemorrhages. They frequently invade the fourth ventricle. Softening is more frequent. It is caused usually by thrombosis, but also by embolism.

Etiology.—Traumatism of the cranium, infectious diseases, intoxications, eclampsia are the causes of hemorrhage. Syphilis is a frequent cause of thrombosis and cardiac diseases of embolism (see Hemorrhage and Softening of the Brain).

Symptoms.—The general clinical picture is that of labio-glosso-laryngeal paralysis (see this chapter). The difference lies in the rapidity of development of the symptoms.

(a) **Hemorrhage.**—The sudden onset may be followed by immediate death. In some cases the patient may remain comatose for hours or even days. In another series of cases (less acute) consciousness is regained and then the picture of bulbar palsy becomes evident, viz. dysarthria, difficulty of mastication and of deglutition. To these symptoms are added **paralysis of the extremities**, which presents several forms. The most frequent form is that of **crossed hemiplegia**, consisting of paralysis of the extremities on the side opposite to the lesion and of the ninth, tenth, eleventh or twelfth nerves on the side of the lesion. Besides a motor hemiplegia, there may be present a hemianesthesia. The symptoms will depend upon the seat of the hemorrhage.

(b) **Softening.**—The symptoms are identical to those of hemorrhage. The difference lies only in the mode of development. There is usually a prodromal period during which (especially in syphilitic cases) headache, somnolence, vertigo are present. The paralytic symptoms of the cranial nerves and of the extremities develop gradually.

Course, Duration, Prognosis.—In hemorrhages of the medulla, with a less sudden onset, death is caused by a difficulty of respiration or by

pneumonia from deglutition in the larynx. In softening, when death does not occur at the end of a few weeks, the symptoms usually show a tendency to improve. In specific cases especially the prognosis is very favorable. If the patient survives and a certain number of motor nuclei of the medulla were involved, atrophy with reaction of degeneration will remain in the affected muscles, but there is not the usual symmetrical distribution which is seen in typical chronic bulbar paralysis.

Diagnosis.—It will be difficult in the acute cases, but when the symptoms are less acute, the above symptoms (bulbar palsy associated with motor or sensory paralysis of the extremities) will enable one to make a diagnosis. Softening will be differentiated from the chronic bulbar palsy by the asymmetrical distribution of cranial nerve involvement and paralysis of the extremities. Pseudo-bulbar palsy will be recognized by the history of apoplectic seizures, by the mental symptoms and the spasmodic laughing and crying. Hemorrhage will be differentiated from softening by the mode of onset and evolution of the symptoms.

Treatment.—It will be that of hemorrhage or softening of the brain. Antisyphilitic treatment should be insisted upon. When difficulty of deglutition makes its appearance, artificial feeding must be resorted to.

H. OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY.

Among the earliest typical cases of thrombotic occlusion of this artery, verified by autopsy is that of Huhn (*N. Y. Med. Jour.*, 1897). Spiller has recently (1908) presented this subject in all its details. The general picture of the disease is briefly as follows: Sudden onset with or without loss of consciousness, unilateral paralysis of palate and vocal cord; difficulty of swallowing and expectorating; hemiataxia; syringomyelic sensory dissociation on the opposite side. In my case (*International Clinics*, V. IV) there was also right hemiasynergia, tendency to fall to the right side, pain in the neck, paresthesiæ on the left side where the objective sensory dissociation was present, paresis of the right side of the face, retraction of the right eyeball, narrowness of the right pupil. Pathologically a softening is found in one half of the medulla. The area affected lies between the spot where the artery begins to pass to the cerebellum and the middle of the nucleus of the hypoglossus. The formatio reticularis with its nuclei, the descending root of the fifth nerve, the nuclei of the seventh and ninth nerves, also the spino-cerebellar tract are all involved.

I. COMPRESSION OF THE MEDULLA

The medulla may be compressed (a) **suddenly** by dislocation or fracture of the atlas or axis; (b) **slowly** by tumors in the medulla itself or in the neighboring tissues and organs, by caries or other diseases of the

nieghboring bony tissue, by basal meningitis and quite frequently by aneurisms of the basilar or vertebral arteries.

Pathology.—The tissue of the medulla may be torn in the sudden cases. In the slow cases the bulb will be deformed or softened. Secondary degeneration is the consequence.

Symptoms.—In cases of gradual compression pain in the occiput and neck are the first symptoms. The head is kept by the patient in an absolute immobility. The neck is hyperæsthetic, active and passive movements are extremely painful. When the compression extends downward to the cervical cord, pain will be present also in the upper extremities. The bulbar compression will be manifested by disturbance of deglutition, of respiration, of heart beats. Death may come on suddenly. In cases of aneurism of the basilar or vertebral arteries, in addition to the occipital pain, there will be also signs of cerebral arteriosclerosis, viz. vertigo, noise in the ears, etc. The characteristic symptom is the intermittent bulbar manifestations (dysarthria, dysphagia, dyspnea, arhythmia, tachycardia). After they have existed for a certain time, they gradually improve and then disappear until the next attack. A paralysis of the extremities (hemiplegia or paraplegia) is almost always present. A very important symptom was pointed out by Hallopeau, Giraudeau and Killian. The heads of their patients were held in forced extension; as soon as they were flexed, respiratory disturbance occurred. When compression is produced by a tumor, in addition to the above symptoms, there will be also general symptoms of cerebral tumor and optic neuritis.

Course, Duration, Prognosis.—In sudden compression death is usually instantaneous from respiratory paralysis. In slow compression the patient is constantly threatened with rupture of the aneurism or softening and destruction of the tissue of the medulla. Cardiac and respiratory disorders are usually fatal. In basal meningitis of specific nature the symptoms may retrograde when under treatment. The disease may last months or years. Prognosis is grave.

Diagnosis.—Compression of the medulla must be differentiated from the typical labio-glosso-laryngeal paralysis. In the latter there is no pain in the neck, no motor or sensory paralysis of the extremities.

Treatment.—Antisyphilitic treatment should be tried in all cases of compression of the medulla, even if there is no history of syphilis. There is no medication that could arrest this exceptionally grave disease.

DISEASES OF THE PONS

The pons is a very important portion of the brain. It contains sensory and motor tracts going to and coming from the brain. Through the

middle cerebellar peduncles it is connected with the cerebellum. Moreover a number of cranial nerves appear at the borders of the pons on their way from their respective nuclei in the medulla. Consequently the symptomatology of pontine lesions must be complex and vary from one case to another according to the part of the pons involved.

HEMORRHAGE, SOFTENING, TUMORS

Pathology.—**Hemorrhagic** foci are usually located in the median line. They may spread downward or forward. They are rare. **Softening** is quite frequent. **Thrombosis** of the basilar artery is usually found. The destruction (softening) sometimes occupies the largest part of the pons; sometimes it is on one side and sometimes on both. In aged people the disintegration of the pontine tissue occurs in small areas, which sometimes are multiple. **Tumors** are not frequent. Tubercles are the most frequent. Next in frequency are gummata. Gliomata may also occur. Cysts, cancer, abscess are very rare. (For a detailed description of the effect of tumors on the nervous tissue, see Tumors of the Brain.)

Etiology.—Inflammatory or degenerative conditions of the arteries will lead to their rupture and produce a hemorrhage. Softening is caused by embolism and thrombosis; the former is exceptional, the latter is caused by a syphilitic or atheromatous degeneration of the blood vessels. In cases of tumors, except gumma and tuberculoma, there is frequently a history of traumatism of the head.

Symptoms. (a) **Hemorrhage.**—The onset is sudden and loss of consciousness especially in large hemorrhages is always the first symptom. In small hemorrhages there may be no loss of consciousness. When the patient has regained consciousness, one will observe the following symptoms, which are quite characteristic of a pontine hemorrhage: (a) contracture of the extremities accompanied by generalized convulsions, or (b) unilateral epileptiform convulsions; (c) conjugate deviation of the eyes and of the head; (d) crossed paralysis.

The conjugate deviation presents special features. If the pontine lesion is destructive and leads to a unilateral paralysis, the eyes will be turned toward the paralyzed side. If it is irritative and leads to unilateral convulsions, the eyes will be turned away from the affected side.

Crossed paralysis is a very frequent occurrence. It consists of a palsy of the limbs on the side opposite to the lesion and of involvement of one or several cranial nerves on the side of the lesion. Besides the palsy the symptoms will therefore depend upon the cranial nerve involved. When the lesion is low down in the pons, the seventh nerve will be

involved; there will be paralysis of the opposite side of the body and facial palsy on the side of the lesion. When the hemorrhage is near the upper border of the pons, the sixth nerve alone may be involved. There will be palsy of the external rectus, hence internal strabismus.

Myosis, dysarthria and dysphagia are quite frequently observed in pontine hemorrhage. Death occurs from cardiac or respiratory paralysis.

(b) **Softening.**—The onset is rarely sudden, more frequently slow. In the latter case it is preceded by prodromal symptoms, such as headache and vertigo.

The attack itself is immediately preceded by paresthesia in the limbs which are to be affected. Then gradually, but progressively, the paralytic symptoms make their appearance. Crossed hemiplegia is the usual result. Dysarthria and dysphagia are quite frequent.

When the softening is due to a thrombosis of the basilar artery, there are also general symptoms besides the crossed paralysis. They are: somnolence, potic neuritis, sometimes delirium.

(c) **Tumors.**—Tumors may originate in the pons or in the vicinity at the base, most frequently in the ponto-cerebellar angle. For the latter see page 217. Similar to cerebral tumors, pontine neoplasms present **general** and **focal** symptoms. The first are: headache, vomiting, vertigo, insomnia, optic neuritis, etc. (see Tumors of Brain). The headache which is frequently occipital may be accompanied by stiffness of the neck. Percussion of the occiput and of the first cervical spinous process may be painful. Optic neuritis appears late or in the terminal stage of the disease. The focal symptoms are chiefly: **crossed** or alternate **paralysis,** which is slow in development. A very frequent occurrence is subjective pain in the paralyzed muscles of the limbs and face (when the latter is involved). The paralysis may be unilateral or bilateral. The cranial nerves-symptoms depend upon the nerve or nerves affected by the tumor. In exceptional cases the nerves alone are involved and there may be no paralysis of the extremities. There are also cases in which the nerves are not involved and there is only paralysis of the limbs. This possibility will occur only in lesions of the upper part of the pons. When the lesion is in the middle of the pons there may be a paralysis of the limbs also of the face on the side opposite the lesion, which then lies above the decussation of the central facial fibers. If the tumor is so extensive as to involve the medulla, cerebellum or other neighboring tissue, the symptomatology will be more complex: unilateral paralysis of the palate, vocal cords, tongue, ninth, tenth, eleventh, twelfth nerves. Polyuria, disturbance of respiration and of heart beat are indications of bulbar involvement. Vertigo, titubation show involvement of cerebellar peduncles. Diminution of central

visual acuity depends upon the involvement of the anterior quadrigeminal bodies; deafness upon a lesion of the posterior quadrigeminal bodies.

Sensory disturbances are present mainly in tumors of the tegmentum (see Anatomy); there may be either hemianæthesia or generalized anæsthesia. Hemianæsthesia is quite frequent. Not infrequently it accompanies the hemiplegia. The disturbance of sensation is according to Marinesco due to the involvement of the mesial fillet and formatio reticularis in its ventral part (see Anatomy).

Like in hemorrhage and softening, dysphagia and dysarthria are also present and even quite frequent in tumors. Dysphagia is caused by involvment of the medulla, dysarthria—by involvement of the central fibers of the hypoglossus. Anarthria implies a bilateral lesion in the pons.

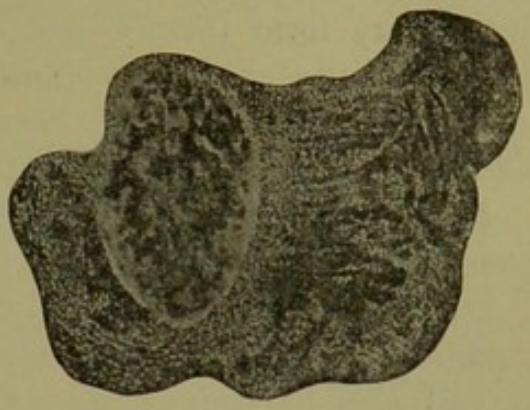


FIG. 80.—TUBERCLE IN THE PONS. SECTION BETWEEN THE POINTS OF EMERGENCY OF THE 7TH AND 5TH NERVES. (*Flatau, Jacobsohn and Minor.*)

Ataxia is frequent in pontine lesions. It is usually unilateral. It is probably due to the implication of the fillet which produces sensory disturbances. When the gait and station are disturbed, the cerebellum or its peduncles are probably involved.

Crossed Paralysis.—From the foregoing remarks it can be seen that crossed paralysis is a common feature of diseases of the pons. This form of paralysis may be **motor** and **sensory**. There are two varieties of crossed paralysis: **superior** and **inferior.**

In the **superior crossed paralysis** (**Weber's syndrome**) there is hemiplegia on the opposite side and palsy of the oculo-motor nerve on the side of the lesion. The hemiplegia is total, viz. face, arm and leg are involved. The facial paralysis has the same characteristics as in cerebral hemiplegia, viz. only the lower half of the face is paralyzed. When this form of crossed paralysis is accompanied by a tremor of the paralyzed limbs it is called: "**Benedikt's** syndrome."

The **inferior crossed paralysis** (Millard-Gubler's syndrome) is characterized by a paralysis of the arm and leg on one side and of the facial nerve, also sometimes of the abducens nerve, on the opposite side. In certain cases the hypoglossus or other cranial nerves participate. When the root of the sixth nerve is involved there is paralysis of the external rectus with internal strabismus, but when its nucleus is affected, the conjugate movements of the eyes toward the lesion will

be abolished if the lesion is destructive, but if the lesion is irritative, there will be a conjugate convulsion of the eyes toward the lesion. Other nerves may also be affected. The involvement of the fifth is not at all rare. Tactile sensation will then be affected on the side of the lesion in the area of distribution of the fifth, and in the limbs on the opposite side. The eighth nerve is rarely involved, but if it is, there will be impairment of hearing and subjective sensations of sounds on the side of the lesion. Crossed paralysis may be exclusively **sensory** or else **sensorimotor.** In the sensory form there is anæthesia of the limbs and trunk on one side and of the face on the opposite. The reason of the crossed character of sensory disturbances is because the decussation of the sensory fibers takes place in the lower part of the medulla (see Anatomy).

Prognosis.—It is unfavorable, especially in tumors. Only in syphilitic cases improvement can be expected.

Diagnosis.—Pontine hemorrhage will be differentiated from intra-cranial hemorrhage in general mainly by the presence of alternate paralysis, but also by convulsions and hyperæsthesia of the affected limbs. The conjugate deviation of the head and eyes presents a very important diagnostic point. Landouzy put down the following rules:

	Pontine Lesion	*Cerebral Lesion*
In paralysis.....	Eyes turned toward the paralyzed side.	Eyes turned toward the non-paralyzed side.
In convulsions.....	Eyes turned toward the unaffected side.	Eye turned toward the affected side.

Softening is recognized by prodromal symptoms. The evolution of tumors is slow and progressive.

The distinction between **extra-pontine** and **intra-pontine** tumors is of great importance, as surgical intervention depends on it. The type of extra-pontine tumors is tumor of the **ponto-cerebellar angle.**

The chief characteristics are as follows:

In tumors of the angle the manifestations of intra-cranial pressure are associated with unilateral palsy (on the side of the tumor) of cranial nerves, especially fifth, seventh and eighth. Hypoacousia and vertigo with tinnitus are often the first signs of localization. To these are frequently added dysarthria, dysphagia and paresis of associated movements of the eyes when the patient turns his head to the side of the tumor. Besides, there are frequently cerebellar manifestations on the side of the tumor (asynergia, etc.). The course is progressive and gradually new symptoms appear.

Tumor of cerebello-pontine angle are usually encapsulated and benign.

Their detection is based chiefly on symptoms of compression of the cranial nerves at the base. A simple decompressive operation is indicated in cases of a very precarious estate of the patient, in cases of multiple tumors and in cases of cancerous metastasis. In all other cases a radical operation is advisable. If the localization is made, one cerebellar fossa should be opened.

Intra-pontine tumors commence often by associated progressive paralysis of the lateral movement of the eyes, predominant on the side of the lesion. It is accompanied by motor and sensory disturbances on the side **opposite** the tumor (crossed paralysis.) The implication of the auditory nerve is only a late symptom.

Treatment.—The management is limited to the specific treatment. Surgical intervention is almost impossible on pontine tumors.

Peduncular Syndrome.—The cerebral peduncles contain motor and sensory tracts connecting the brain with the medulla and spinal cord. The sensory fibers lie in the upper or dorsal portion, the motor in the lower or ventral portion (see Anatomy). Besides, the superficial origin of the third nerves lie between the crura near the upper border of the pons. Hemorrhage, softening, and abscess occur very rarely. Tumors are less infrequent. They are usually tubercular. The characteristic feature of a lesion of the crus will therefore be a palsy of the third nerve on the same side and hemiplegia on the opposite side. If the tegmentum is involved, the hemiplegia will be accompanied by a hemianæsthesia. This is Weber's syndrome mentioned above. If the paralyzed limbs are affected with tremor, we have Benedict's syndrome. Ptosis, external strabismus, diplopia, mydriasis—are the ocular symptoms due to third nerve palsy. If the lesion of the crura is very superficial and slight, there will be only third nerve palsy without hemiplegia. If the tumor invades both crura and both third nerves, there will be bilateral opthalmoplegia and paralysis of the four limbs. Sometimes the affected limbs may present ataxia (Krafft-Ebing, Marinesco and others).

CHAPTER XIX

DISEASES OF THE SPINAL CORD

ALL the affections of the spinal cord may be classified into two great groups: **systemic** and **diffuse.** In the first the lesion is confined to a certain system of fibers (tracts) or to portions of the cord which have a certain definite function (cells of anterior cornua). In the second the lesion involves gray and white matter in a diffuse manner.

A. SYSTEMIC DISEASES OF THE SPINAL CORD

I. Tabes Dorsalis (Locomotor Ataxia) (Posterior Sclerosis)

The first intimation of the existence of the disease was given in a vague manner by Romberg in 1851, but it is mainly Duchenne that presented a full and clear description of the malady.

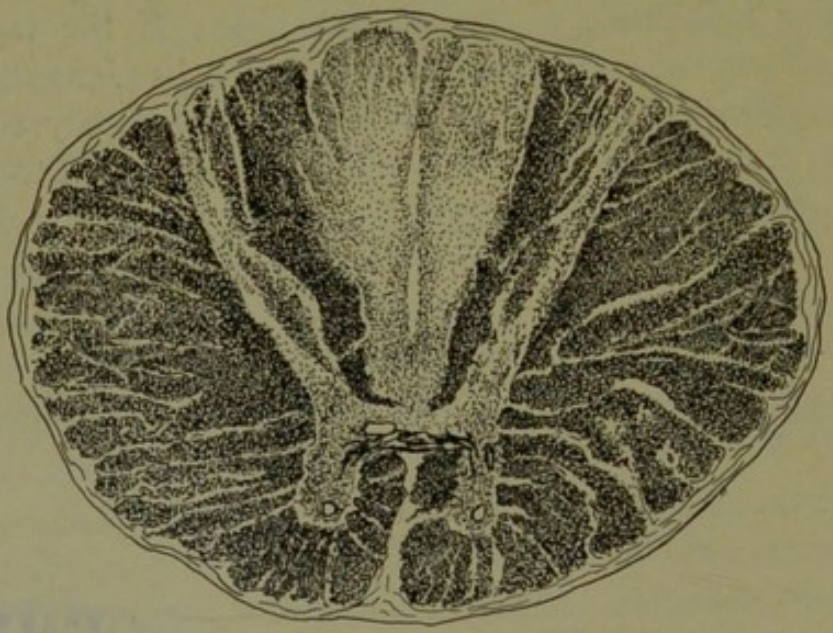


FIG. 81.—POSTERIOR SCLEROSIS. *(Original.)*

Pathology.—The chief characteristic lesion of tabes is a gray **degeneration of the posterior columns** and **of the posterior roots.** In advanced cases this condition can be seen even macroscopically. In the majority of cases the disease begins in the dorso-lumbar segment of the cord. The initial lesion is in the external portion of Burdach's columns, which

corresponds to the ascending intra-spinal fibers of the posterior roots. Lissauer's tract, the fibers of which surround the end of the posterior roots, is also involved in the first stage of tabes. In the dorsal and cervical segments of the cord Goll's columns are mainly affected. In advanced cases the degeneration affects both tracts (Goll's and Burdach's) through the entire cord. The **posterior roots** participate in the pathological process, so much so that some authors are inclined to consider them with Nageotte as the point of departure of the affection. Nageotte considers radicular neuritis as the primary lesion of tabes. He also calls attention to a meningitis at the level of the posterior roots, which is now proven to be a constant condition. The **spinal ganglia** in which the posterior roots originate have been found altered in a number of cases (atrophy of the cells and of their prolongation). The sensory nerves, the peripheral sensory prolongations of the spinal ganglia, are frequently involved. It can be therefore seen that the following important sensory elements are affected in tabes: peripheral sensory nerves, spinal ganglia, posterior roots, posterior meningitis, posterior columns in the cord. Otherwise speaking, a disease of the ascending **sensory neurones** constitutes the characteristic lesion of tabes.

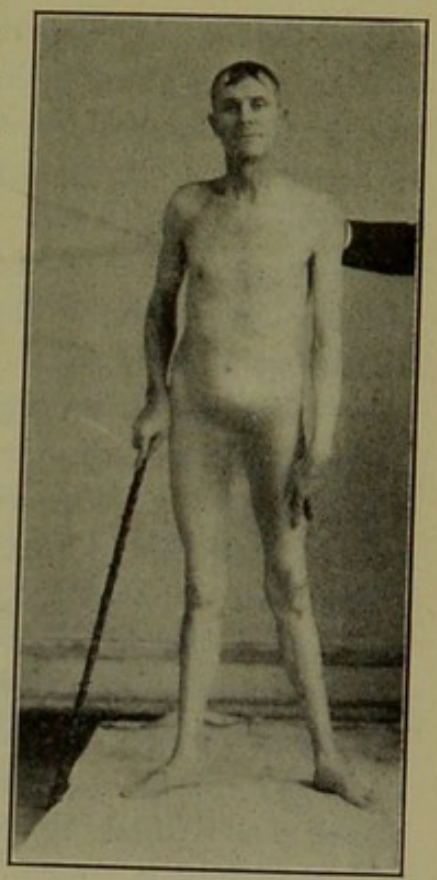


FIG. 82.—TABES. STATION: LEGS WIDELY SEPARATED, FEET EVERTED.

The histological changes consist of an enormous diminution or of a complete disappearance of the white fibers. The fibers that persist are markedly atrophied because of breaking up of the myelin and of reduction in size of the axis-cylinders. They are all substituted by proliferated neuroglia and connective tissue. The blood vessels are frequently altered: their walls are thickened (endo- and peri-arteritis).

When the disease ascends to the medulla, the roots of some of the cranial nerves become involved. The **optic nerve is very frequently affected** (gray degeneration). The roots of the fifth and eighth nerves, and the nucleus of the tenth nerve, have been found degenerated in some cases. Changes in the cells of ciliary ganglion have also been observed.

The gray matter is also sometimes diseased. In the posterior cornua and Clarke's columns the short fibers emanating from the cells are found

degenerated or atrophied. The cells of the anterior cornua are rarely found altered except in cases of tabes associated with muscular atrophy.

The meninges at the level of the posterior roots are thickened and present a certain degree of leptomeningitis.

The **pathogenesis** of tabes is debatable. In view of the fact that it is a disease of the sensory neurone, it is difficult at present to determine at what level of the entire sensory neurone lies the point of departure of the pathological process. According to the majority the degeneration of the posterior roots and columns follows the initial alteration of the spinal ganglia. Others believe that the posterior roots open the disease and still others are inclined to believe that the initial lesion is in the peripheral sensory nerves.

Symptoms. I. **Motor.**—(a) The **station** and **gait** are characteristic in a well-developed case of tabes. The patient experiences a difficulty in standing: for fear of falling he separates his legs widely and even then his body oscillates. When an attempt is made by him to close his eyes and bring his feet close together, there is a tendency to fall. The latter test is called **"Romberg's sign."** In walking there is an **uncertainty,** an **incoördination** of movements of the legs, there is **ataxia.** The patient lifts his legs high, feet everted, then throws them forward and laterally, and being unable to control the movements, he drops them on the ground with force. This **ataxic gait** is sometimes so pronounced that the patient is unable to make a few steps without falling. In initial stages of tabes the ataxia may be extremely slight. The patient notices then that he is unsteady in going up or down stairs or in walking in a dark place.

When the incoördination is not marked in the gait, the ataxia may be revealed by making the patient to start to walk suddenly, stop suddenly or turn abruptly. Also can it be detected in a recumbent position: when he is told to raise the leg and touch with his foot your hand, this act will be accomplished after several lateral oscillations of the leg (Fig. 82).

When the upper extremities are affected, there is a difficulty in performing fine or delicate acts, as writing, threading a needle, etc. When the patient is asked to touch the tip of his nose with the tip of his index, the latter will make several movements above and below or laterally before the nose will be reached. Another test for ataxia of the upper extremities is to bring the tips of the two indexes together after the arms have been widely separated. Ataxia in tabes is due to loss of sense of position in the muscles and articulations.

(b) **Hypotonia of the Muscles.**—There is an unusual flaccidity and relaxation of the muscles of the affected limbs, so that the latter can be easily given any position. The entire limb, for example, can be extended

and raised, so that it is brought in contact with the trunk. The knee-joint may be overextended and produce a genu recurvatum. In the upper extremities the fingers can be so extended as to form a right angle with the dorsum of the hand. The hypotonia may also affect the muscles of the trunk, so that exaggerated movements (passive or active) can be produced.

II. **Sensory.**—They are **subjective** and **objective.** Tabetic **pain** is very characteristic and one of the earliest subjective symptoms. It is usually **lancinating,** knife-like, very sharp and brief. Sometimes it is burning or boring, associated with numbness or tingling. In the majority of cases it is paroxysmal and may last from a fraction of a minute to an hour. The attacks may be frequent or appear only at rare intervals. Between the paroxysms the skin may remain hypersensitive. The pain occurs usually in the lower extremities and ulnar side of the arms. It frequently invades the viscera and constitutes then a characteristic symptom, called **"crisis."** In gastric crisis the pain is in the hypochondrium and an attack may be followed by vomiting. There are also: vesical, renal, testicular, laryngeal and anal crises.

Instead of being intermittent, the pain may remain permanent and fixed. This occurs in the so-called **"girdle pain,"** which consists of a sense of constriction around the waist or chest. The phenomenon is frequent and typical of tabes. Among other subjective sensations there may be instead of or besides pain also various **paræsthesiæ,** as numbness, tingling, coldness, burning, etc. They may affect any portion of the body, but more frequently the lower extremities.

Loss of position of the limbs is present in a large number of cases. The patient is unable to tell the position of his limbs (lower most frequently) while in recumbent position. It can be elicited by closing his eyes and placing one leg over the other or by flexing and extending a portion of a limb.

The objective sensory disturbances are: **anæsthesia** and **analgesia.** The loss of sense of pain and touch is met with in tabes over small areas. In the upper extremities it is usually on the ulnar side of the forearms. In the lower extremities it is usually on the soles of the feet: it is quite common for tabetics to have the impression of **walking on cotton** or on a carpet. The loss of pain-sense may not be confined to the skin; it may be present also in the deeper tissues, as muscles, bones, joints. Painless fractures or dislocations are not uncommon. Vibration sense (see page 69) is also lost. The following visceral analgesias are not infrequently observed in tabes: testicular, epigastric, mammary, tracheal, ocular; pressure on all these organs provokes no pain. Pressure

on the ulnar nerve (Biernacki) and on the external popliteal nerve (Bechterew) causes no pain. Abade called attention to analgesia observed when the tendon Achilles is pinched. The loss of profound sensibility is sometimes a very early sign of tabes.

In exceptional cases there may be **hyperalgesia** instead of analgesia. In such cases the slightest touch will cause unbearable pain.

The **temperature sense** may also be altered to the same extent as touch and pain.

In some cases there may be a **perverted sense,** as for example a prick with a needle will give the sense of burning. Finally there are cases in which the **localization of sensations** is disturbed, as for example the touch or pin prick will not be felt by the patient on the spot touched, but on a remote part of the body.

III. **Special Senses.** (a) **Vision.**—Among the earliest ocular signs in tabes is the **Argyll-Robertson pupil.** It may precede the ataxia ten or fifteen years. It is characterized by a sluggishness or loss of light reflex and preservation of the accommodation reflex. It means that when a light is thrown into the pupil, the latter remains immobile instead of contracting. This sign may be bilateral as well as unilateral. In advanced cases the accommodation reflex may also be abolished. In some cases Argyll-Robertson pupil may be only transitory, disappear and reappear.

Erb called attention to the following phenomenon. Normally when the skin is pinched, the pupils contract; in early tabes this reflex disappears.

Tabetic **pupils** are quite characteristic. In the majority of cases they are very small (**myosis**). In some cases they may on the contrary be much dilated. They may also be irregular in contour and unequal: one myotic, the other mydriatic.

Paralysis of the ocular muscles is frequent. It presents these peculiarities, that it does not affect all the muscles equally and that it is transient in the early stages of tabes. **Ptosis** is very frequent, internal or external strabismus is also quite common. The patient often complains of **diplopia.** The latter is a very early sign. These palsies disappear and reappear until the disease is well developed, when the ocular paralysis becomes permanent.

As to the **visual function,** there is almost always a **diminution of visual acuity.** The loss of vision may be acute or more frequently chronic. Ophthalmoscopic examination reveals an **atrophy** of the optic nerve (gray atrophy). Optic atrophy and Argyll-Robertson pupil are early and certain signs of tabes, even when the other symptoms are hardly noticeable. In some cases the onset of blindness arrests the evolution of the

other tabetic symptoms. Contraction of the visual fields especially for colors is not rare.

(b) **Audition.**—When the trigeminal area becomes involved (see Pathology), the result will be a diminution of the acuity of hearing which may go to complete deafness, also various noises in the ears and finally vertigo analogous to that caused by ear diseases.

(c) The **olfactory** and **gustatory** senses are occasionally found to be disturbed.

IV. **Reflexes.**—Loss of **patellar tendon** reflex (**Westphal's sign**) is one of the earliest symptoms of tabes. At first the knee-jerks may be only diminished, but later they disappear entirely. The loss or diminution may be unilateral, but more frequently bilateral. In doubtful cases it is necessary to have recourse to the method of reinforcement (**Jendrassik**): the patient is told to place one hand in the other and to pull; a short blow over the patellar tendon will then enable more readily to determine whether the knee-jerk is present or absent. The patellar tendon reflex persists in the "superior tabes" (see Pathology). Loss of **Achilles tendon** reflex is very frequent in tabes. Its disappearance may occur long before that of the knee-jerk.

The explanation of the abolition of the tendon reflexes is found in the breaking up of the reflex arc, the sensory portion of which is diseased. The cremasteric and anal are very frequently involved: they are diminished or lost.

V. **Sphincters.**—Disturbances of the function of the **sphincter of the bladder** appears early in the course of tabes. Difficulty of expelling the urine is common. Some patients do not feel the necessity of urinating. In some cases there is on the contrary marked frequency and imperative micturition. In the advanced cases incontinence is the usual occurrence.

The **sphincter** of the rectum is also commonly involved. Constipation, difficulty of defecation, tenesmus, incontinence—all occur in tabes.

VI. **Trophic Disturbances.**—They affect the general nutrition and individual organs or tissues. The majority of tabetics are usually pale, emaciated, with drawn features, sunken eyes.

Among local dystrophies the **arthropathies** occupy the first place. They were first described by Charcot in 1868. They may be of **atrophic** and **hypertrophic** types. The first consists of a more or less complete destruction of the cartilage and of the bony extremities of the joint; the capsule is in a state of relaxation. Extreme mobility of the joint is the consequence. Luxation or subluxation of the bones occur then easily. Quite frequently the synovial membrane, which is either extremely thin or thick, contains an abundant serous fluid. In the hypertrophic variety

there is formation of new bony tissue, of bony excrescences in the joint. The principal characteristics of tabetic arthropathies are: absolute absence of pain and a special œdema, which upon pressure does not leave a depression. As to the **painless joint,** it is so remarkable that patients thus affected will walk with dislocated joints without the least suffering.

The seat of the arthropathy is mostly in the knee-joint; next in frequency is the foot, then the hip, shoulder, elbow and inferior maxillary bone. The **"tabetic foot"** deserves special mention. It presents a

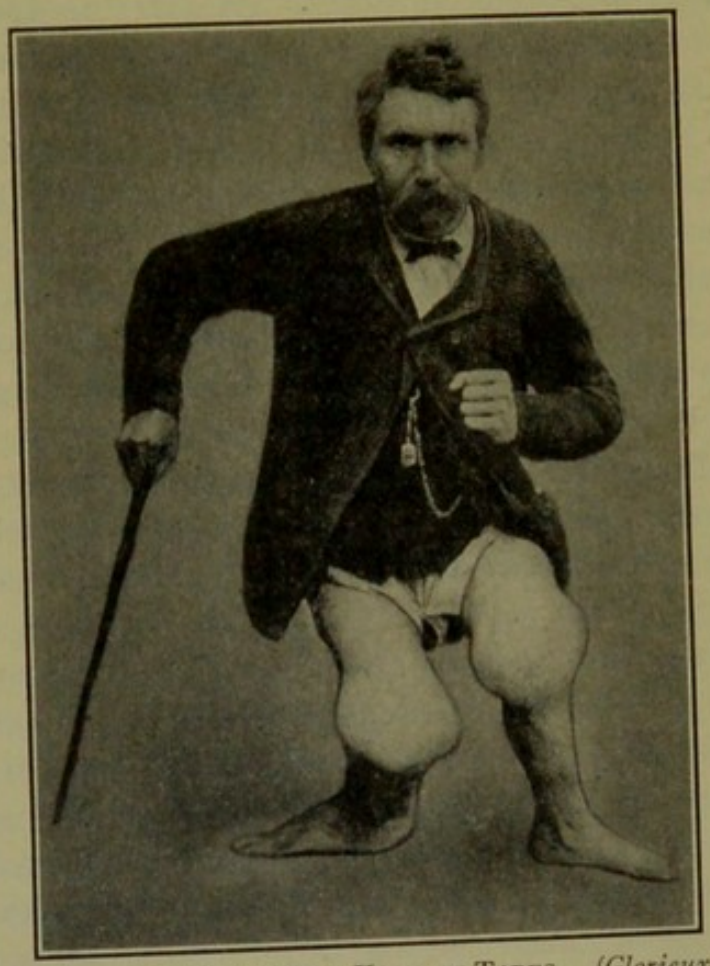


FIG. 83.—BILATERAL ARTHROPATHY OF THE KNEE IN TABES. (*Glorieux and Van Gehuchten.*)

thickening of the internal border of the foot and pathologically consists of an involvement of the ligaments, bones and articulations. The patient walks on the inner side of the foot, which is everted.

The **bones** also suffer in tabes. There is rarefication of the osseous tissue. **Spontaneous fractures** take place with great facility: a very slight traumatism is likely to produce them. They are also painless. The formation of a callus is usually normal.

Among other trophic disturbances should be mentioned: "perforating ulcer" (**mal perforant**). It consists of a painless ulceration on the plantar surface of the foot and especially at the level of the great and little toes.

Muscular atrophy not infrequently occurs in the lower extremities, but also occasionally in the upper. Hemiatrophy of the tongue had been observed. **Cutaneous disturbances,** as herpes, gangrene, zona, hyperhidrosis, falling of the hair and of the teeth, brittleness of the nails, occasionally develop in the course of tabes.

Vasomotor disturbances are frequently observed. They are: coldness of the extremities, anemia or blanching of the extremity of a limb ("dead finger"), œdema of the extremities.

There are at present two views concerning the pathogenesis of the trophic disturbances. According to some neurologists the latter are the result of changes in the cord, namely in its anterior cornua. Others believe in a peripheral origin, viz. in a neuritis of the nervous filaments distributed in the bones, articulations, etc. As to the arthropathies, carefully collected records show a history of trauma as an exciting cause. (see my contribution in *Med. Record*, 1909).

VII. **Visceral Disturbances.** (a) **Gastro-intestinal Canal.**—Apart from "gastric crises," mentioned above in connection with sensory disturbances, tabetics not infrequently present digestive disturbances. Fournier called attention to "tabetic anorexia," in which the patient loses the sensation of hunger. The intestinal trouble consists of tenesmus, of imperative or frequent defecation. Diarrhœa is quite frequent in the preataxic period.

(b) **Larynx.**—Laryngeal disturbances may consist of dyspnœa, paroxysms of cough and "laryngeal crises." The pathogenesis of the latter is obscure. However most frequently a paralysis of the dilators of the glottis is found. The cause of the laryngeal disturbances lies probably in the involvement of the ninth, tenth and eleventh nerves.

Among the rare symptoms may be mentioned the syndrome of Avelis, viz. hemiatrophy of the velum palatinum associated with paralysis of the recurrent nerve. It is particularly encountered in tabes of the upper portion of the cord. The paralysis of the internal branch of the eleventh nerve which produces the hemiatrophy of the palate is probably due to a meningeal involvement of the root fibers of that nerve.

(c) **Genital Apparatus.**—It is frequently disturbed. Sexual desire with orgasm is increased at the beginning of tabes. As the disease progresses, impotence gradually makes its appearance. Sterility is frequent in women.

(d) **Blood Vessels.**—Atheromatous changes are present in many cases. This is probably the cause of paroxysms of "angina pectoris" occasionally observed in tabes.

(e) **Brain.**—Mental disturbances are sometimes observed, especially

toward the end of the malady. It is mainly cerebral **depression,** but may be also cerebral exaltation. Kraepelin speaks of hallucinatory psychoses occurring in the course of tabes, but he does not affirm that they are directly and exclusively conditioned by tabes. The amaurotic form of tabes is particularly associated with some mental manifestations such as delirium, hallucinations. Apoplectiform seizures may occur. The cerebral symptoms occur probably in those forms of tabes which terminate in paresis. The relation of both diseases is remarkable. There are tabetics in whom symptoms of paresis develop and paretics in whom symptoms of tabes are present. The involvement of the posterior columns in paresis is almost constant.

Claude (Encéphale, 10 Déc., 1912) has recently brought forward anatomical facts showing the existence of syphilitic meningo-encephalitis in tabes, viz. sclerotic and gummatous nodules, old adhesions of the dura, sclerotic thickening of the pia, foci of hemorrhagic softening with peripheral inflammatory reactions, foci of œdematous encephalitis. During life the patient presented a delirious state, confusion, disorientation. It is evidently a case of tabes with localized syphilis of the central nervous system, but not typical of paresis.

Course, Duration and Termination, Prognosis.—All the cases of tabes are not identical to each other. Generally speaking, **three periods** are observed in the course of the disease: **preataxic, ataxic** and the **terminal.** This division is by no means present in every case. There are cases which are ataxic from the very onset. It is impossible to foretell in any given case the mode of the evolution of the symptoms. There are also mild and aggravated forms. In certain cases the symptoms reach a certain degree of development and remain stationary. Others progress very rapidly. In the **"amaurotic form"** the blindness and optic atrophy may be the only symptoms for years before other signs of tabes make their appearance.

The course may be extremely slow and last even thirty years, although on an average the disease lasts ten or fifteen years. The sphincter disturbances hasten the disease. Death is the usual termination. Tabetics usually die in cachexia, but also from some intercurrent disease (pneumonia, tuberculosis). Sometimes infection from cystitis or pyelonephritis or else from a bedsore is likely to shorten the patient's life. The prognosis is therefore grave.

Return of the patellar tendon reflexes in the course of tabes deserves some comment. After having been abolished for some time, the knee-jerk may reappear. This may happen either after an attack of hemiplegia, after the development of amaurosis, finally after a rigorous antisyphilitic

treatment. The mechanism by which the return of the reflex is done is not entirely clear. At all events, that the reflex may return, it is necessary that the anatomical pathways are not completely destroyed, that the loss of the reflex is not absolute.

Diagnosis.—The described **seven** cardinal symptoms are sufficient for making a diagnosis of tabes. The **eye manifestations** require special emphasis. The ocular symptoms enumerated above may be encountered in syphilis and therefore amenable to treatment; consequently the prognosis is more favorable. A few differential points are necessary. If ocular symptoms in tabes present marked fluctuation, they are due to syphilis itself and therefore improvement may be expected from treatment. If they are due to tabes, they are beyond reach. Myosis and reflex immobility of the pupil or dilatation of pupil with paralysis of accommodation apparatus are more likely signs of syphilis, hence amenable to treatment. Simple atrophy of optic nerves is very frequent in tabes, exceptional in syphilis. In tabes the papilla is pale from first; in syphilis the pallor comes on gradually after visual disturbances have developed. Benefit from specific treatment will also enable one to differentiate these disturbances. Syphilitic iritis or perforation of palate are never encountered in tabes. Reflex immobility of pupils is one of the earliest and most frequent symptoms of tabes. It may precede ataxia for a number of years.

Recognition of **incipient** tabes is of utmost importance. The following manifestations are to be borne in mind. Some disorder in the function of the bladder, such as delay in micturition, or slight incontinence after drinking; pain suggestive of rheumatism, neuralgia of the trigeminal and intercostal nerves; paræsthesia in the perineal and scrotal regions; the ocular disturbances; absence of Achilles tendon reflex; relaxation of muscles—hypotonia especially of the knees; tendency to ataxia which can be detected after a careful examination of individual muscles of segments of the limbs; lack of perception of vibration of the tuning fork when applied to the tibia; gastric crises—consisting at first of retching, salivation and hiccough. In spite of the facility with which a diagnosis of tabes can be made, in some cases difficulties may be encountered. The following are the affections from which tabes should be differentiated.

Multiple neuritis presents in common with tabes pain, loss of knee-jerks, incoördination, Romberg's sign, but the symptoms absent in tabes are: tenderness of the nerve-trunks and sensory disturbances, which follow the distribution of the nerves. Finally Argyll-Robertson pupil is never present in neuritis.

Multiple sclerosis can be recognized by nystagmus, intention tremor and characteristic staccato speech.

Friedreich's ataxia presents like tabes loss of knee-jerks, but it is differentiated by the existence of nystagmus and special speech and by absence of pain.

Cerebellar diseases (tumor, hemorrhage, etc.) simulate sometimes tabes, but the difficulty in walking is not like a tabetic ataxia, but a titubation. Moreover cerebellar asynergia does not exist in tabes. Finally absence of pain and of visceral disturbances are in favor of a cerebellar disease.

Recent researches have demonstrated that in the course of tabes a **lymphocytosis** takes place in the cerebro-spinal fluid of the majority of cases. Wasserman reaction in the cerebro-spinal fluid is almost invariably positive in untreated cases. These two factors are certainly a valuable addition to the semeiology of tabes in doubtful cases.

Etiology.—Syphilis plays the most important if not the exclusive rôle in the genesis of tabes. According to Fournier, 93 per cent. of tabetics present a clear history of syphilis. Erb and Oppenheim observed patients personally free from specific infection, but whose parents had had syphilis. With the discovery of Wasserman reaction the syphilitic origin of tabes is finally settled.

The posterior sclerosis is produced not directly by syphilis, as the disease develops many years after the initial syphilitic lesion. Tabes belongs therefore to Fournier's **"parasyphilitic affections."** As to other causes mentioned by some writers, they are: cold, exertion, excesses and traumatism. In my opinion they may be considered only as predisposing factors.

The age at which tabes occurs is usually between thirty and forty, although juvenile tabes is not unknown. I have seen as young as eighteen and as old as sixty-five. Tabes is infrequent in women. It is rare in negroes.

Treatment.—In view of the syphilitic etiology of tabes, mercury and iodides have been recommended, but it is doubtful if these drugs are capable of altering a sclerosis of the posterior columns. However as some favorable results have been obtained by competent neurologists, it is advisable to apply this treatment in every case of tabes.

Mercurials should be given first. Inunctions or hypodermic injections are preferable to internal administration. Precautions must be taken against mercurial intoxication. The patient should be instructed to observe himself closely. In case marked salivation, swelling or soreness of the gums and of the tongue, pain in the abdomen, diarrhœa, ele-

vation of temperature are noticed, mercury should be discontinued without delay. In one case under my care the patient failed to report to me in spite of my urgent warning and kept on using the mercurial inunctions; only a few doses brought on such an enormous swelling of the tongue that a tracheotomy had to be performed for relief from threatening suffocation. In order to obtain the full effect of mercury, it should be administered as long as there are no signs of intolerance. As to the daily dose of mercury, it is advisable to begin with a small quantity, say a half of a dram, twice a day (in an adult). At the end of three or four days the dose can be increased to a dram, if there is complete tolerance. After a sufficiently long trial (say two or three weeks) mercury should be substituted by iodides. Sodium iodide gives the same therapeutic results as potassium iodide and has this advantage over the latter, that it is less apt to disturb the stomach. Its initial dose should also be small, say 10 minims of a saturated solution three times a day. At first the dose should be increased five minims every three days until the dose of 50 minims is reached. Then the increase should be very slow: about one or two minims every other day. The drug can be continued indefinitely until intolerance shows itself. The latter is manifested by loss of appetite, digestive disturbances (pain, eructation) and diarrhœa. The question of intolerance is a relative one. I have had patients who have been unable to take more than five minims t. i. d. and patients that could take 500 minims t. i. d.

Arsenical preparations have been recently brought forward. The newly discovered Salvarsan by Ehrlich and Hatta, although a very valuable drug in primary and secondary stages of syphilis is practically valueless in tabes. It may in some cases improve the general nutrition but it has very little or no effect upon the course of the disease. This subject will be discussed fully in chapter on Cerebro-spinal syphilis. Cacodylate of sodium, another arsenical preparation, administered hypodermically has also a tendency to improve the patient's general health, but it has no effect on tabes itself.

Among other drugs used in treatment of tabes can be mentioned: chloride of gold, nitrate of silver and ergot. The latter has been recommended by Charcot to combat the genito-urinary disorder. The special symptoms of which the tabetics complain can be combated by usual medicaments. But there is one manifestation which requires special attention. This is **"gastric crises."** Because of the intensity of the pain, of frequent vomiting and the asthenia which the latter produces, gastric crises are the most distressing manifestations of tabes.

The crisis is the result of motor and secretory reflex phenomena

secondary to a hyperæsthesia of the gastric mucosa. The latter is controlled by the pneumogastric and especially by the sympathetic. These two nerves send filaments to the major splanchnic through the solar plexus and from there through the rami communicantes they reach the posterior roots of the sixth, seventh, eighth and ninth dorsal pairs. To decrease the irritability of these nerves local applications have been advised, such as ice, cauterization, spray of ethyl chloride over the region of the stomach; internally chloroform, opium, etc. They all may occasionally give temporary relief.

Surgical procedures have been more successful. Lumbar puncture with evacuation of 10 or 30 c.c. gave in some cases considerable relief, but still better results were obtained when the puncture was followed by some anesthetizing fluid such as cocain, eucain, novocain, stovain in the amount of 1 c.c. of 1:10 solution. Epidural injections have given similar results to König and others. None of these fluids could be considered absolutely safe, as accidents, such as paralyses or respiratory disturbances have been observed. Besides, not in all cases improvement has been noticed. A more satisfactory drug for intraspinal injection is magnesium sulphate. Its dose is 1 c.c. to each 25 pounds of body weight; the solution is 25 per cent. With this drug fewer accidents and more frequent improvements have been observed.

Recently operations of Foerster and Franke came to the front. Both have met with successes and failures. Both have for object to interrupt the sympathetic pathway of the gastric sensibility. Foerster attacks it at the level of the posterior roots of the sixth, seventh, eighth, ninth and tenth thoracic nerves. He cuts these roots on both sides. The operation is a complex one and the mortality is quite high. However, excellent results have been obtained in some cases. Recurrences of pain have been also observed.

The operation of Franke is decidedly less complex and attended by a lesser mortality, but the success is not as frequent as in Foerster's operation. Franke attacks the sympathetic pathway by tearing out the central ends of the intercostal nerves corresponding to the roots utilized in Foerster's operation. Both operations gave favorable and unfavorable results. As the latter is considerably less serious than the first, it may be tried at first, and when no results, Foerster's operation may be undertaken.

Sicard (Presse Médicale, 1912) has recently devised a new procedure which seemingly gave him very satisfactory results. He advises to penetrate after a laminectomy into the epidural space and without opening the dura tear out the spinal ganglia. It is therefore an extradural

ganglionectomy. It has the advantage of avoiding an injury to the dura and consequently there is no loss of cerebro-spinal fluid. The number of spinal ganglia to be removed on both sides as well as the number of roots to be taken into consideration for the operation are to be determined according to the seat of the painful phenomena.

Finally injections of alcohol or stovain into the point of emergency of the intercostal nerves (Koenig) have been tried. Exsner (*Wiener klin. Wchn.*, 1912) suggested a method of treating gastric crises of tabes by severing the vagus nerve on both sides. He found the nerve frequently diseased.

Stretching the solar plexus has also been recommended and favorable results have been reported by some surgeons. (Audibert. Thèse de Lyon, 1912.)

External Treatment of tabes has been applied in various forms, but the majority of them present only a historical interest. **Suspension,** for example, or forced flexion of the spinal column, also elongation of nerves, were imagined with the object of improving ataxia and of relieving pain. Massage is a good procedure for keeping up the nutrition of the muscles; so it is with electricity. Among all the mechanical means the **"systematic exercises"** of the ataxic limbs have proved to be most valuable. Frenkel, of Hayden, made a special study of them and brought them up to a regular method based upon the exact knowledge of physiologic functions of various muscles. It consists of slow exercises of various segments of the limbs. The patient is taught to sit down, get up, bring his limbs together, raise one limb at the time, walk in a certain direction, etc. By this procedure, kept up persistently for weeks and months, patients learn how to use their limbs and may become so skillful that they may dispense with their canes or crutches.

In treatment of tabes general hygienic measures should not be neglected. Stimulants should be avoided. Good nutritious food and fresh air are indispensable. Hydrotherapy in the form of a Scotch douche or baths is a good adjuvant to other procedures. Special care should be taken of the sphincters. In case of incontinence of urine the patient must be provided with a special urinal. In case of retention catheterization is necessary. In case of cystitis the bladder should be washed out with some mild antiseptic, such as boric acid, and urotropin administered internally. Bed-sores must be taken special care of. Infection may originate from this particular source as well as from the bladder, and hasten death.

Fatigue must be avoided, although mild outdoor exercises are advisable.

Anorexia or other dyspeptic symptoms must be promptly remedied,

as it is very important to have the patient to assimilate abundant and nutritious food. In recent years treatment with X-rays has been tried. Favorable results are being reported. It is claimed that the pain in the limbs and crises are particularly benefited. Incoördination may also improve. The exposure to the rays should last fifteen minutes and made at the level of the dorso-lumbar region. Twenty or thirty séances are necessary to obtain improvement.

Juvenile Tabes.—Tabes has been observed in very young individuals and even in children of nine years of age. Hereditary syphilis can always be traced in the family history. Wassermann reaction is positive in a much larger proportion than in adults. Atrophy of the optic nerve is according to Barkan (*Wien. klin. wchn.*, March, 1913) found in 80 per cent. of all the cases on record. The Romberg sign and ataxia are less frequent in children. Mercury does not arrest the progress of tabes. Only one case of juvenile tabes is on record with necropsy.

II. Spastic Paraplegia (Primary Lateral Sclerosis)

Pathology.—It consists of a primary degeneration and sclerosis of the pyramidal bundles in the cord. In the majority of cases recorded the lesion overstepped the boundary and some changes were found also in Goll's columns, occasionally also in the direct cerebellar tract. In a very few cases the lesion was strictly confined to the lateral columns. In my case (*New York M. J.*, 1912) only the pyramidal tract was involved; the lesion extended to the medulla at the level of decussation. The disease is rare.

Etiology.—Except the family form of spastic paralysis, which will soon be described, nothing definite is known in regard to the causes of the affection. As the disease is very rare, very few cases are on record. The disease probably belongs to the category of congenital abnormalities of development of the central nervous system. Syphilis may produce the picture of lateral sclerosis.

Symptoms.—The disease is characterized by a slow development of spasticity with loss of power in the lower extremities. The gait gradually becomes more and more difficult and the patient is unable to raise his limbs off the ground: his feet scrape the floor. Upon passive movements great resistance is felt. The limbs are rigid. The abnormal reflexes which are usually found whenever the motor tract is involved (see hemiplegia) are all present here, viz. increased knee-jerks and Achilles' tendon reflex, ankle-clonus, Babinski's phenomenon, Oppenheim's and paradoxical reflexes.

The sensations, the state of the bladder and rectum, the consistency and electrical reactions of the affected muscles are all normal.

Family Spastic Paralysis.—The spastic paraplegia just described may sometimes be found in several members of the same family. Strümpell was the first to call attention to this occurrence. In the majority of cases the male members are more frequently affected than the female. The disease usually appears between fifteen and thirty years of age. In one family that came under my observation there were seven members affected with spastic paralysis: four of them began to show the spasticity at the age of three; six of them were males and one a female. In families thus affected there is usually present some degenerative basis; either insanities, various neuroses or alcoholism, consanguinity, syphilis. The symptoms

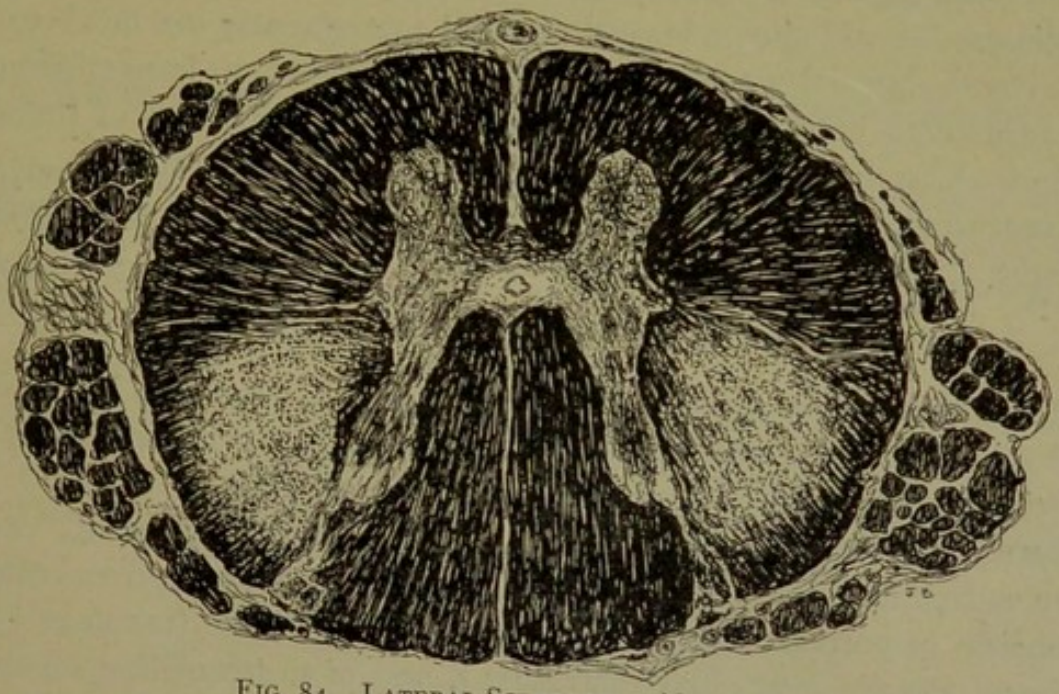


FIG. 84. LATERAL SCLEROSIS. (*Original.*)

are the same as described above. The feet are frequently deformed and may present all forms of deformation, most frequently equinovarus. The lower extremities are mainly involved. The upper ones are rarely affected.

In some cases this type of the disease remains unchanged until the end of the patient's life, but in the majority of cases, years after the onset, new symptoms develop and usually the symptomatology of multiple sclerosis becomes apparent. The involvement of upper extremities, the staccato speech, the intention tremor, nystagmus, optic atrophy—all are typical of the latter affection. It is interesting to note that the intelligence is invariably intact.

Closely allied to family spastic paralysis stands Little's disease, which

is a cerebro-spinal involvement of the pyramidal tracts, but the cerebral symptoms which are usually present from the very beginning make it a disease apart; they are: speech disturbances, strabismus, athetoid movements, epilepsy (see Cerebral Palsies of Childhood).

Prognosis.—In the pure type of spastic paraplegia (which is rare) and in the family type the prognosis is unfavorable. The disease is progressive. Complete paralysis with marked contractures is inevitable. Death is rarely the result of the malady itself, but mostly of some intercurrent disease, as infection or tuberculosis.

Diagnosis.—As spastic paralysis may be the ultimate consequence of myelitis, of amyotrophic lateral sclerosis, compression of the cord, syringomyelia, Friedreich's disease, multiple sclerosis, the diagnosis of spastic paraplegia as a primary affection should be made very cautiously. The disease, as said above, is very rare and consequently can be diagnosed only after the special symptoms of the other diseases have been eliminated. The occurrence of the spastic paralysis in several members of the same family is the most important characteristic feature of the disease.

Treatment.—It is the same as in Little's disease. See methods of Foerster, Schwab and Allison; also of Stoffel (pages 122 and 123).

PARAPLEGIA OF THE AGED

Paraplegia of the aged has its orgin in vascular sclerosis in the brain or in the spinal cord producing a general disturbance in circulation and nutrition of the central nervous system.

Paraplegia of cerebral origin is caused by "lacunar softening," a condition so frequently met with in senile brains (Marie, Ferrand and others). It consists of perivascular degeneration of brain tissue (see page 83) in the basal ganglia *irregularly* distributed, and of a descending secondary degeneration in the pyramidal tracts. The posterior columns of the cord are also irregularly degenerated. The symptoms begin insidiously without apoplectic features. They consist of a spastic feebleness in the lower extremities (the upper limbs are but very slightly involved) which gradually develops into a total spastic paraplegia with or without sphincter involvement. Finally dementia, bed-sores and dysarthria make their appearance. There is usually an inequality of the paretic phenomena on both sides. The contractures, if present, are never so marked and constant as in the following type.

In Paraplegia of spinal origin there is a disseminated and diffuse sclerosis in the posterior columns but especially in the lateral columns. The sclerosis is irregularly distributed at different levels of the cord. Clinically the paretic condition is symmetrically distributed in both

lower limbs. The onset is, like in the first variety, insidious. The evolution of the symptoms is longer. There is frequently pain which is continuous and occasionally paroxysmal. The sense of position is frequently perverted. When the patient walks, his legs are widely separated. There is an uncertainty in movements and sometimes the legs suddenly give way. The tendon reflexes are exaggerated. The spasticity gradually increases; contractures in flexion-position develop. Finally appear sphincter disturbances, general emaciation and tendon retraction. Bed-sores may precipitate the fatal termination. The preservation of the mental faculties is a distinguishing feature. The latter together with the slow course and the symmetrical distribution of the spastic and paralytic phenomena will enable one to differentiate it from the cerebral form.

III. Ataxic Paraplegia (Postero-Lateral or Combined Sclerosis)

Pathology.—The lesion consists of a degeneration and sclerosis of the posterior and lateral columns. In the posterior portion of the cord Goll's columns are mainly affected. In the lateral portion of the cord the crossed

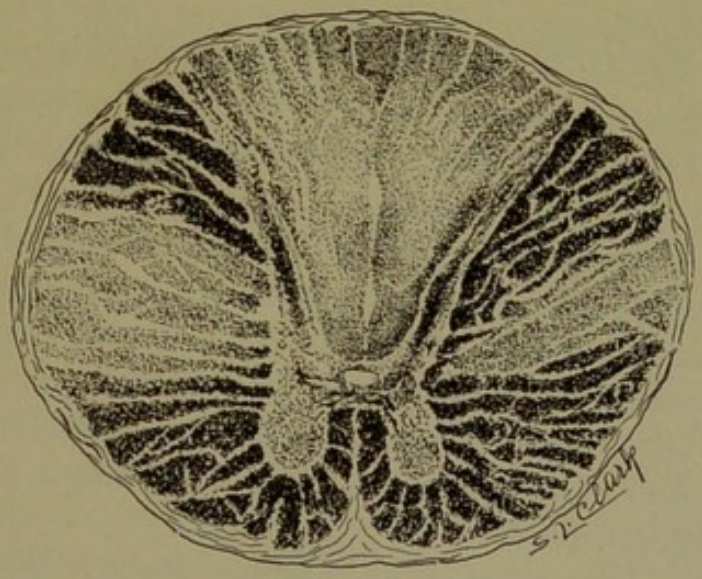


FIG. 85.—COMBINED SCLEROSIS. (*Original.*)

pyramidal tract is chiefly and always involved, but quite frequently the direct cerebellar tract and occasionally Gowers' bundle are also diseased. The neuroglia tissue is proliferated. Changes are also observed in the cells of Clarke's columns.

Symptoms.—From the pathological condition it can be seen that the symptoms of tabes and spastic paraplegia should be expected. When the posterior columns are more and earlier involved than the lateral, the

predominant symptoms will be those of tabes. In case the crossed pyramidal tract is mainly affected, the chief symptoms will be those of spastic paraplegia. In the majority of cases the tabetic symptoms are few in number and the following are the manifestations of the disease: ataxia, Romberg's sign, spasticity and weakness of the lower extremities, spastic gait, increased knee-jerks, ankle-clonus, Babinski's sign, Oppenheim's and the paradoxical reflexes. The sensations, if they are altered, present some objective changes. At first there is a slight diminution of sensations to all forms. Later in a fully developed case a more or less complete loss of sensations is observed. The anæsthesia is confined to the lower extremities and may reach the abdomen. Paræsthesia in the lower limbs, such as numbness, tingling and others, are an early symptom. They may persist during the entire course of the affection. The main tabetic symptoms: pain, bladder disturbance and eye changes are extremely rare. Loss of control of the bladder sphincter occurs only in the terminal period when bed-sores and poor general health indicate the approaching end.

In **Anemia** or **Pernicious Anemia** the clinical picture is somewhat different from the usual type of ataxic paraplegia. Sensory disturbances appear at the beginning; they consist of paræthesia, diminished sensations and sometimes lancinating pains. In my case (*New York M. J.*, 1909) there was a distinct syringomyelic sensory dissociation. Muscular weakness and paresis affect the upper extremities as well as the lower. The ataxia is more of a cerebellar type (see Cerebellum) than of a tabetic character.

In pernicious anemia degeneration is found not only in the posterior and lateral columns, but also in other portions of the cord. In my case (loc. cit.) the condition was diffuse and involved also Gower's tract.

In chronic intoxication with **Ergot** and in **Pellagra** degeneration of the posterior and lateral columns sometimes occurs. In ergotism—the symptoms are more of tabetic nature, while in pellagra more of the type of spastic paraplegia.

Course, Prognosis.—The outlook is unfavorable. The disease runs a rapid course: from a few months to two years.

Diagnosis.—When the tabetic symptoms are marked, the disease is differentiated from true tabes by the absence of the chief symptoms of the latter (Argyll-Robertson's pupil, optic atrophy, bladder disturbances, etc.) and by the presence of spasticity with the abnormal reflexes mentioned above.

From **Friedreich's disease**, which is also a postero-lateral sclerosis, it will be differentiated by the absence of the chief symptoms of the first,

viz. nystagmus, disturbed speech, choreiform movements, early onset and family character of the disease.

Multiple sclerosis will be recognized by intention tremor, staccato speech, nystagmus.

Transverse myelitis in its chronic form assumes frequently the form of spastic or ataxic paraplegia, but in the former the sensory and trophic disturbances, sphincter involvement and the evolution of the symptoms will easily reveal the true nature of the disease.

Etiology.—Syphilis may be the cause. The age at which the disease occurs is between twenty and forty. In the course of anemia, pernicious anemia, pellagra, ataxic paraplegia is sometimes observed. As the blood vessels are frequently found intact, the toxic element which is probably at work in pernicious anemia follows the route of the nerve fibers in the cord and produces a degeneration. In the absence of the above causes, a congenital weakness of the sensory and motor neurones of the cord is probably the true nature of the disease.

Treatment.—Antisyphilitic drugs (mercury and iodides, also salvarsan) should be always tried. In cases of anemia, appropriate remedies (iron, arsenic) should be administered. As to the ataxia and spasticity, they will be relieved by the same means as advised in tabes and spastic paraplegia.

IV. Friedreich's Disease (Hereditary Ataxia)

This affection was described first by Friedreich in 1861 as a peculiar variety of tabes occurring in childhood and having a hereditary character.

Pathology.—The spinal cord is diminished in size and very slender. The changes affect the white and the gray matter, but it is the first that is particularly affected. The most pronounced degeneration is in Goll's columns, which are involved through the entire cord. Burdach's tract is affected mainly in the lumbar region and only a small portion of it is sclerosed. Direct cerebellar and Gowers' tracts are invariably involved. The majority of observers believe that the crossed pyramidal bundle is also diseased, but considerably less than the posterior columns, and the degeneration commences later. The affection is therefore essentially a **combined sclerosis.**

As to the gray matter, the cells of the posterior cornua are diminished in number and size. Clarke's columns present the most changes: the cells are atrophied and the number of fibers is considerably reduced. The neuroglia tissue which takes the place of the normal substance is proliferated and more developed in the dorsal than in the lateral columns.

The meninges are sometimes found thickened at the level of the posterior columns. The dorsal roots are also in a state of sclerosis.

Usually the changes just described are confined to the cord, but in a few cases the medulla and cerebellum were found also altered. The involvement of the latter organ led Marie to describe a separate type under the name of heredo-cerebellar ataxia (see the latter). The peripheral nerves have also been found atrophic or degenerated.

Etiology.—The fundamental characteristics of Friedreich's disease from the etiological standpoint is that it occurs in several members of the same family. The symptoms begin to appear before the age of fourteen. Edinger has proposed the following explanation (Ersatz-Theorie). When portions of the nervous system are not developed or not resistant, overwork may lead to their degeneration. Thus the beginning of the symptoms at age of puberty in Friedreich's disease finds its reason.

Cerebral diseases have been observed in relatives. Syphilis, alcohol, do not play any special rôle. Males are somewhat more frequently affected than females.

Symptoms. Motor.—The first symptom to appear is disturbance of gait. Slight in the beginning, it gradually becomes more and more marked. The patient walks with his legs widely separated, and after he raises them he drops them heavily on the ground. At the same time the entire body oscillates for fear of falling. This **ataxia** is not tabetic, but of **cerebellar type**. The **station** is also ataxic. While Romberg's sign is not present, nevertheless the patient's body oscillates to and fro, his feet change position very often, when he is told to stand still. The upper extremities are also affected, but here the ataxia appears later, long after the legs become affected. **Choreiform** movements are frequent: the gesticulations are present not only in the arms, but also in the head and trunk. As the patient sits unsupported, nodding movements of the head and swaying of the trunk are seen. **Athetoid** movements may also occur. The limbs are very weak and as a rule flaccid but there are no paralytic symptoms. **Intention tremor** is quite frequent. The **reflexes** are changed: the knee-jerks are usually abolished, but Babinski's sign is present. In some cases the knee-jerk is preserved until late in the course of the disease. The abdominal reflex gradually disappears. The tendon reflexes of the arms disappear later than those of the legs.

Sensory.—General sensations are usually not affected, except perhaps in the last stage, when anæsthesia appears. Egger observed in his cases a diminution or loss of vibratory sense of tuning-fork. The special senses are not involved. The **eyes,** however, present one special symptom which is very frequent in Friedreich's disease, viz. **nystagmus.** It consists of brief and repeated movements of the eye-globes when the

patient is told to turn his eyes to the right and to the left, without moving his head. The ocular movements are intact.

Cerebral.—**Vertigo** is frequent. **Intelligence** is normal, except towards the end, when dullness and childishness make their appearance. The **speech** is almost always affected: it is slow, the articulation is difficult, irregular; some words are pronounced abruptly, some with deliberation. During conversation the muscles of the face are affected with irregular contractions the movements of the face are ataxic.

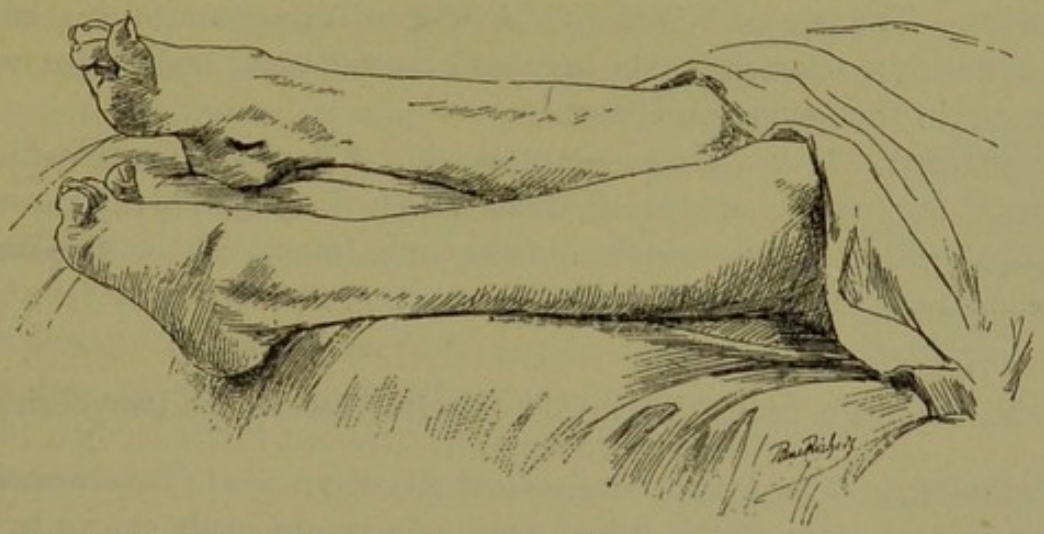


FIG. 86.—CHARACTERISTIC FEET IN FRIEDREICH'S ATAXIA. (*Bouchard and Brissaud.*)

Trophic.—There is a special deformity of the foot which is characteristic of the disease. It is of the **equinus** type: the foot is short, as if it was pressed antero-posteriorly; the plantar surface is concave, while the dorsal is prominent; the toes are "claw-like" because of forced extension. This condition is usually **bilateral.** In a few cases the same deformity was observed in the hands, which also assume the claw-like position (main en griffe). **Scoliosis,** or more frequently kypho-scoliosis, is observed in an advanced stage; it is particularly marked in the dorsal region. The sphincters are as a rule intact.

Course and Prognosis.—The disease is essentially progressive, but slow in the development. The earliest symptoms are: ataxia and hyperextension of the great toe. The upper extremities become involved after the lower. The eye symptom and the peculiar speech appear still later. Within a period of five years the clinical picture is complete. The patient is then bedridden and loses all power in his limbs. Remissions have been observed, but any intercurrent acute disease hastens the course of the malady. It may last an indefinite number of years, but the outlook is invariably grave. Death usually occurs from some intercurrent disease.

Diagnosis.—With **tabes** Friedreich's disease may be confounded

because of the ataxia and the loss of knee-jerks. In the latter affection there is no true tabetic incoördination (see Tabes), but a cerebellar titubation. Moreover the presence of choreiform movements, nystagmus, disturbance of speech, the absence of sensory disturbances, of sphincter involvement, of pupillary changes, will enable one to make the diagnosis of Friedrich's disease.

In **Multiple Sclerosis** there are nystagmus and intention tremor, but in Friedreich's disease the nystagmiform movements of the eye-globes are only in transverse direction, while in multiple sclerosis they are in all directions. The tremor in Friedreich's disease is considerably less marked than in multiple sclerosis. The knee-jerks in the latter disease are usually increased, in the former abolished.

Treatment.—It is purely symptomatic. There are no remedies for arresting the course of the disease, which is inevitably progressive. Massage and systematic exercises of the limbs (motor reëducation) may be of benefit (see Treatment of Tabes).

V. Infantile Spinal Paralysis (Acute Anterior Poliomyelitis)

Pathology.—The lesion consists chiefly of an acute inflammation of the anterior cornua of the spinal cord. In recent cases the microscope shows dilatation of the blood vessels, leucocytic infiltration of their walls (arteritis); the ganglionic cells surrounded by this inflammatory tissue undergo degeneration and atrophy: their protoplasma (Nissl's bodies) becomes disintegrated, the nucleus and nucleolus fall out and the prolongations (dendrites and axis cylinders), whose existence depends upon the integrity of the cells, undergo atrophy. The morbid process therefore consists of inflammatory softening. The latter may be found in small foci or occupy a large portion of the cornua. The destroyed tissue is gradually substituted by proliferated neuroglia, so that when the cord is examined, several years after the acute onset, one can see cicatricial tissue in the substance of the anterior cornua, and in the midst of it deformed, dilated and thickened blood vessels. The entire cornu becomes diminished in size, and if the other is intact, the contrast between the two is striking. The anterior roots, which are the prolongation of the axis-cylinders emanating from the cells, undergo atrophy. The motor fibers of the body which are the continuation of these roots and the muscles supplied by the nerves of the anterior roots also suffer in their nutrition: the muscular fibers undergo granular degeneration and are replaced by fibrous or adipose tissues. The bones of the affected limbs participate in the general and progressive atrophy, so that an entire

limb may be arrested in its development and remain very small, if the disease occurred in infancy and was neglected.

Etiology and Pathogenesis.—All the evidences are in favor of an **infectious** cause of the disease. The onset of the disease and the occasional epidemics occurring in certain localities leave no doubt as to its infectious nature. On two occasions within a period of two years I observed a series of eight cases and of twelve cases in one section of this city. According to Marie, an embolus of infectious nature reaches the anterior cornua through a branch of the anterior spinal artery. The most recent experimental investigations of Levaditi, Landsteiner, Popper, Flexner, Lewis and others have established the fact that the upper respiratory passages and the intestinal tract are the usual seat of the virus. Expectoration and the stools of an infected individual are the sources of contamination (Kling, Weinstedt and Pettersson). Flexner, Clark and Frazer have demonstrated (*Jour. Amer. Med. Assn.*, 1913) the occurrence of the virus in the naso-pharynx of healthy persons who have been in close contact with an acute case of poliomyelitis. Contagion by dust, clothing and shoes has also been established. Insects, such as flies, have been shown to carry the virus and transmit it. The original idea of Wickman as to the fact that the disease is contagious is now completely confirmed. The existence of epidemic foci may pass unobserved in large cities. The season of the year appears to be an important predisposing cause of poliomyelitis. Summer and early autumn are the most favorite seasons. It seems that a certain immunity is acquired by a country in which an epidemic attack once occurred. Thus in Sweden the provinces affected severely in 1905 were apparently free from the disease in 1911. The immunity produced by an abortive attack will probably explain the resistance of older children and adults.

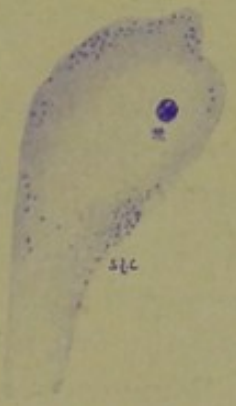


FIG. 87.—DEGENERATION OF A CELL OF THE ANTERIOR CORNU IN A CASE OF ACUTE ANTERIOR POLIOMYELITIS. (*Original.*)

As to the nature of the virus, it is as yet impossible to determine it, as the microörganism has not yet been discovered. It has been shown by Flexner, Lewis and Levaditi that the unknown microbe belongs to the class of very small microorganisms as it is able to pass through the filter, a fact which is not observed with the majority of well defined microörganisms. Finally Rosenau has recently demonstrated that the barn-fly (stomoxys calcitrans) is a possible disseminator of the poliomyelitic infection. Through this agency he succeeded in transmitting

poliomyelitis from monkey to monkey (*J. Am. Med. Ass.*, 1912). Anderson and Frost were able to confirm it; they also succeeded in transmitting the infection to a fresh monkey with the emulsion of the cord of an animal infected by the flies.

Infantile paralysis is a disease of early childhood. It should be borne in mind that it may also occur in adult life, but this is rare. The boys are more frepuently affected than girls. Among predisposing etiological factors **trauma** may be mentioned. I have seen a number of cases in which the onset dates from a fall. Starr mentions cases of poliomyelitis which developed immediately after exposure to cold, such as swimming in very cold water.

Symptoms.—The onset is always sudden and resembles that of an acute infectious disease. The incubation period usually lasts from 1 or 2 days to 10 days. Fever, general malaise, anorexia, vomiting, diarrhœa or else coryza, angina, bronchitis and sometimes convulsions are the usual initial symptoms. In some cases only one or two of them are present and in others they are so mild that they may be overlooked. In still others none of the premonitory symptoms is observed. The child suddenly becomes paralyzed.

Some cases begin with symptoms of **meningitis:** rigidity of the neck and trunk, somnolence, agitation, headache, convulsive movements of the eyes, tenderness of the spine, general hyperæsthesia, articular pain. Exanthemata or erythemata are not infrequently seen. Children frequently complain of pain in the back or in the affected limbs. In a day or two and sometimes later the principal symptom, viz. **paralysis,** makes its appearance, and it progresses so rapidly that it reaches its climax in twenty-four hours. It affects one limb, two symmetrical limbs (usually the lower extremities) or an arm and leg on the same side, or one arm; it may also involve an arm on one side and a leg on the other. In some cases all the four extremities may become paralyzed. In a few days the paralysis retrocedes: motility returns in certain groups of muscles, so that at the end of a few weeks the paralysis becomes fixed in one or two extremities. When the lower limbs are paralyzed, there may be: a *thigh type* and a *leg type*. In the former the muscles on the inner side of the thigh escape; the iliacus, glutei and the antero-external muscles of the thigh are affected. In the leg type the peronei and anterior tibial are mostly paralyzed; the posterior tibial less frequently. When the upper limbs are involved, there may be also two types: upper and lower arm types. In the first the scapular muscles, deltoid, biceps and supinator longus are paralyzed. In the second the muscles below the elbow except the supinator longus are affected. Finally an entire limb may be involved.

The most charcteristic feature of the paralysis is the **flaccidity.** The **tendon reflexes** of the affected limbs are either totally abolished or greatly diminished. This condition is the general rule. The following varieties have been observed: (1) exaggeration of both knee-jerks; (2) loss of one knee-jerk, and exaggeration of the other; (3) a transient exaggeration immediately preceding their abolition.

The paralyzed muscles soon begin to degenerate and **atrophy** and their contractility gradually decreases. Their electrical irritability suffers a radical change: under faradism there is at first a diminished response and later complete loss of response. Under galvanism there is not only a quantitative, but also a qualitative alteration: **reaction of degeneration** appears quite early. Later all contractility to electrical stimulation is lost.

FIG. 88.—INFANTILE SPINAL PARALYSIS, SHOWING ATROPHY OF RIGHT LOWER EXTREMITY, AND SCOLIOSIS.

Parallel with the increasing atrophy, the growth of the muscles is arrested, contractures are formed because of the predominance of the antagonistic muscles, and all these factors lead to **deformities.** Among the latter the most common is pes equinovarus. Scoliosis, lordosis, are also met with. Finally the hands may also occasionally be affected: a claw-like hand is the most usual deformity. Deformities of joints is a frequent consequence of poliomyelitis: paralysis of the muscles surrounding the joints leads to a relaxation of the latter, so that the heads of the bones fall from their sockets. In a certain number of cases the bony tissue of the limb may be arrested in its development and then one can see an adult with one or two extremely thin limbs. The **skin** covering such a limb is very thin, ulcerates after the slightest erosion, is always cold, cyanosed. The **sensations** and the **sphincters** are intact. In some cases I observed a marked tenderness in the paralyzed limbs (Figs. 88, 89, 90).

Course, Termination, Prognosis.—The clinical picture just described may vary considerably. Thus the paralysis may be only transitory and last only a few days or else two to three weeks. In such cases the atrophy

will never develop. In other cases the disease spreads and involves the nuclei of the medulla, and if the ninth and tenth nerves are involved, the patient dies from bulbar symptoms.

The typical cases when properly and early enough treated may regain considerable of the lost power in the limbs, but they never recover completely. The majority of cases remain with some infirmity: either the pes equinus persists or the atrophy with some impairment of locomotion becomes permanent. In some cases, many years after the first attack, the patient is taken with another in some other limb, which then runs the

FIG. 89.—INFANTILE SPINAL PARALYSIS SHOWING DEFORMITIES OF THE LOWER LIMBS.

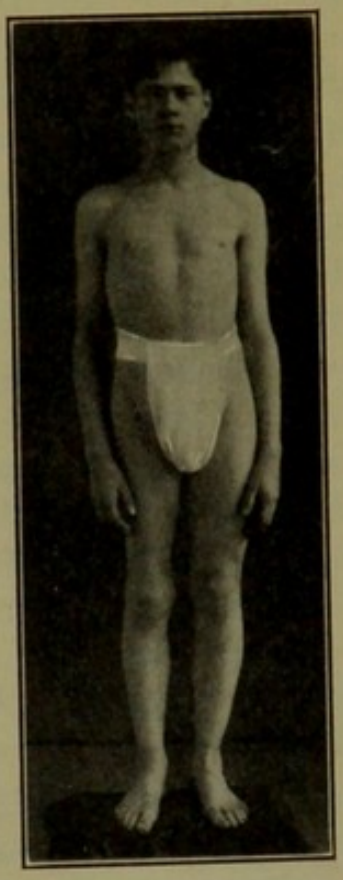


FIG. 90.—ATROPHY OF RIGHT THIGH. SEQUELA OF ACUTE ANTERIOR POLIOMYELITIS IN CHILDHOOD.

same course as the old spinal palsy. In a case that came under my observation (*Amer. Med.*, 1903) symptoms of amyotrophic lateral sclerosis began to develop á propos of an injury years after the infantile spinal paralysis.

Generally speaking, the prognosis depends a great deal upon the electrical reactions of the muscles. Reaction of degeneration is usually an unfavorable sign for recovery of the paralyzed muscles. Loss of faradic contractility is not always an indication that the muscular contractility will not return. I have seen cases of return of this form of

electrical reaction long after the onset of poliomyelitis. As to life, the outlook is good except when the medulla is involved.

Diagnosis.—The sudden onset with its characteristic prodromal symptoms, the flaccid palsy, the loss of reflexes, the atrophy, are all typical enough for recognition of the disease. Nevertheless there is a certain group of affections with which acute poliomyelitis may be confounded. There is a form of poliomyelitis in which the most conspicuous symptom is pain along the spine and in the limbs. The pain is considered as being due to **spinal meningitis.** In some cases the onset is so tumultuous that epidemic **cerebro-spinal meningitis** is thought of. Pain in the head, rigidity of the neck, pain along the spine, Kernig's sign and even disturbance of consciousness may be present. The **differential diagnosis** will be as follows: Meningeal symptoms of poliomyelitis disappear rapidly and they are distinctly spinal. In cerebro-spinal meningitis they are both cerebral and spinal. In the first the paralysis is paraplegic monoplegic or hemiplegic and flaccid in type. In the second the paralysis is spastic and cranial nerves are frequently involved. The reflexes are diminished or lost in the first, but increased in the second. The toe phenomenon is absent in the first, present in the second. Lymphocytosis in the cerebro-spinal fluid is characteristic of the first, leucocytosis of the second. In the first the fluid is clear, in the second cloudy. In the first there are very few polymorphonuclear cells, in the second—abundant. No microörganism is found in the cerebro-spinal fluid of poliomyelitis, meningococci are found in that of cerebro-spinal meningitis.

In **serous spinal meningitis** in which pain and paralysis also lymphocytosis are present, the type of paralysis and the condition of reflexes are not the same as in poliomyelitis.

In **Multiple Neuritis** are present: pain in the limbs, paralysis and altered reflexes like in poliomyelitis. The points of difference are: in the second there are general manifestations indicative of an acute infection; they are absent in the first. In the second paralysis extends further than in the extremities, the muscles of the trunk are involved. In the first the trunk is intact. In the second the palsy is irregular and more marked in the muscles of the hip and thigh. In the first the paralysis is regular and more marked in the most peripheral muscles of the limbs. Atrophy sets in much earlier in multiple neuritis than in poliomyelitis. Objective sensations are rarely disturbed in poliomyelitis; in polyneuritis hyperæsthesia is common and pain, burning, etc., are frequently complained of in the fingers and toes. Polyneuritis is frequently cured without leaving any trace, but this is not the case with poliomyelitis, in which deformities of the limbs are the rule.

Acute Transverse Myelitis is exceptional in children. Besides, involvement of the sphincters is common. In poliomyelitis the sphincters are not involved.

Birth or Obstetrical Palsy follows a difficult labor or instrumental delivery; there are always sensory disturbances. The atrophy and objective sensory disturbances followed a radicular distribution.

Cerebral Infantile Hemiplegia will be recognized by the spastic paralysis and the abnormal reflexes usually found in hemiplegia (see this chapter).

In **Hysterical Paralysis** the reflexes are preserved and the electrical reactions are normal.

The foregoing study permits the following grouping of varieties of acute poliomyelitis:

(1) **The Spinal Type.**—It is the most frequently observed.

(2) **The meningeal type,** in which the onset presents meningeal symptoms (see above). Generally in a few days they clear up and the picture of poliomyelitis becomes evident.

(3) **The polyneuritic type,** in which there is hyperæthesia and pain in the affected limbs (see above).

(4) **The bulbar type,** in which the nuclei in the medulla become involved in the course of poliomyelitis: the prognosis here is grave.

(5) **The abortive type,** in which the symptoms are very mild, last but a short time and totally disappear.

Treatment.—At the onset of the disease the child should be kept in bed and every effort made to reduce the temperature with cold baths or spongings and coal-tar products (antipyrin, phenacetin). Cauterization and revulsion in the form of cupping or mild counter-irritation with mustard plasters along the spine may be of benefit. Urotropin taken by the mouth sets free formaldehyde, and the latter is then found in the cerebro-spinal fluid. It acts therefore as an antiseptic. It may therefore be useful. A child of three can be given 1–2 gr. two or three times a day. Pain can be combated by sedatives such as bromides. Purgatives should never be neglected. The naso-pharynx should be taken special care of (see Etiology), and treated with antiseptic irrigations or applications. Ergot has been advised internally. After the acute stage has subsided, an effort is to be made to improve the general nutrition by a substantial diet and hydrotherapy. Iodides can then be administered; cod-liver oil, arsenic, hypophosphites, are useful. **Electrical** treatment and **massage** must be instituted as promptly as possible. It is advisable at first to apply the galvanic current with the positive pole on the affected muscles. When improvement is noticeable, the faradic current will be more useful. Daily applica-

tions of electricity for ten or fifteen minutes, aided by massage of the limbs and kept up persistently will render great service.

When improvement is commencing to be noticeable, the patient is taught to exercise the affected muscles voluntarily. Such efforts on the patient's part are of great benefit. As to the duration of the electrical treatment and massage, it should be kept up indefinitely. I have seen cases which began to show signs of improvement at the end of one to two and even three years.

To guard against contractures systematic exercises and gymnastics are advisable. When contractures and deformities are present, orthopedic applicances **may** be of some benefit. The use of mechanical apparatuses should not begin too early, as the weight of them will impose too much work on the muscles which commence to improve and thus overfatigue them. I have seen bad results from too early application of braces. On the other hand a properly applied apparatus may prevent a tendency to deformity. The time of using braces must be individualized in each case; no set rule can be given for all cases. If after a sufficiently long trial the orthopedic appliances fail, surgical intervention should be resorted to. In certain cases **tenotomy** of shortened tendons will place the deformed limb in a straight position, provided there is enough muscle left to be relied upon. Recently attempts have been made to connect muscles which are intact with the atrophied muscles. A healthy tendon or muscle is divided and sewed to the atrophied tendon or muscle, respectively.

I have seen some very good results from such procedures. Instead of anastomosing muscles, healthy nerves have been divided and their central ends united with the peripheral ends of divided nerves in the atrophied muscles. Some good results have been reported.

Recently Schwab and Allison, also Stoffel, devised methods to combat contractures and deformities. (See pages 122 and 123.)

Prophylaxy of Acute Poliomyelitis.—The above described results obtained from experimental investigations (see Etiology) gives us a clear idea of what preventive measures should be. It is necessary also to emphasize the fact that the virus may resist a very long time. For example, the virus mixed with sterile milk or water kept at temperature of the room maintains its pathogenic activity during thirty-one days (Landsteiner and Levaditi). Salivary and nasal secretions are carriers of poliomyelitic virus and are chiefly the sources of contagion. As desiccation does not destroy the activity of the virus, contagion may take place through dried up secretions. Rigorous antiseptic measures applied to the nose and throat in cases of epidemics, or when in contact with individuals affected with the disease, are urgent. Isolation is equally urgent.

VI. Chronic Anterior Poliomyelitis and Amyotrophic Lateral Sclerosis

They belong to the systemic diseases of the spinal cord and are described in the chapter on Muscular Atrophies.

B. NON-SYSTEMIC DISEASES OF THE SPINAL CORD

I. Myelitis

A. Acute Myelitis

Acute myelitis is an acute inflammation of the spinal cord secondary to an infection or intoxication.

Pathology.—The lesion is essentially diffuse. It is irregular in distribution. When several foci are present, the disease is a **disseminate myelitis;** when one focus extends upward or downward, it presents a **diffuse myelitis.** When the entire transverse section of the cord is involved, the disease is a **transverse myelitis.** According to the seat of the lesion, myelitis may be: **cervical, dorsal, lumbar.**

Irrespective of the localization of the focus, the pathological anatomy in all varieties of myelitis is the same. The cord is usually congested, œdematous, and the diseased portions are soft. The softening undergoes three distinct phases: at first the stage of red softening, later that of white softening and still later the stage of sclerosis.

In the first period (red softening) the blood vessels are dilated and their walls are infiltrated with leucocytes. The cells of the gray matter undergo disintegration, and the axis-cylinders are swollen and varicose.

In the second period (white softening) **necrosis** takes place: the entire cord tissue is very soft and appears white; the cells are colorless and deformed, without nuclei and prolongations. The nerve fibers lose their myelin sheaths, remain naked and unsupported. Ascending and descending degeneration in the white columns becomes evident. Instead of the normal nerve tissue there is a large number of granular cells.

In the third period there is formation of cicatrices at the expense of the neuroglia tissue, which at that time proliferates in abundance: instead of being soft the cord becomes hard. As death may occur during any one of these stages, the histological picture will vary according to when the pathological process was arrested in its development.

The meninges usually participate in the myelitic process. They are thickened (especially the pia-mater) and adhere to the cord. If the cells of the anterior cornua are extensively involved, the anterior roots and the peripheral nerves undergo alterations and the muscles supplied by these nerves atrophy. With reference to the pathology of

myelitis it is interesting to say a few words of Buzzard's conception. He believes that myelitis is the result of a thrombotic softening of the cord and that the usual causes of myelitis produce an inflammatory process in the vessel wall which permits the formation of thrombi, resulting in softening of the cord. The special nature of distribution of the blood vessels in the spinal cord is such as to directly favor the occurrence of thrombosis, especially in the lumbar region where myelitis is so frequently met with. This peculiarity consists of a great length of the small arteries, especially of those supplying the lower portion of the cord. Acute myelitis is therefore, according to Buzzard, a thrombotic softening.

Etiology.—Acute myelitis is always the result of an **infection** or **intoxication.** Cold, traumatism, emotions which formerly were considered as direct causes, are in reality only predisposing factors. In favor of this view speak the occurrence of myelitis in the course of acute and chronic infectious diseases, also experimental investigations. Influenza, typhoid fever, small-pox, scarlet fever, pneumonia, gonorrhœa, whooping cough, measles, tuberculosis and syphilis are all diseases which may become complicated by myelitis. A neighboring inflammatory process in the meninges or vertebræ may extend to the cord and develop myelitis.

During **puerperium** myelitis has been observed. In all these various infections of a general or local nature, either the microörganism itself or its toxin, and perhaps both, are the immediate causes of myelitis: they penetrate the cord through the blood vessels. Experimentally it has been proven beyond any doubt, I believe, that myelitis can be produced by inoculations of various microbes and of their toxins (Roux, Yersin, Grancher, Manfreda, etc.).

Symptoms.—In the chapter on "Pathology" various forms of myelitis have been mentioned. The most frequent of all is

Transverse Myelitis.—The usual seat of this form is the dorso-lumbar region, but it may also occur in the cervical and dorsal regions. The onset in the majority of cases preceded by prodromal symptoms. They are: pain in the back, paræsthetic disturbances (tingling, numbness, etc.), retention of urine, also some weakness in the extremities. The pain is not confined to the back, but it also radiates toward the limbs. The prodromal period usually lasts from a few hours to several days. After myelitis is established, the following symptoms become characteristic.

Motor.—In the dorso-lumbar form the legs present a complete loss of power. If the myelitis is cervical, the arms are paralyzed. The paralysis is flaccid in transverse myelitis of the dorso-lumbar cord, spastic in dorsal and cervical myelitis. The patellar tendon reflexes are abolished in the first, increased in the other two forms. Babinski's sign and paradoxical

reflex may be present in all the three forms. Curiously enough there is an antagonism between the latter two reflexes. At the beginning Babinski's phenomenon is quite rare, but the paradoxical very frequent. Later on, when contractures appear, the paradoxical becomes less marked, but Babinski's reflex more pronounced. Oppenheim's reflex may sometimes be present. Ankle-clonus may and may not be present.

As the disease gradually advances and passes into a chronic state, contractures develop. In the cervical form of myelitis the paralysis may affect not only the arms, but also muscles of the neck, the intercostal and abdominal muscles, the lower extremities, and the diaphragm, and thus cause difficult breathing. In myelitis of the eighth cervical segment there is also paralysis of the small muscles of the hands and of the flexors of the fingers.

Sensory.—They are constant. The patients complain of violent and continuous pain in the back, which in the cervical form radiates to the arms and in lumbar form to the legs. The tabetic **girdle pain** is present here. Sensations of cold or heat, tingling, burning, etc., are also very frequent. Objective sensory disturbances are quite marked. The sense of touch, pain and temperature in the upper and lower extremities and on the body (according to the seat of the lesion) is greatly diminished and sometimes completely lost in the areas innervated by the diseased segments and below these segments. In some cases there is a dissociation of the three forms of sensations (see Syringomyelia).

Trophic disturbances are usual. The affected extremities are cyanosed and œdematous. When a portion of the body undergoes pressure, ulceration soon develops. This is particularly frequent in the sacral region, but it occurs also on the gluteal areas and at the level of the trochanters. The decubitus is only present in the dorsal and dorso-lumbar varieties of transverse myelitis. The ulceration is usually superficial, but it may be so deep as to expose the bone. In a case that recently came under my observation, there was at first a cervical myelitis, but later the lesion spread in a descending manner. The bedsores were so marked that over the entire lumbar region the cutaneous and muscular tissues were totally destroyed and the largest part of the lumbar vertebræ and of the sacrum were exposed. In the same patient the prepuce was totally gangrenous and a portion of the dorsum of the penis was ulcerated. Muscular atrophy of the lower extremities is rare, but frequent in the upper limbs in myelitis of the lower cervical cord, absent when the upper cervical segment of the cord is involved. If atrophy occurs, reactions of degeneration (CaCC > AnCC) are present. In myelitis with involvement of the eighth cervical

segment there will be, besides the above mentioned paralysis and atrophy, also small pupils and narrow palpebral fissure.

Sphincters are always involved in the dorsal and dorso-lumbar myelitis. At the beginning there is retention. Later incontinence develops. Both sphincters are almost equally involved. Cystitis is a frequent complication. Impotence frequently exists in myelitis of any segment of the cord.

The clinical pictures of myelitis at various levels of the cord as just given are not always clear-cut. They may vary from one case to another. It depends upon the extent of the transverse section. If, for example, in the cervical form the lesion is sufficient to cut off all impulses passing through the diseased segment, the paralysis of the body below the lesion will be flaccid, all reflexes and sensations are lost. So it is with any other segment of the cord. If the lesion affects only one side of the cord, Brown-Séquard's form of paralysis will be present.

Syphilitic myelitis, which is the most frequent of all form, presents certain special features. It will be discussed in detail in the chapter on Syphilis of the Nervous System.

Tubercular myelitis may occur: (1) as a complication of cerebro-spinal tubercular meningitis (tubercular meningo-myelitis), (2) as a complication in Pott's disease (either as a compression myelitis or a secondary myelitis following a caseous pachymeningitis); (3) it may also occur in the course of Pott's disease as primary parenchymatous myelitic foci which develop without meningitis, without vascular and interstitial lesions, without caseation and very probably due to tuberculine circulating in the cord. This form of myelitis is analogous to lesions found in the liver and kidneys of tubercular individuals. Philippe and Cestan have reported such a form of myelitis (*Rev. Neurol.*, 1899).

Course, Termination, Prognosis.—While in the majority of cases the complete picture of acute myelitis develops within a period of from a few days to a few weeks, there are nevertheless cases with a sudden onset and rapid death. Usually the disease is progressive and becomes chronic. Great improvement and even complete recovery are possible, especially in cases that follow an acute infectious disease. In such cases the improvement in sensations precedes that in the motor power. The paraplegia is usually spastic. The improvement may be maintained for many years and the patient may resume his usual occupation and be only slightly disturbed by the sphincters. The greatest amount of disturbance occurs in myelitis of the lowest segment of the cord. Here the atrophy of the muscles and the condition of the sphincters are exceedingly obstinate. In some cases the disease acquires an ascending or de-

scending course. In the first case it may reach the medulla and then life is greatly threatened. In other cases the decubitus and cystitis, which may become purulent, are capable of killing the patient. Finally intercurrent diseases are a source of great danger to myelitic patients. Remissions in the course of myelitis are possible. The patient apparently improves to a great extent, and under the influence of an insignificant cause may suffer a relapse. The outlook in acute myelitis is always doubtful.

Diagnosis.—**Multiple neuritis** is sometimes confounded with myelitis. Tenderness of the nerve trunks, the predominance of the sensory disturbances, integrity of the sphincters, irregular involvement of the motor apparatus and especial involvement of extensor groups of muscles—all these symptoms are characteristic of multiple neuritis. Besides, in myelitis the objective sensory disorder is of segmental type, and more profound. In neuritis the limits of the sensory disturbances are not well defined, the latter are present only at the periphery, and, what is important, the tenderness of the nerve trunks is associated with anæsthesia or analgesia of the skin.

Hysterical paraplegia may simulate acute myelitis. In the former there is usually a history of a shock or of a great emotion, of a traumatism. The onset is sudden. The reflexes and the sphincters are intact. There are no trophic disturbances. Finally the existence of hysterical stigmata will aid in making the diagnosis.

Hematomyelia presents a very sudden onset besides the history of trauma.

Spinal compression presents usually a gradually oncoming paralysis, but when the paralysis is sudden or rapid like in transverse myelitis, it is usually caused either by caries or malignant disease of the vertebræ. Besides, the pain in compression myelitis is unusually intense and agonizing.

Treatment.—In the first stage of the disease the patient should be given absolute rest. A purgative should be administered. Pain will be controlled by salicylates and coal-tar products. In case of retention of urine catheterization is necessary. Local applications to the spine, as vesicants or cauterization, have been recommended, but in view of the sensory disturbances which are constantly present in myelitis, and the facility with which ulcerations are formed, it is advisable to avoid local irritation of the skin. Inunctions of mercury and internal administration of iodids should be instituted as soon as possible not only in syphilitic cases, but also when no history of direct infection could be obtained. (For the mode of administration and dosage, see Tabes.)

In addition to the drug treatment hygienic measures are of great im-

portance. Special and prompt care must be taken of the **bladder** and of the **decubitus,** as complications not infrequently come from these two sources and death may ensue. Purulent cystis is often the result of infection from repeated catheterization. Decubitus is also infectious in nature. It is therefore essential to see that urine and feces should not come in contact with the skin, that aseptic and antiseptic precautions be taken in the strictest possible manner. In cases of retention of urine, each aseptic catheterization must be followed by a washing out of the bladder with some mild antiseptic solution, as boric acid. By doing so, cystitis with all its grave consequences may be avoided. In order to avoid undue pressure of the skin, as the latter is usually the immediate cause of decubitus, the patient's position in bed should be frequently changed and the bed cloth kept smooth.

After the acute stage has subsided, massage becomes indicated, but in this latter procedure great care must be exercised that the skin is not injured. In a patient under my observation a small erosion of the skin of one leg caused by a masseuse became ulcerated and during three and a half months the ulceration remained practically unaffected by various medications. Electricity is a valuable adjuvant to massage. It should be avoided in cases of spasticity, but it may be useful in flaccid paralysis. Both procedures are important in treatment of atrophy. Galvanism at first and faradism later or both alternately may be employed.

Hydrotherapy in the form of sponging, or ablutions followed by gentle rubbing, is a good adjuvant in treatment of myelitis.

Spasticity of the extremities can be relieved by passive movements and warm baths.

Acute Diffuse Myelitis

This is a rare disease. Pathologically it consists of a myelitic focus having an ascending (more frequent) or descending course. It is known under the name of acute ascending or Landry's paralysis. It will be described later together with multiple neuritis.

Acute Disseminated Myelitis

This affection consists pathologically of myelitic foci disseminated throughout the central nervous system. Its clinical picture is that of disseminated sclerosis. The latter will be described later.

B. Chronic Myelitis

Chronic myelitis as a **primary** affection occurs in traumatism, in compression of the cord, in infections of long standing (syphilis, leprosy), in

intoxications of long standing (diabetes, pernicious anemia, ergotism). The lesion and the symptoms are slow and long in development. They will be described in separate chapters.

As a **secondary** affection it follows acute myelitis. It is only with this form that we will be concerned here.

Pathology.—If a myelitic cord is examined years after the acute onset, it will be found shrunken and small in size. A transverse cut will show that the cord is discolored and grayish in appearance. Histologically it presents a destruction of cells and disappearance of myelin around the nerve fibers, also in some places disappearance of axis-cylinders. Instead of the normal nervous elements, there is seen a large amount of proliferated neuroglia and connective tissue. Secondary ascending and descending degenerations are distinct. The meninges are frequently found thickened as a result of a previous participation in the inflammatory process. The walls of the blood vessels are also thickened.

FIG. 91.—CHRONIC MYELITIS, SHOWING DEGENERATION OF WHITE AND GRAY SUBSTANCE. (*Original.*)

Symptoms.—They are practically the same as in acute myelitis, when the latter are fully developed, viz. spastic paraplegia with the usual abnormal reflex phenomena, disturbances of the sphincters, sensory disturbances, as paræsthesia, hyperæsthesia or anæsthesia. What characterizes chronic myelitis particularly is the **mild** degree of the disturbances in the sphincters and in the sensory sphere. The spasticity on the contrary is much pronounced. Atrophy of the affected limbs also occurs and the electrical contractility is usually diminished. Reactions of degeneration are rare. The extent of the loss of power in the extremities is various.

In some cases it is absolute, in others only partial and in still others extremely slight. Bed-sores are rare.

Course, Prognosis.—While the disease is usually progressive, in a great many cases it presents stationary periods. At all events it is extremely slow in development. The prognosis is unfavorable, as the destroyed tissue can never be replaced by healthy nervous elements. Death usually occurs from some intercurrent disease or from bulbar involvement, when the myelitis is of ascending character or when confined to the cervical cord.

Diagnosis.—The history of the onset and the grouping of the characteristic symptoms are usually sufficient for making the diagnosis. Sometimes, however, the diagnosis may present some difficulty. In **progressive muscular atrophy,** for example, the muscular wasting is regular and typical in distribution, while in chronic myelitis, if atrophy occurs, it is irregular. Moreover in the latter affection there are sensory disturbances and they are absent in the first. Finally the state of the sphincters will remove the least doubt, as they are intact in progressive muscular atrophy.

Primary Spastic Paraplegia will be recognized by the absence of sphincter disorder, of sensory disturbances and of a history of an acute onset.

A **tumor of the spine** will be recognized by its slow development and by the unusual intensity of pain.

Treatment.—It is largely the same as that of the advanced stage of acute myelitis (see above).

II. Hematomyelia (Hemorrhage in the Spinal Cord)

Hemorrhage in the cord may occur in the course of acute myelitis or specific myelitis, tumors, pernicious anemia and in cases of softening caused by an embolus or thrombosis. In such cases the hemorrhage is a **secondary** condition, merely an accident in the course of those affections.

Hemorrhage in the cord may also be **primary.** It is with the latter exclusively that we will be concerned here.

Pathology.—The hemorrhage may be capillary or in the shape of one or several foci of more or less large size. The favorite seat for primary hemorrhages is the gray matter, because of the great vascularity of the latter. They are particularly marked at the level of the enlargements. They may extend transversely or longitudinally; they may also occur in the central canal. The cervical region is the most frequent seat of trau-

matic hematomyelia; the dorsal is next in frequency. On section the cord appears soft. At first the blood destroys the cells and the axis-cylinders undergo secondary degeneration. Later on, when the clot begins to disappear, the neuroglia tissue around it proliferates. Above and below the hemorrhagic foci tracts of nerve fibers will undergo ascending and descending degeneration. The walls of the blood vessels are thickened and sclerosed. The hemorrhagic focus is eventually absorbed and replaced either by a scar or, as it happens in large hemorrhages, by a cyst, or a cavity surrounded by a thick capsule.

Etiology.—Primary hematomyelia may be **spontaneous** or **traumatic.**

In the spontaneous cases the underlying cause is a state of congestion of the cord. The hemorrhage then occurs after a great physical effort, viz. violent exercises, coitus, paroxysms of whooping cough. It has also been observed in purpura, in hemophilia, in cases of suppression of menses or of hemorrhoidal bleeding, sudden decompression (see Divers' Disease). Spontaneous hematomyelia is **rare.**

Traumatism is the most frequent cause of hematomyelia. Fracture, dislocation of the vertebræ, obstetrical intervention, dystocia forced, inclination of the head, a blow or a fall on the back, a fall on the feet, are the usual causes. In such cases there may be a predisposing factor, such as preëxisting vascular changes.

Symptoms.—The onset is exceptionally sudden (Vulpian's **spinal apoplexy**), sometimes with loss of consciousness. The patient experiences a sharp pain, the seat of which depends upon the level of the injured vertebræ. The patient falls and immediately paralytic symptoms, disturbances of sensations and of the sphincters appear. Sometimes these symptoms appear only a few hours after the onset, but pain in the spine is always present. The lower extremities are usually involved, but according to the seat of the lesion there may be paralysis of all four extremities or only of the arms. The paralysis is flaccid, when the lesion is in the dorso-lumbar segment of the cord; if it is in the cervical cord, the paralysis of the lower extremities will be spastic. The reflexes are exaggerated in the spastic palsy, abolished in flaccid paralysis. The reflexes as a rule are abolished immediately after the onset, irrespective of the level at which the injury occurs.

The muscles gradually undergo atrophy.

The sensory disturbances at the beginning will consist of a numbness and tingling in the affected limbs. Objective sensory disturbances are always present. They consist of a marked diminution of all forms of sensations or of a complete anæsthesia of the affected limbs. Sometimes a syringomyelic sensory dissociation may be evident: it consists of a loss of

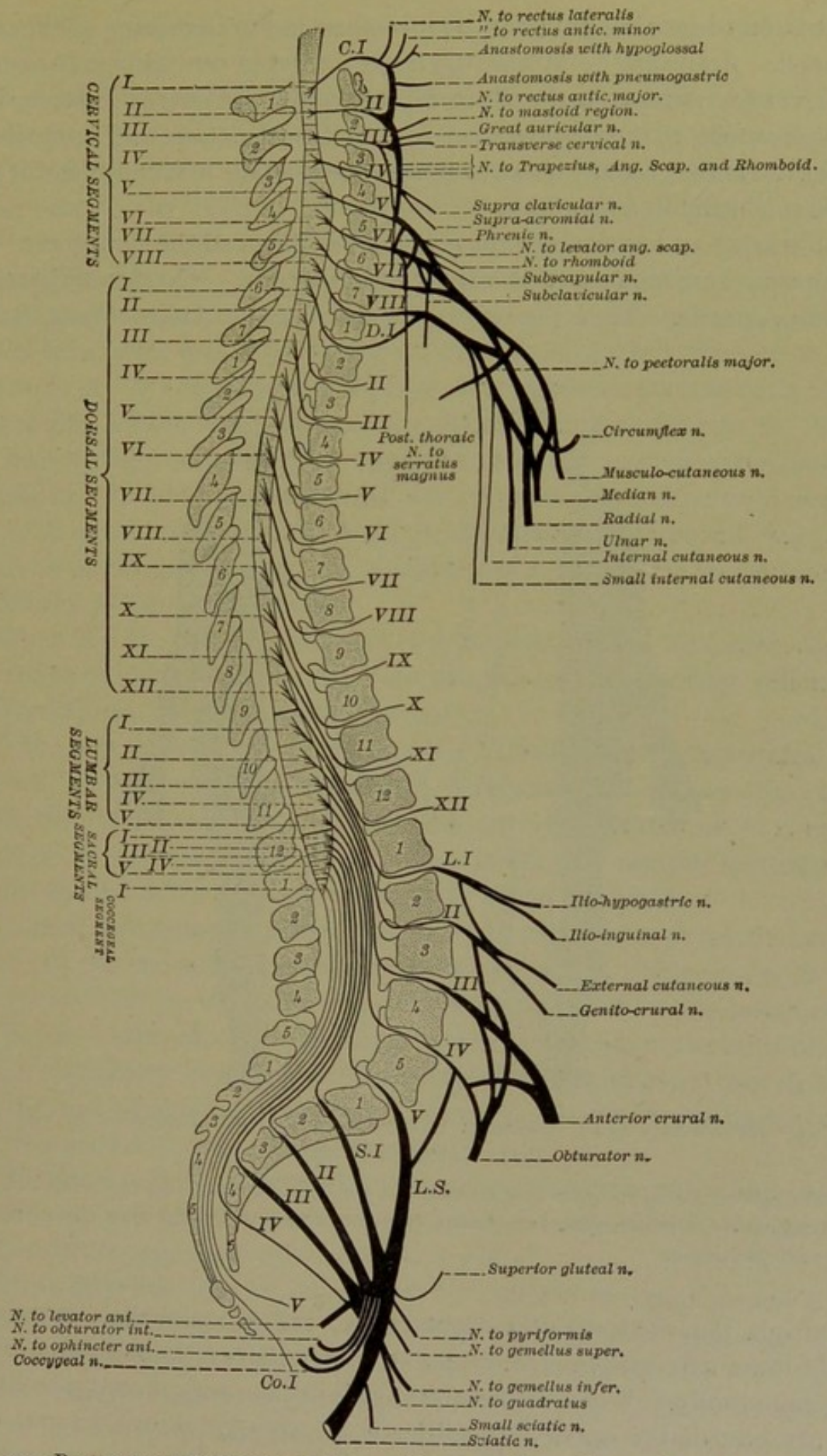


FIG. 92.—RELATION OF SEGMENTS OF SPINAL CORD AND THEIR NERVE ROOTS TO THE BODIES AND SPINES OF THE VERTEBRÆ. (*After Dejerine and Thomas.*)

diminution of sense of pain and temperature and preservation of the sense of touch. According to Minor, the latter occurs in central hematomyelia.

A very important and early symptom which persists is a paralysis of the sphincters of the bladder and rectum (constipation and retention of urine). There is usually a rise of temperature at the beginning, but it goes down to normal in a few days.

If death does not occur at the end of a few days, all the symptoms begin to improve and some of them may even disappear. The flaccid paralysis becomes spastic. Then the reflexes are increased. Ankle-clonus, Babin-

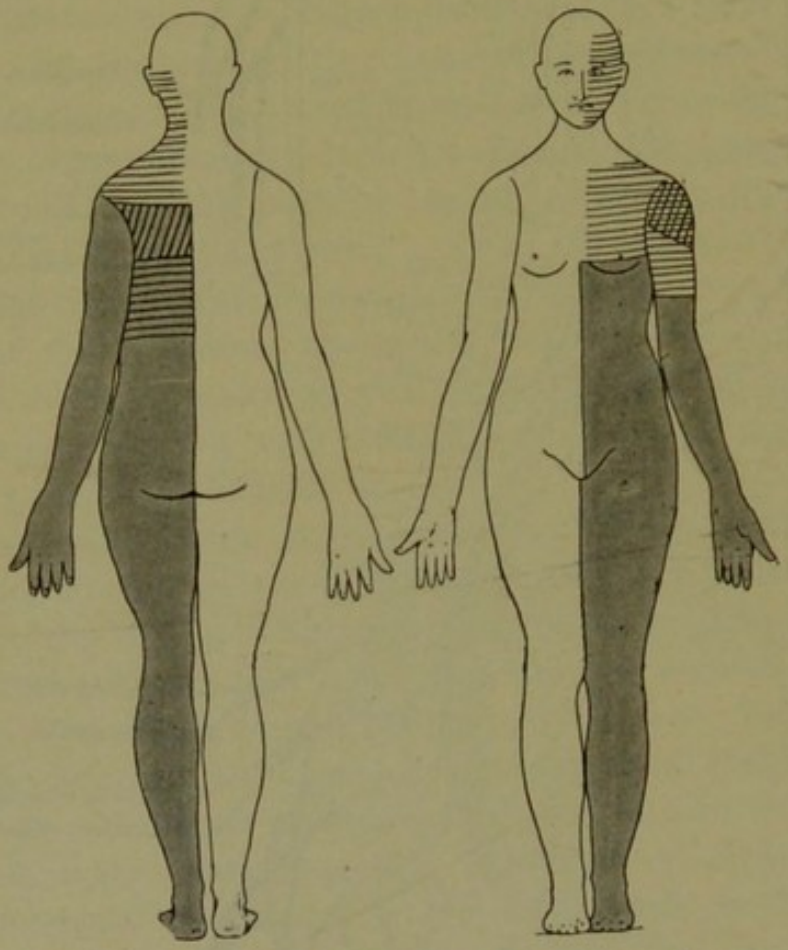


FIG. 93.—BROWN-SÉQUARD'S PARALYSIS FOLLOWING A GUNSHOT FRACTURE OF THE RIGHT FIFTH CERVICAL VERTEBRA.

Paralysis of the right upper and lower limbs and sensory disturbances on the left. The black indicates thermo-anæsthesia and analgesia; the portion shaded in lines—hypalgesia and thermo-hypæsthesia.

ski's sign, paradoxical reflex, Oppenheim's reflex, make their appearance in the lower extremities. Muscular atrophy with reactions of degeneration develop rapidly. When the hemorrhage is in the cervical cord, the muscular atrophy of the upper extremities is of Aran-Duchenne's type. The sensory disturbances usually disappear, but sometimes syringomyelic

dissociation persists. The condition of the sphincters improves to some extent.

In hematomyelia of the lowest **cervical** segment, in addition to the spastic paralysis and muscular atrophy of the upper extremities and sensory disturbances of syringomyelic type, there are also **oculo-pupillary** disturbances, viz. myosis, narrowness of the palpebral fissure. When only the eighth cervical and the first dorsal segments are involved, the sensory disturbances occupy a longitudinal band at the inner border of the entire limb and the atrophy is limited to the muscles of the hands. When the seventh, sixth and fifth cervical segments are affected, the sensory disturbances are on the external border or on the anterior surface of the limb; the atrophy affects the muscles of the shoulder, arm and forearm. When the hemorrhage occurs in the upper cervical segment, paralysis of the phrenic nerve and of the diaphragm and consequently difficulty of respiration will ensue. In hemorrhages of the **thoracic cord** there will be spastic paralysis of the lower limbs, paralysis of the abdominal muscles, and involvement of the sphincters.

When a hemorrhage occurs in the lowest part of the cord, viz. in the conus medullaris, there will be a special symptom-group which will be described in "Diseases of the Conus."

If the seat of the hemorrhage is in one half of the cord, the clinical picture will be that of **Brown-Séquard's paralysis.** It consists of a loss or diminution of sensations on the side opposite the lesion and of a motor paralysis on the side of the lesion; these motor and sensory disturbances are distributed in the portions of the body below the lesion.

Course, Termination, Prognosis.—Death may occur in a few hours or days. In the majority of cases improvement follows. The prognosis cannot be made before the acute stage has subsided. When the hemorrhage is in the cervical cord, the outlook is very serious because of the involvement of the respiratory centers. Disturbance of the sphincters may lead to cystitis and ascending nephritis. Even in the most favorable cases complete recovery can never be expected.

Diagnosis.—In **Syringomyelia** paralysis, muscular atrophy and sensory dissociation are characteristic, but a sudden onset with a very marked paraplegia are only met with in hematomyelia.

Acute Myelitis will be recognized by absence of atrophy, and if the latter is present, it is usually slight. Besides, in myelitis the onset is not so abrupt and the paralytic symptoms are predominant.

In **meningeal hemorrhages** there is a very intense pain in the back along the nerves, the spinal muscles are rigid, but no sensory dissociation.

Treatment.—At the onset absolute rest is necessary. Immobilization

of the body is advisable in order to avoid another hemorrhage. The patient should lie on his side. Counter-irritants are to be avoided, as they may lead to ulcerations. Every effort should be made to avoid secondary infections. Ergot had been recommended. In the chronic stage massage, warm baths and systematic exercises (see Treatment of Spastic Paraplegia) should be applied.

III. Caisson Disease (Divers' Paralysis)

Divers or workers in caissons, being under the influence of high atmospheric pressure while at work, present nervous disturbances when they return to the surface.

Pathology.—The most constant microscopical changes found in almost every case are: congestion of brain and cord and internal organs in acute cases, softening in chronic cases. In the incurable cases of long standing, in which the condition remains permanent until death, lesions of typical chronic myelitis are the usual findings. In one case that came under my observation the patient lived three months. The autopsy showed softened areas in the gray matter and in the posterior and lateral columns. Ascending and descending degenerations could be traced. Leyden reported a rupture of the dorsal cord.

Etiology.—The immediate cause of the disorder of the nervous system is the lessened atmospheric pressure when the workers return from the caisson, but the primary factor is the increased atmospheric pressure beneath the surface.

Symptoms.—Shortly after the return to the surface and after a prodromal stage, consisting of pain more or less severe in the large joints in the muscles, especially in those of the back, also in the epigastrium and sometimes over the entire body, a paralysis occurs. The most frequent form of this is paraplegia, but sometimes hemiplegia is observed. The pain is of a neuralgic character and it may be so severe that it doubles up the patient, hence the common term "the bends." The paralysis attacks more frequently the lower extremities than the upper. Sensations are always disturbed: there may be total anæsthesia in the limbs and at the same time intense pain. The onset and the character of the paralysis is very similar to that of transverse myelitis. If we take into consideration the frequent involvement of the sphincters (retention and constipation) and the sensory disturbances, the resemblance to myelitis will be complete. The involvement of the motor and sensory apparatuses may be complete or only partial or else unequally distributed. There may be no relationship between the objective sensory disturbances and the degree of paralysis;

either may be affected without the other. In some cases there may also be vertigo, headache, vomiting, slight confusion, convulsions and double vision. Prostration is present in severe cases. In fatal cases deep coma, irregular respiration and symptoms of cardiac paralysis announce approaching death. As a not infrequent occurrence may be mentioned small perforations of the ear-drums or such a congestion of the inner ear as to give the picture of Ménière's disease. Aphasia has been occasionally observed. Ecchymosis of the skin is also an occasional occurrence.

Pathogenesis.—There are two views for the explanation of the symptoms. According to one of them, the so-called gaseous theory, the blood while under high pressure becomes overcharged with gas (oxygen and carbonic acid). When the surface is reached, the gas attempts to escape through the lungs. In the meantime the superfluous gas circulates in the blood in bubbles, and may either form emboli or escape through the vessel walls into the surrounding tissues and consequently produce considerable pressure. The spinal cord suffers the most, because besides being situated in a hermetically closed cavity, it has a slow return circulation (because of a large number of plexuses). According to the other view, there is a congestion followed by stasis. The high pressure drives the blood from the periphery to the internal organs, especially to the nervous system. The blood vessels of the latter having no support from counter-pressure remain dilated. A paralysis of their walls takes place. When the atmospheric pressure is diminished, the paralyzed vessels cannot follow and stasis will be the result.

Prognosis.—An attack may last from several hours to six days. When the symptoms are pronounced, death usually ensues. When the paralytic and other symptoms are mild, improvement and even recovery may follow. In some cases the myelitic symptoms persist indefinitely.

Treatment.—It is that of myelitis (see above). Prophylaxis is the most important part of the treatment. Bad physical health, diseases of the kidneys and heart, alcoholism, obesity and hunger are all contra-indications for subjecting one's self to high atmospheric pressure. The return to the surface should be done in caissons supplied with locks, so that the reduction of pressure be made very gradually. Haldane and Boycott (*Jour. of Hygiene*, Cambridge, 1908) have laid out the following rules for gradual decompression: when a diver has been working at a depth of 144 feet for ninety minutes, he should ascend at once to 50 feet, then rest for ten minutes. From that time on he should come up 10 feet four times in succession with intervals of ten, twenty, thirty and thirty-five minutes, after which he leaves the water.

IV. Syringomyelia

Syringomyelia is characterized by a formation of a cavity or cavities in the spinal cord.

Pathology.—The macroscopical aspect of a syringomyelic cord is often quite characteristic. It may be flat, soft or fluctuating. Its cervical portion, the usual seat of the lesion, is unduly enlarged. A transverse section will reveal the presence of a **cavity**; the latter may be single or multiple. It is mostly situated in the cervical segment, it may also occupy the entire length of the cord. Most frequently it is found in the **posterior commissure** and **posterior cornua.** In some cases it extends so far backwards that it destroys the posterior roots at their entrance into the cord. The white matter (lateral columns) becomes involved

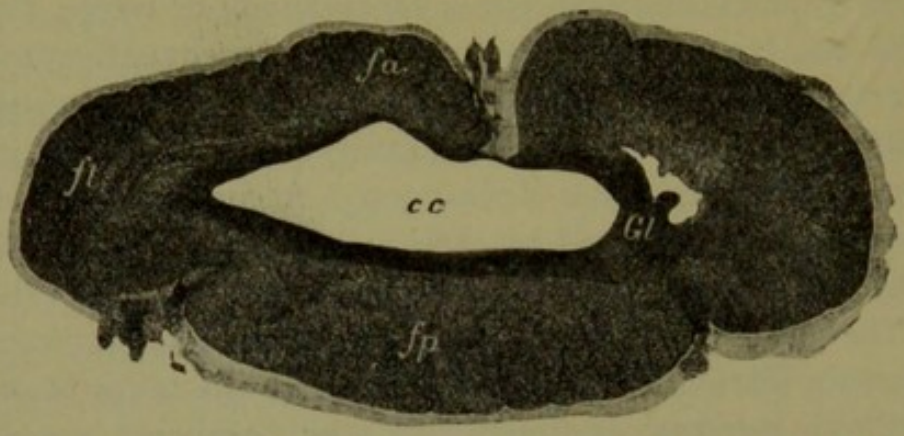


Fig. 94.—Hydromyelia of the Upper Dorsal Cord. (*Strümpell.*)

when the cavity has destroyed the gray substance. The cavity may extend into the medulla. A large cavity may open into the central canal. Sometimes the entire pathological process consists of a primary enlargement of the central canal. The latter constitutes **hydromyelia.**

The contents of syringomyelic cavities is a fluid analogous to the cerebro-spinal fluid. Microscopically a **gliomatous** formation is noticed, in the midst of which lies the cavity. This tissue consists of glia cells and glia fibers.

The cavity is due to a softening and collapse of the center of the gliomatous tissue. The normal nervous elements affected by the latter are naturally destroyed. The tracts of nerve-fibers degenerate according to their directions. Cavities may be also the result of **inflammatory** processes within the cord or of **hematomyelia** (see this chapter). In such cases, instead of the characteristic elements of glioma, the cavity is surrounded by sclerosed tissue. These two forms, while clinically they may present the typical symptoms of the disease, are nevertheless not the

true syringomyelia, which is essentially a gliomatosis and has for basis very probably a developmental anomaly: the nests of glia cells left in the central canal begin through some cause (trauma or others) to proliferate and lead to new formations.

Etiology.—Trauma apparently plays an important rôle, as there are a number of cases on record, in which the symptoms begin to develop shortly after the shock. Guillain called attention to this fact that an infected wound may be the cause of syringomyelia by means of an ascending neuritis. Exposure to cold, exhaustion are also mentioned by some writers among the causes of the disease. The consensus of opinion is that a congenital defect of the central canal is the most frequent cause. Most frequently the disease begins at the age of between ten and thirty. There are also cases with an onset in infancy. Males are more frequently affected than females.

Symptoms.—They are: **sensory, trophic** and **motor.**

Sensory.—They constitute the **sensory dissociation** characteristic of the affection It consists of **loss of sense of pain and temperature with preservation of the sense of touch.** The involvement of temperature sense (thermoanæsthesia) is a very grave sign. The patients often being unaware of it, burn their hands. In one of my recent cases the hands and forearms were literally covered with cicatrices as a result of unnoticed burns. It is only accidentally the patient would notice that her hands came in contact with fire or a hot stove. Application of ice to the hands did not give her the slightest sensation of cold; she felt only the contact of a solid object.

The loss of pain-sense (analgesia) is usually of the same intensity as that of temperature. The patient just mentioned developed a cellulitis of the palm of one hand and at no time she experienced pain. An incision was made by the surgeon without a general or local anæsthetic, the wound was cauterized and no pain was felt by the patient. She also had a dislocated shoulder on one side and she could not tell how and when it appeared. A close questioning elicited a history of a fall. She therefore did not feel the pain at the time of the dislocation. The characteristic sensory dissociation is not absolute in every case. Pain and temperature senses may be only diminished, or one may be more or less pronounced than the other. The tactile sense in advanced cases may sometimes be also affected.

It must be also mentioned that sometimes the thermal sense may be **perverted,** viz. heat is taken for cold and cold for heat. Another characteristic feature of the sensory disturbances lies in their **radicular distribution,** viz. longitudinal or parallel to the limbs. The upper extremities and the trunk are more frequently involved than the lower limbs.

Subjective sensory disturbances are usually present. **Pain** is frequent, especially at the beginning. The sense of pressure is diminished. Astereognosis is sometimes observed.

The existence of the sensory dissociation can be explained by this anatomical fact, that Gowers' tract, which contains fibers for conducting pain and temperature, originate in the cells of Clarke's column of the opposite side. As they cross the median line they are interrupted by the syringomyelic cavity, the usual seat of which is near the central canal (see Anatomy).

Trophic.—Muscular atrophy is almost constant. The cause of it is the involvement of the anterior cornua by the cavity. It has usually

FIG. 95.—SYRINGOMYELIA. DEFORMITY OF HANDS AND FINGERS, CICATRICES ON SOME FINGERS AS A RESULT OF SELF AMPUTATION OR PAINLESS ULCERS. ŒDEMATOUS HANDS.

the form of Aran-Duchenne's type (see Progressive Muscular Atrophy). Here we find the typical onset in the small muscles of the hands, the **claw-like** hand, the **preacher's hand** (Charcot). The latter is due to overaction of the extensors of the forearm, while the muscles supplied by ulnar and median nerves are paralyzed. The remaining muscles of the upper extremities as well as those of the shoulders follow the atrophy of the muscles of the hands. Later on the muscles of the back, intercostal and

abdominal and finally those of the lower limbs undergo atrophy. Deformities of the feet follow. The atrophy does not always follow the regularity just mentioned. There are great variations in its distribution. The character of this distribution is chiefly radicular.

Fibrillary contractions in the affected muscles are constant. Reactions of degeneration or only a quantitative diminution of electrical contractility may be present.

Besides the muscles other tissues suffer in their nutrition. The **skin** may break and leave sores. It may become glossy, covered with vesicles and eruptions, the nails suffer, the phalanges are deformed. Morvan's disease, which is characterized by painless panaritiæ, is a variety of syringomyelia: the tendons and terminal phalanges become necrosed, are gradually eliminated and slowly a cicatrix is formed. During this entire process there is **no pain.** The perforating ulcer on the feet is frequent in syringomyelia. Raynaud's disease is sometimes observed, although it never reaches the phase of gangrene. Marinesco described an **œdematous hand** (main succulente), which does not pit on pressure, as pathognomonic of the disease (Fig 95).

Arthropathies are quite frequent. It may be only hydarthrosis or a destruction of the epiphyses with subsequent dislocation.

The **bones** also suffer in their nutrition. Spontaneous fractures occur. Formation of a callus is slow and difficult. Scoliosis associated with kyphosis is not infrequent.

Vasomotor disturbances consist of cyanosis of the extremities, œdema, hyperhidrosis.

Motor.—Paralytic symptoms develop slowly and progressively. Spastic paralysis is a frequent symptom. In some cases it is very pronounced and contractures deform the trunk and the extremities. In such cases the reflexes are exaggerated and the toe phenomenon will be present. When the posterior columns become involved, tabetic symptoms will be manifested, viz. ataxia, Romberg's sign, loss of reflexes. Various oscillatory movements, tremors, choreiform movements have been observed in some cases.

The sphincters are usually not involved.

Ocular symptoms are present when the lesion is in the lower cervical region, and in the first dorsal segment: myosis, retraction of the eyeball, narrowing of the palpebral fissure, Argyll-Robertson pupil, and defective sweating.

When the cavity extends to the **medulla,** bulbar symptoms develop. Difficulty of deglutition, paralysis of the vocal cords, irregular action of the heart, facial paralysis, hemiatrophy of the face and tongue, ocular palsies

with nystagmus are then observed. Sometimes the lesion involves the trigeminal nerve; anæsthesia over its area of distribution will be the consequence.

Course, Termination, Prognosis.—The disease is essentially chronic and its onset is usually slow. It lasts may years, during which there may be periods of remission, but it is inevitably fatal. Death ensues either from bulbar involvement or from some intercurrent disease (tuberculosis, pneumonia, etc.).

Diagnosis.—**Progressive muscular atrophy** of spinal origin has the same distribution of wasting as syringomyelia, but in the former the characteristic sensory disturbance of the latter is absent.

Amyotrophic lateral sclerosis will be also recognized by the absence of sensory dissociation and of trophic disturbances.

Syringomyelia presents in some cases **tabetic** symptoms (ataxia, loss of reflexes, Romberg's sign, arthropathies), but the absence of characteristic ocular symptoms, of sphincter disturbances of **tabes** will soon decide the diagnosis.

Leprosy is characterized by muscular atrophy, sensory and trophic disturbances. The atrophic hand of leprosy resembles that of syringomyelia, but the mutilations of the phalanges in the latter never reaches the extent of those of leprosy. The mutilated toes (rare in syringomyelia), muscular atrophy in the lower limbs (rare in syringomyelia), absence of scoliosis, absence of increased reflexes—are all in favor of leprosy. In the latter disease sensory dissociation is rare.

In **Morvan's disease** the number of painless panaritiæ is considerable. Sensory dissociation is usually absent.

Treatment.—It is only symptomatic The ulcerations will be treated antiseptically. For muscular atrophy massage and electricity are advisable Care should be taken of the skin in giving massage, as erosions are likely to lead to ulcerations, which are slow in healing Surgical operations should be avoided Internally iodides, arsenic and iron may be given

During the last few years favorable results have been obtained from treatment of Syringomyelia with X-rays. Among the most recent contributions to the subject may be mentioned that of Allaire and Denès (Gaz. Méd. de Nantes Nov., 1912).

Morvan's Disease

Under this name is desrcibed an affection which has been observed in Normandy and which according to some neurologists can be considered as a clinical variety of syringomyelia, in which, in addition to the anæs-

thesia and analgesia, paretic symptoms with muscular atrophy, there are also multiple painless **panarities.** However the sensory dissociation is usually absent. The sense of touch is affected as much as the pain and temperature senses. The sensory disturbances are distributed like in neuritis, viz. decreasing from the distal to the proximal end of the limb.

V. Diseases of Conus Medullaris and Cauda Equina

The lower portion of the spinal cord, called conus medullaris, is surrounded by bundles of nerve fibers coming from lumbar and sacral segments (cauda equina) The study of this important portion of the cord is comparatively recent. Anatomically the conus corresponds to the second lumbar vertebra and its filum terminale begins at the level of the middle of this vertebra. The conus comprises the last three sacral and coccygeal segments. Physiologically conus medullaris is a very important portion of the cord. It contains special and independent centers for micturition, defecation, erection, ejaculation and for the anal reflex. Diseases of the conus present a special clinical picture which deserves a separate description.

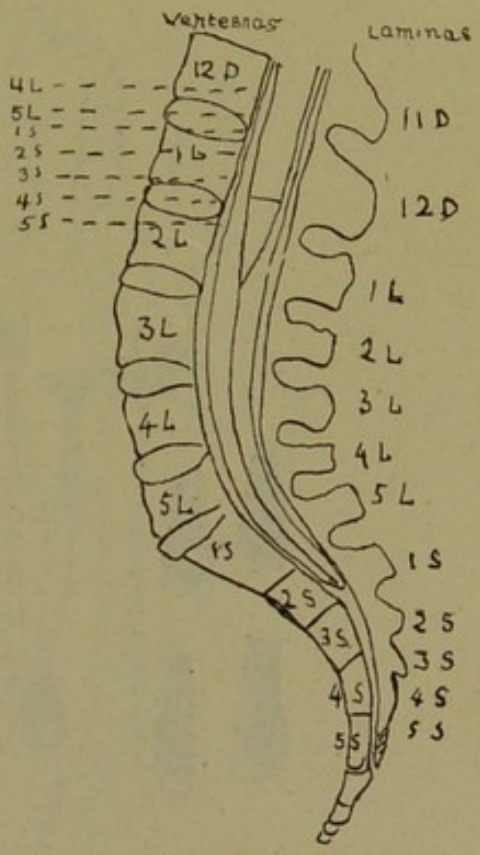


FIG. 96.—CONUS MEDULLARIS (*After Raymond.*)

Pathology.—All possible lesions of the spinal cord studied on the preceding pages may also affect the conus. We may therefore have those of myelitis, of hemorrhage, of tabes, etc.

Etiology.—Trauma is the most frequent of all causes. In cases of fracture of the lumbar and sacral vertebræ, a fragment is likely to press directly upon the conus or the roots. Secondary myelitis will follow. Trauma may also produce a hemorrhage in the conus. A blow over the lumbar region may produce a traumatic myelitis of the conus. An injury below the second lumbar vertebra will damage only the cauda equina; at the level of the second lumbar will involve the conus and the cauda equina. An injury immediately above the second lumbar vertebra will damage the conus alone. The lowest part of the spinal cord is not infrequently the seat of **tumors.** The latter may originate in the vertebræ

or in the cutaneous coverings or else be primary (sarcoma, lymphangioma, glioma, gumma). Abscess due to tuberculosis of the vertebræ may also involve the conus.

Symptoms. Conus.—From the anatomical fact mentioned above, namely that the conus is the center for the third, fourth and fifth pairs of sacral nerves (Fig. 92), also from the physiological character of the conus, viz. that it contains special centers for micturition, defecation, etc. (see above), there is no difficulty in presenting a clinical picture of

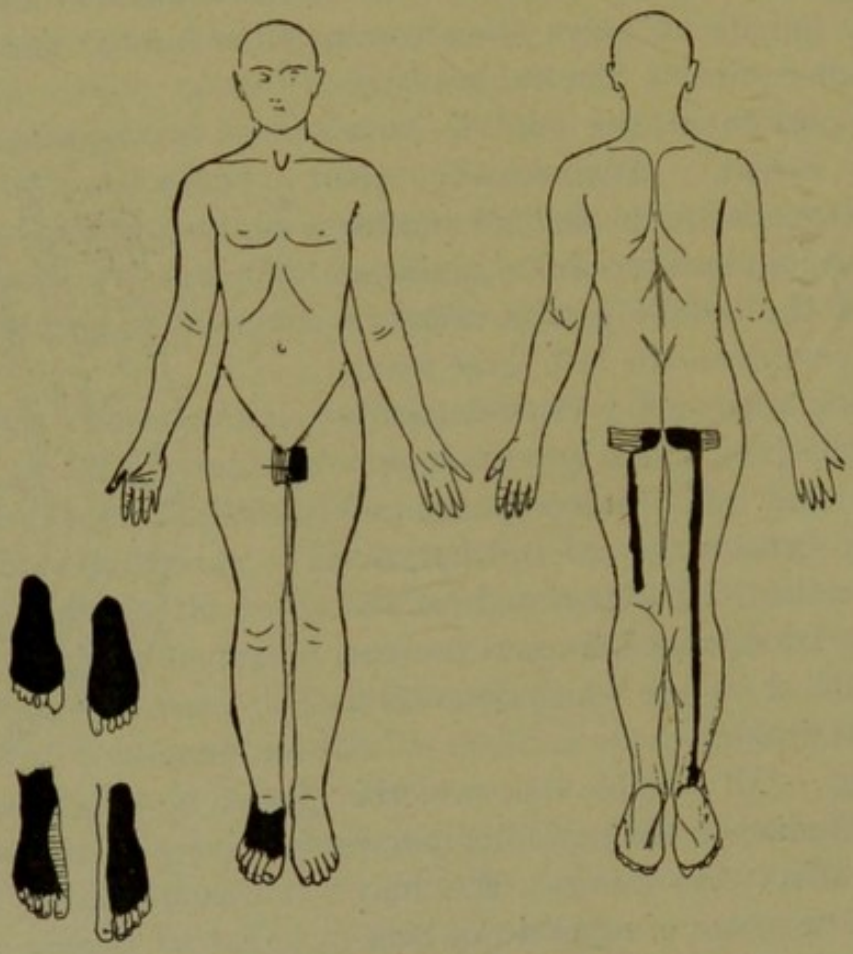


FIG. 97A.—SOLID PORTION THERMOANÆSTHESIA; SHADED, REVERSED TEMPERATURE SENSE.

diseases of the conus. The symptoms therefore are (Figs. 97a, 97, 98):

1. Anæsthesia or sensory dissociation of the external genital organs, perineum, anus, inferior gluteal region and the postero-superior area of the thighs (saddle-shaped anæsthesia). It may also extend to the mucous membrane of the genito-urinary apparatus. The tenderness of the testicles is intact.

2. Paralysis of the bladder and rectum. There may be incontinence or retention.

3. Involvement of the sexual function and of the sensation of ejaculation.

4. Pain is a frequent but not constant phenomenon. If it is present, it may be of great severity. As to the motor power of the lower extremities it is normal. The reflexes are equally unaffected.

The onset of these symptoms is sudden in traumatic cases, slow in cases of tumors. As the conus is immediately surrounded by the nerve bundles (cauda equina), a lesion of one is bound to involve the other and the symptomatology therefore is complex.

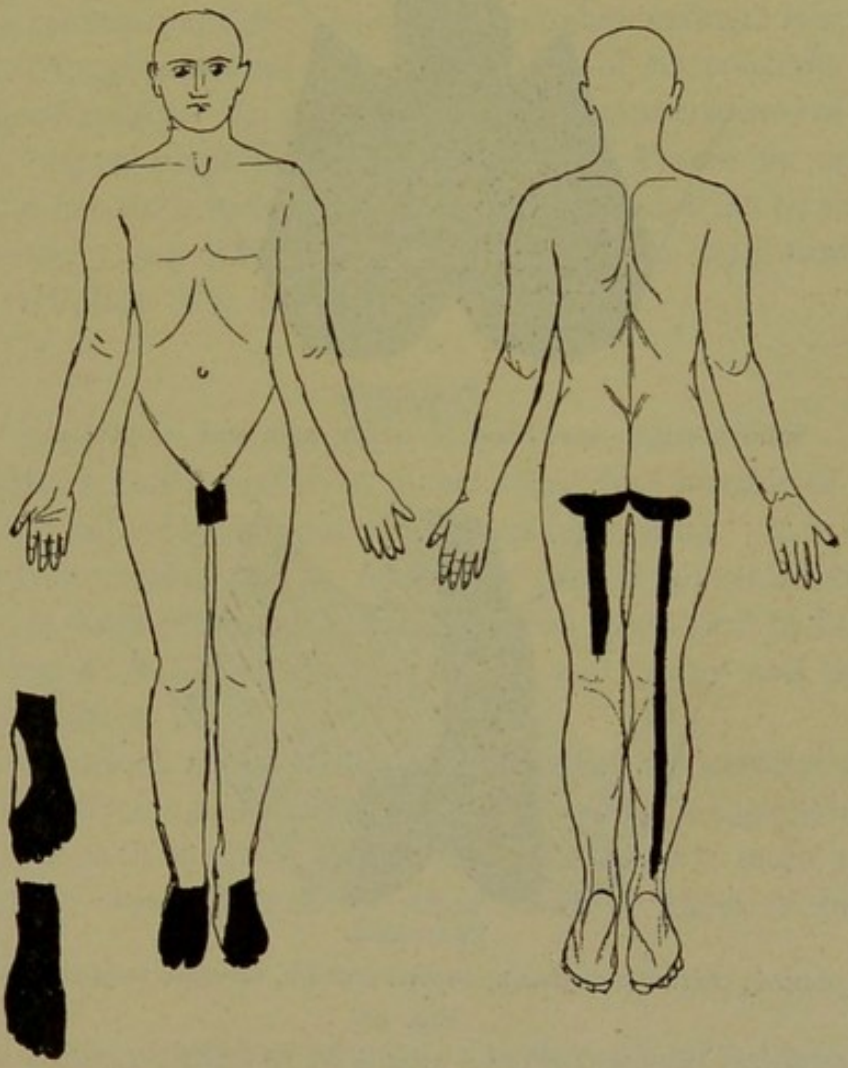
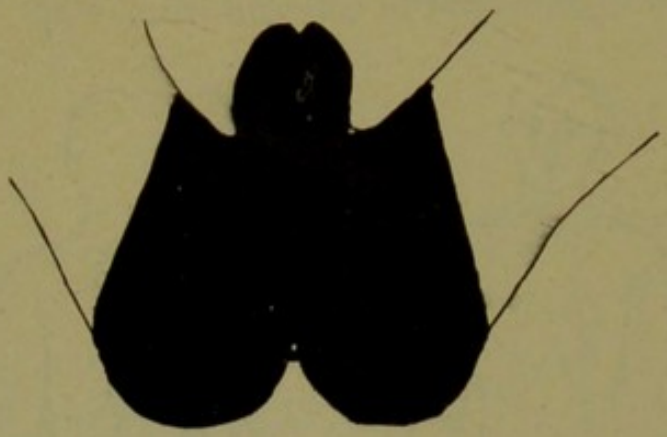


FIG. 97.—SOLID PORTION, ANALGESIA.

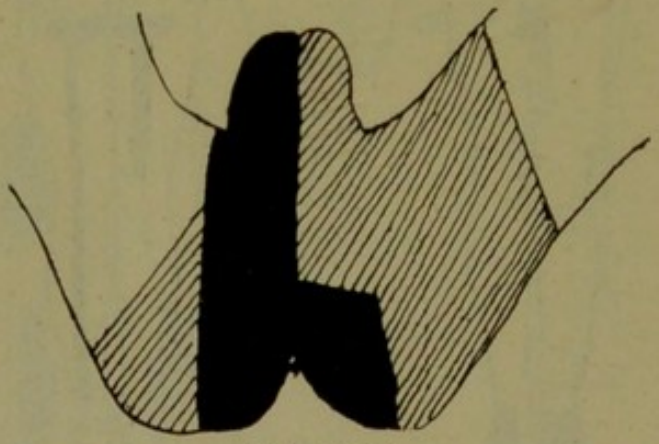
Cauda Equina.—If a tumor is situated below the conus, the main symptoms will be those of involvement of the roots, viz. pain and objective sensory disturbances. The pain is intense in the lumbo-sacral region and in the limbs, chiefly in the region of both sciatic nerves; it is constant and increased upon the slightest movement. The objective sensory disturbances may be only anæsthesia. They occupy the lower extremities beginning from the lower two-thirds of the thighs downward, the perineum, anus and the genito-urinary organs. There is anæsthesia of the mucous

membranes of the urethra, bladder, vulva, vagina. The paralysis is flaccid at the beginning and during the entire course of the diesase. There is a high-steppage gait. Muscular atrophy appears early in the lower limbs. Reactions of degeneration are present. The reflexes Achilles, plantar and anal are abolished, but the knee-jerks are intact. The sphincters are disturbed. Bed-sores are frequently present.



PERINEUM.

Splid portion, Anesthesia to touch, pain and temperature.



PERINEUM.

Solid portion, thermo-anesthesia; shaded portion, reversed temperature sense.

FIG. 98.

Course, Prognosis.—In traumatic cases death may follow the injury, but usually the condition becomes chronic. The outlook is never favorable, but much better in lesions of the cauda equina than of the conus. The involvement of the sphincters is permanent and eventually leads to the consequences described in myelitis.

Diagnosis.—It is very important to localize the lesion in the conus or in the cauda equina. In the first case operative procedures are impossible, while in the second case they are indicated.

Lesions of cauda equina are usually very painful, the pain radiates toward the legs, the symptoms develop gradually. The pain is present be-

low the second lumbar vertebra; flexion of the thigh over the pelvis while the leg is extended provokes pain. The function of the bladder and rectum improves. On the other hand pain above the second lumbar vertebra, well-marked anæsthesia and especially sensory dissociation, exceedingly slight or no improvement at all in the function of the sphincter—are all symptoms of the conus.

Treatment.—Not much can be expected from external applications, as counter-irritation, revulsion in traumatic cases. The rational treatment is surgical intervention. In cases where hemorrhage is suspected, a puncture in the spinal canal with evacuation of its contents may give immediate good results. In cases of fracture prompt removal of fragments is urgent. Tumors must be equally removed. Before an operation is undertaken, a detailed study of the case must be made as to the localization of the sensory and other disturbances in order to determine the exact field of the operation (Fig. 92).

Epiconus

In 1900 Minor called attention to lesions of that portion of the spinal cord which lies immediately above the conus (epiconus). It is frequently involved in traumatisms and it presents a special symptomatology, viz. (1) integrity of knee-jerks, (2) integrity of the sphincters of bladder and rectum, (3) loss of Achilles reflex, (4) serious and protracted paralysis of the external popliteal nerve.

Frequently lesions of the conus and epiconus are associated and not rarely the cauda equina is simultaneously involved. The special symptoms characteristic of each of these portions will enable one to make a diagnosis of the complex cases as well as when each of them is individually affected.

VI. Disseminated or Multiple Cerebro-spinal Sclerosis (Insular Sclerosis)

Pathology.—The disease is characterized by islets of sclerosis disseminated throughout the entire central nervous system. They can be seen also in some of the cranial nerves, viz. optic, olfactory and trigeminal. On transverse section yellowish spots of various sizes can be seen even with a naked eye. Their distribution is very irregular, they do not show any special predilection for any particular tract, but they are frequently found in the white substance of the cord, brain and cerebellum, in the anterior portion of the pons and in the pyramids of the medulla. Histologically

the sclerosed patches present a characteristic picture: the myelin is destroyed, but the axis-cylinders are remarkably preserved. The latter fact is probably the reason of absence of secondary degeneration. Sometimes the axis-cylinders are swollen, but they rarely disappear, except in very old cases. The neuroglia tissue in the diseased area proliferates abundantly. The blood vessels of sclerosed patches are sometimes, but not always, found altered (endo- and periarteritis) The cells are usually spared. As to the origin of the sclerotic process, some believe that proliferation of the neuroglia is the initial process, to others the initial lesion is a myelitis: the nerve-fibers are primarily affected and the neuroglia secondarily

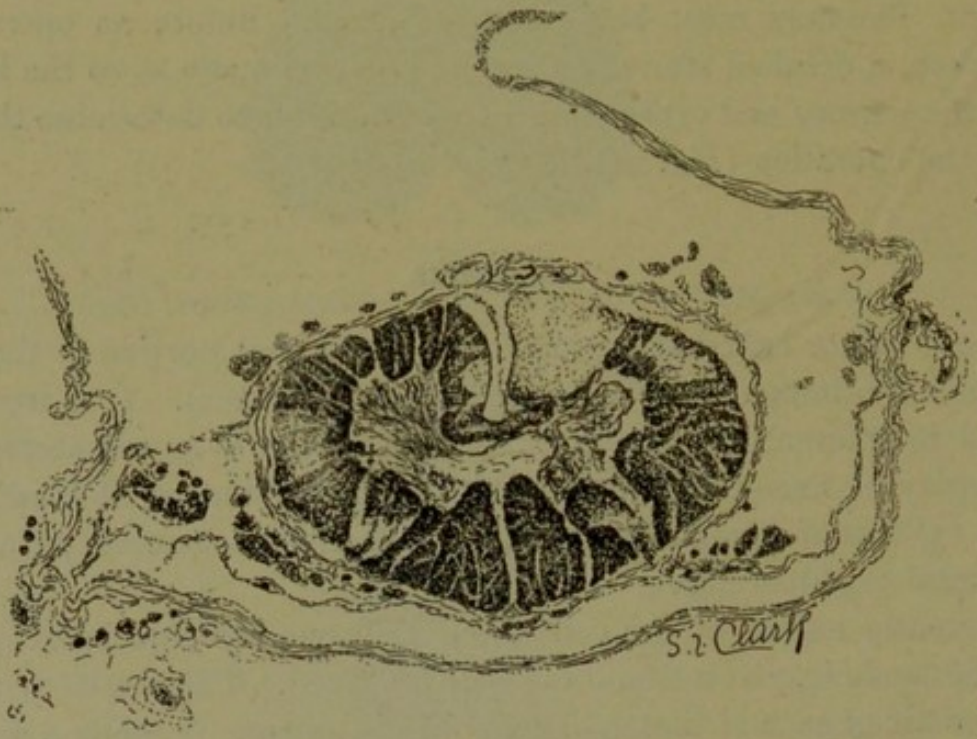


FIG. 99.—SCLEROSIS OF PYRAMIDAL, GOWERS' AND DIRECT CEREBELLAR TRACTS. (*Original.*)

Symptoms.—In view of the disseminated character of the sclerotic lesions, the clinical picture naturally varies from one case to another and depends upon the seat of the sclerotic islets. If the lesion is in the posterior columns, symptoms of tabes will be present. If the lateral motor columns are affected, spastic paraplegia will be the symptom. If the posterolateral columns are involved, ataxic paraplegia will be the result. If Gowers' tract is diseased, symptoms of syringomyelia will be observed. If the anterior cornua suffer, muscular atrophy will develop. If the internal capsule has a plaque of sclerosis, hemiplegia will follow

As the essential feature of the sclerotic process is its dissemination in various portions and at various levels of the central nervous system, a combination of symptoms of all the diseases mentioned is expected. In

fact there are several clinical forms of multiple sclerosis. They depend upon the predominance of one or another group of symptoms

There is, however, a **series of symptoms which are very frequently present** in this disease and without which a diagnosis of multiple sclerosis is almost impossible. If they are not all always present, at the same time some of them at least will help to decide the diagnosis. They are:

1. **Tremor.**—It is of a special character: it appears only upon a voluntary act. It is an **"intention tremor."** Should the patient, for example, attempt to carry a glass of fluid to his mouth, he is bound to spill it, and the nearer he gets it to his mouth the more the tremor will be pronounced,

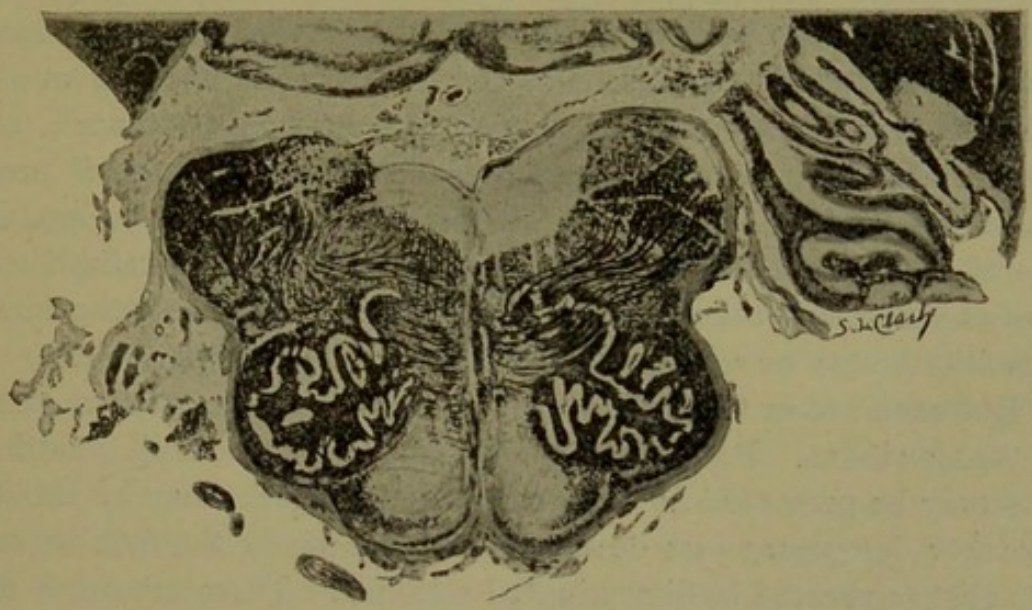


FIG. 100.—PLAQUES OF SCLEROSIS IN PYRAMIDS AND VARIOUS NUCLEI. (*Original.*)

so that the glass will strike the mouth and the teeth in all directions and he will fail to drink the contents. Writing is one of the first acts in which tremor is manifested: at first all the letters are irregularly, unequally separated and placed at different levels. In a more advanced state writing is illegible and then impossible. The legs, the trunk and the head may also be affected by tremor, but this is more rarely met with. The tremor may be at first unilateral or predominate on one side, later it becomes generalized.

Not only upon voluntary acts irregular jerky movements are observed, but sometimes jerky spontaneous acts take place, such as sudden crying or laughing. They occur without any relation to a preceding emotion.

2. **Disturbance of Speech.**—The patient speaks slowly, pausing between words and between syllables. In advanced cases he accentuates each syllable. It is a **scanning, syllabic, staccato** speech. Besides, the pronunciation of certain letters is especially defective, viz. b, c, g and r.

As a rule the first words are pronounced distinctly, but soon the speech becomes indistinct. This is probably due to fatigue of the muscles concerned in speech.

3. **Nystagmus.**—It consists of an oscillation of the eye globes, when the patient turns his eyes to an extreme position laterally or vertically. Lateral nystagmus is more frequent than vertical. In advanced cases the least movement of the eye globes will produce a nystagmus.

4. **Disturbance of Gait.**—It is most commonly **paretic** and **spastic.** As soon as the patient commences to walk, the lower limbs become contracted, the legs extend over the thighs, the feet over the legs, and adduction is extreme. Moreover the patient has a tendency to walk on his toes, he topples over when he walks quickly. Sometimes there may be a gait which is **spastic and ataxic** at the same time. The patient stands with his feet widely separated and soon as he attempts to walk, ataxia is evident and there is a tendency to fall. Incoördination, if present, is of a **cerebellar type** (titubation) (see Cerebellar Diseases). Oppenheim speaks also of an **oscillating gait,** characterized by a generalized tremor as soon as the patient takes the first step. In advanced cases the paralysis and spasticity may be so pronounced that walking is impossible.

5. **Reflexes** are in the majority of cases increased in all four extremities. Ankle-clonus, Babinski's sign, Oppenheim's and paradoxical reflexes may be present. In some cases the knee-jerks may be abolished.

The five symptoms just described, and especially the first three, are almost always present in disseminated sclerosis. There are a few others which, while not very frequent, are met with in quite a large number of cases. They are:

1. **Visual.**—A partial **optic atrophy** is the most frequent. It may be unilateral or bilateral. In the earlier stages of the disease there is only pallor of the optic nerves, which is unequally distributed in both eyes. A progressive diminution of visual acuity, contraction of the visual field, central scotoma for certain colors are the other ocular changes. Transitory visual defect in one or both eyes is quite common. Occasionally palsies of ocular muscles occur. The sixth nerve is more frequently involved than the third. The reflexes of the pupils are very occasionally affected.

2. **Apoplectiform or Epileptiform Seizures.**—The first are more frequent than the latter. The onset of hemiplegia is accompanied by a loss of consciousness. The paralysis is usually transient, but it may persist. When the paralysis persists, the disease is considered as the hemiplegic form of multiple sclerosis. The hemiplegia may be complicated by aphasia. The apoplectiform attacks have a tendency to repetition.

This latter fact together with the transient character of the attacks makes the resemblance to the attacks in paresis very striking. Indeed errors of such diagnoses have been made.

3. **Sensory Disturbances.**—Abnormal subjective sensations, as tingling, cramps and sharp pain of neuralgic nature, are quite frequent. Headache is not infrequent. Neuralgia is in the domain of the fifth nerve. Hypæsthesia or hyperæsthesia are occasionally observed. Anæsthesia is rare.

Among the **rare** symptoms of multiple sclerosis may be mentioned:

(1) Vertigo; Charcot observed it quite frequently at the onset; (2) bulbar symptoms, viz. difficulty of swallowing, acceleration of pulse, atrophy of the tongue, palsy with atrophy of facial muscles; (3) muscular atrophy (without RD.), (4) spasmodic and involuntary laughter, (4) mental feebleness, (5) disturbances of the sphincters, (6) trophic disturbances of the skin and articulations, (7) glycosuria and polyuria.

Forms of Multiple Sclerosis.—Not all cases present the typical picture described above. In some cases the chief symptoms are either absent or very slightly manifest. Charcot called such cases "formes frustes." According to the seat of the lesions multiple sclerosis may be: **cerebral, spinal** or **cerebro-spinal.** Each of these may present varieties depending upon the level at which the lesion is found. Thus in the brain it may be: cerebral proper, cerebellar, pontine, bulbar, basal. In the cord it may be: cervical, dorsal, lumbar, sacral, or else according to localization on the transverse section it may be: posterior tract form (ataxic), postero-lateral form (spastic ataxic), lateral tract form (spastic), poliomyelitic form; syringomyelitic form.

Oppenheim has called special attention to several uncommon types, viz. **cervical, sacral, pseudo-tabetic** and **ocular.**

Cervical.—It is characterized by ataxia of the upper limbs, anæsthesia in hands and fingers, astereognosis. Sense of touch alone is involved, but not pain and temperature. The tendon reflexes of arms cannot be elicited. In the lower limbs spastic phenomena are present. Pathologically there is multiple sclerosis in the posterior columns.

Sacral.—Symptoms of bladder and rectum are prominent. Sensory symptoms are present in the anogenital region. Achilles and anal reflexes are absent. The disease runs in remissions.

Pseudo-tabetic.—It presents the symptoms of tabes except Argyll-Robertson pupil and optic nerve changes which are different from those in tabes. The disease runs an acute course unlike tabes.

In each of these three forms eventually in the course of the disease other symptoms of the main affection will make gradually their appear-

ance, but the symptoms just enumerated will throughout remain most conspicuous.

In the **ocular form** the disease may begin in and remain restricted to the optic nerves for years before other symptoms will begin to make their appearance. Involvement of the ocular motor nerves may be associated.

In the **cerebral form** the predominating symptom is hemiplegia. The latter may develop gradually, usually with remissions and exacerbations, or by apoplectic seizures. Hemiplegia following apoplexy usually disappears partially or even totally but has a tendency to recur: after several attacks it may remain permanent.

In the **basal or ponto-bulbar form** the conspicuous symptom will be crossed paralysis. In the **cerebellar form,** titubation, nystagmus, asynergia are the most prominent symptoms.

Course, Termination, Prognosis.—The course of the disease is very irregular. It is usually chronic and progressive, but there may be stationary periods and even amelioration. Then again some external influence, as a cold, excesses, exertion, traumatism, an intercurrent infectious disease, may aggravate the symptoms. At the onset there may be either exclusively cerebral symptoms (vertigo, headache, apoplectic attack) or only spinal symptoms, especially spastic paraplegia, or else visual symptoms.

Prolonged amelioration is rare and recovery is still rarer. The disease may last from ten to twenty years, but also less than two years. Death may result from bulbar paralysis or from some intercurrent disease such as pneumonia.

Diagnosis.—**Tremor** from **mercurial** intoxication is similar in character to that of multiple sclerosis, but in the former affection the tremor may appear spontaneously, while in the latter only upon a voluntary act.

Paralysis Agitans will be recognized by its passive tremor which usually disappears upon a voluntary act.

Hysteria sometimes presents: vertigo, hemiplegia, tremor and difficulty of speech—all symptoms of multiple sclerosis. Buzzard says: disseminated sclerosis in its earlier stages is of all organic diseases most commonly mistaken for hysteria. The presence of sensory disturbances and of special stigmata will help considerably in making the diagnosis of hysteria. Westphal and others described a form of **pseudo-sclerosis,** in which except nystagmus all other symptoms of multiple sclerosis are present and from which patients make a complete recovery. The following symptoms are present: speech-defect, slowness in movements of the eyes and facial muscles, fixed expression, tremor, plus knee-jerks; psychic disturbances, apathy, sometimes delirium; finally apoplectiform attacks. The patho-

genesis of the disease is obscure. Nothing of significance could be found at autopsies. However, in Strümpell's cases a leathery consistence of the brain and a slight degeneration of the crossed pyramidal tract in the cord were found.

In cerebellar tumors there may be titubation, nystagmus and optic neuritis, all symptoms of multiple sclerosis, but the persistence of intense headache and vomiting characteristic of tumors, abscence of spastic paralysis, absence of remissions and recurrences will promptly decide the diagnosis.

In **Paresis** there may be disorder of speech, tremor and spastic gait, but the character of these disturbances is so decidedly different from those of multiple sclerosis that an error is hardly possible. The paretic speech is tremulous, but not syllabic. The tremor is constant and independent of voluntary effort; it is a fibrillary tremor evident chiefly in the tongue and lips. Besides, the mental symptoms of paresis will soon help to arrive at a conclusion. Positive Wassermann reaction and cytological examination of the cerebro-spinal fluid will render further aid in establishing the diagnosis of paresis.

The hemiplegic form of multiple sclerosis may sometimes be confounded with a cerebral **hemiplegia** caused by a hemorrhage or softening, but the tremor, nystagmus and scanning speech are absent in the latter. Moreover in disseminated sclerosis the apoplectiform attacks are usually transient and prodromal symptoms absent.

Etiology.—Traumatism is considered as one of the causes. I have personally observed several cases in which the symptoms began to develop shortly after a severe trauma. Oppenheim observed the disease after **intoxications** with metals and carbonic dioxide. P. Marie considers **infectious** diseases as a frequent cause. Strümpell believes that multiple sclerosis is a **congenital disease.** Syphilis (congenital) is also considered by some as a cause. A neuropathic heredity has been traced in some cases. The affection appears usually at the age of from twenty-five to forty-five and rarely in children.

Treatment.—Rest, nutritious food, avoidance of exertion and internally iodides, arsenic, silver, cod-liver oil, may be tried, but little can be expected from medications. If syphilis is suspected, salvarsan and mercurials may be tried. Electricity should be avoided, for fear of increasing the spasticity. For the latter warm baths, massage and passive movements are advisable. For treatment of apoplectiform seizures see "Apoplexy." Thiosinamin, fibrolysin, tiodine, which is thiosinamin-ethyl-iodide, have been used for absorption of sclerotic tissue. The last particularly has been tried in disseminated sclerosis. W. Murrell (*Med. Press and Circular*, 1909)

obtained very favorable results. In his cases nystagmus, speech, ataxia have all improved greatly. The drug is administered hypodermically in 0.20 to 1 c.c. Injections are made daily. In one of his cases Murrell used 134 injections. Local swellings with rise of temperature are observed but they disappear soon.

X-ray treatment has given satisfactory results to Raymond, Babinski, Marinesco, etc. Tremor, ataxia, speech, writing, also general health, all improved from repeated séances of exposure to X-rays.

SECONDARY AFFECTIONS OF THE SPINAL CORD

I. Traumatic lesions of the cord (concussion, contusion, sudden compression).

II. Slow compression of the cord (tumors, caries of the vertebræ, Potts' disease).

I. Traumatic Lesions of the Cord

A trauma of the spinal column, whether it is a fracture, dislocation or else a blow, a shock of any sort (a fall on the feet or on the buttocks) may be followed by an injury of the cord itself. The latter under these circumstances may undergo a **concussion,** a **contusion,** a **sudden compression, laceration** or a complete **severance.**

A. **Concussion.**—The modern investigations tend to prove that material changes may occur in the cord in so-called concussion. Rupture of small blood vessels, destruction of axis-cylinders, some rarefaction in the protoplasma of cells, have been found in some instances.

Symptoms.—Immediately after the accident the following condition may be observed: paralysis of all or only the lower extremities; the reflexes are lost, the sensations are diminished and the sphincters are disturbed. Soon, however, all these symptoms begin to improve and gradually the patient recovers all the functions.

The chief characteristic of concussion is its transitory nature. But if in some cases the above symptoms remain, evidences of an organic lesion of the cord are then present. In every case of concussion of the spinal cord, hysteria should be thought of. Charcot had shown long ago that disturbances following railroad accidents, particularly the so-called "**railroad spine,**" belong to traumatic hysteria. Stigmata of the latter should be looked for in such cases.

B. **Contusion.**—It presupposes a trauma of greater severity and accordingly the symptoms are more pronounced. It is, however, difficult to

draw a sharp line between concussion and contusion. In both cases some lesions mentioned above have been found in the cord and in both cases recovery may follow. The main clinical difference between the two lies in the longer duration, in the greater severity and in the greater possibility for the symptoms to remain permanent.

It is important not to lose sight of this fact that an accident, whether it is a concussion or contusion, may be the exciting cause for development of chronic organic diseases of the cord, provided there is a predisposition or when the latter preëxisted in a latent stage. The injury in such cases hastens the progress of the dormant preëxisting affections.

C. **Sudden Compression, Laceration and Severance of the Cord.**—Fracture and dislocation of the vertebræ are the causative factors in this condition.

Pathology.—The state of the cord is various according to the intensity and the suddenness of the injury. Sometimes the cord at the level of the traumatism is reduced to an unrecognizable soft mass; above and below that level the nervous tissue gradually undergoes degenerative changes: chromatolysis of cells and ascending or descending secondary degeneration of tracts of fibers. Should a secondary infection occur, an abscess is likely to develop in the cord.

When the traumatism of the cord is less severe, only a hemorrhage may occur either in the cord itself or in the membranes. Rupture of the roots or of the spinal ganglia may also take place. The secondary changes above and below the injured area will develop like in the first case, but to a less extensive degree. It should be borne in mind that in injuries of the cervical and upper dorsal segments the cord alone may be injured, because the roots at those levels leave the cord transversely. In injuries of the lower dorsal and lumbar segments there is usually a simultaneous involvement of cord and roots, for the latter have an oblique direction.

Symptoms.—From the foregoing remarks it can be seen that the symptomatology will vary in each case. In injuries of the cervical and upper dorsal portions of the cord there may be only cord symptoms, while in injuries below that level there will be both cord and root symptoms. A fracture or dislocation of the vertebræ will produce a deformity of the spine which can be recognized by the usual tests for surgical diagnosis. It must, however, be borne in mind that no deformity may be observable when a fracture-dislocation occurs at the level of the fifth or sixth cervical vertebra. The same may occur in tuberculous, non-suppurative caries of the vertebræ in the lumbar region. In such cases skiagraphy is of great aid. It can accurately determine the nature and the seat of the trouble. As to the condition of the nervous system, there is a distinction

to be made between a simple sudden compression or a severe crushing of a segment of the cord produced by a fragment of the bone or by a dislocated bone. In both cases **paralysis** will be the most conspicuous symptom. When the cord is severely injured, the paralysis will be **flaccid** and **absolute** and the **reflexes** totally **abolished.** In simple compression, on the contrary, the **paralysis is spastic** and the reflexes are increased.

The **sensations** will also differ in both cases. In **severe injuries,** when the cord is totally lacerated or crushed at the point of the injury, the parts **below** the lesion are totally **anæsthetic** to all forms (touch, pain and temperature) and even the subjective sensations are abolished. Immediately above the anæsthetic area there is a hyperæsthetic zone which is due to an irritation caused by the traumatic focus. In **incomplete injuries,** in simple compression **pain** will be present in the limbs and follow the course of the nerves. It is sharp, lancinating in character. The patient will also complain of cold, heat, tingling or other abnormal sensations. The objective sensibility is also disturbed: there is a **diminution** of all the senses or sometimes a dissociation (see Syringomyelia). In localizing the seat of the lesion it should be borne in mind that the upper border of the **anæsthetic** area is not at the level of the diseased vertebra, but it is decidedly lower (see Anatomy and Fig. 92).

The **sphincters** are involved. Retention and constipation are frequent.

Trophic disturbances are frequent. **Bed-sores** appear rapidly at points of pressure (sacrum, trochanter, heels). Œdema, eruptions, hydrarthrosis, muscular atrophy, are not infrequent complications.

Injuries to the **upper cervical region** deserve special mention. Death may be sudden (involvement of the phrenic nerve). If the patient survives, there may be paralysis of the muscles supplied by the cervical nerves, bulbar symptoms, radiating pain in the area of distribution of the cervical nerves. Erection of the penis, which may be persistent, is a common symptom in injuries at this level of the cord.

In injuries of the lower cervical and upper dorsal segments of the cord, besides the symptoms described in the general symptomatology, there are special ocular signs, viz. myosis and narrowing of palpebral fissures.

Course, Termination, Prognosis.—If the cord is totally destroyed at the level of the injury, recovery will never follow: a myelitis will be the consequence, and the patient may die from bed-sores or cystitis with ascending urinary infection. In cases of mild or incomplete compression, ascending or descending degeneration will be the result and the paralytic symptoms are permanent. In injuries of the cervical region death may be instantaneous. Dislocations bear a less grave prognosis than fractures.

In some cases after prompt surgical intervention recovery had been observed.

Treatment.—When a sudden compression of the cord occurs, the patient must be handled with extreme care, as any additional movement is likely to increase the compression. Surgical intervention (laminectomy) is the only treatment in such cases. As it is sometimes difficult to tell the degree of the damage done, it is advisable to postpone the operation for some days and during that time observe the paralytic symptoms. When there is no tendency to improvement, a prompt operation is indicated. No operation is necessary in cases of progressive improvement. In cases of an evident fracture of vertebræ, a prompt intervention is urgent.

In order to determine the exact area of the injury in the cord and thus outline the level at which an operation is to be performed, there are two means at our command. First, an exact and thorough study of the sensory and motor disturbances (see Anatomy, Fig. 92). Second, an X-ray examination of the spine.

Surgeons are divided as to the time of interference: some are in favor of an early operation, others operate not before a few weeks have elapsed after the injury. I have seen favorable and unfavorable results in both cases. The following rules, however, may be kept in mind as a guide.

Early Intervention is indicated: when the nervous disturbances may be attributed to a fracture of an arch of the vertebra; when skiagraphy shows the presence of the bullet (in case of fire arms) in the spinal canal; when in case of fracture with dislocation a bloodless reduction of the dislocation has failed and the patient presents signs of a partial lesion.

Late Intervention is indicated: when early intervention was not done and the nervous symptoms do not improve spontaneously; when in the course of a partial lesion there is an ulterior aggravation of symptoms (compression by a callus). In cases of total lesion an operation is contraindicated. The comparative value of early and late operations can be seen from Chipault's statistics: in early operations he obtained 6 per cent. recoveries, 6 per cent. improvements, 8 per cent. with no change and 79 per cent. deaths. In late operations he obtained 8 per cent. recoveries, 27 per cent. improvements, 39 per cent. with no change and 25 per cent. deaths.

Some claim that hemorrhages into the spinal canal are not an indication for laminectomy. I have seen favorable results following prompt operations in such cases.

As to the irremediable conditions, such as paralysis, spasticity and sphincter disturbances, the treatment is the same as in the systemic diseases of the cord (see those chapters).

II. Slow Compression of the Cord. Tumors. Caries of Vertebræ—Potts' Disease

Etiology.—The cord may undergo slow compression from **tumors** or from deformed and **displaced vertebræ.** Tumors may originate in the cord itself, in the meninges, in the perimeningeal cellular tissue or in the spine. Displacement of the vertebræ occurs frequently in caries of the bony structure of the spine—in Potts' disease. Here the changes in the cord are due not only to the direct pressure of the deformed vertebræ, but also to the tubercular pachymeningitis created by the vertebral disease.

Pathology, Tumors.—(1) Those of **the cord** itself are not infrequent. Thus Schlesinger found that among 302 tumors of the cord 125 were within the cord substance. According to Flatau the relative frequency of extra- and intramedullary tumors is as 2.6 to 1. **Tubercle** is the most frequent of all. Its favorite seat is in the enlargements of the cord. It frequently coincides either with tuberculosis of the vertebræ or of the lungs. It originates from the blood vessels. It presents a hard mass which may undergo softening in the center. Around it the nervous tissue, which is under pressure, proliferates and secondary degeneration develops. Gummata are not very rare. **Gliomatous** tumors of the cord have been described in the chapter on Syringomyelia. (2) **Meningeal** tumors are usually benign in character. They originate on the inner surface of the dura. The most frequent is **sarcoma.** Other tumors are: psammoma, myxoma, fibroma, syphiloma, tuberculoma, lipoma and echinococci. The latter two develop externally to the dura. (3) In the perimeningeal cellular tissue in addition to the above tumors carcinoma has been observed. (4) Tumors of the vertebræ are carcinoma and sarcoma. The first is usually metastatic (from cancer of stomach or other organs). The second originates in the surrounding tissue. The vertebræ thus affected are soft; the spinal column is bent and thus compresses the roots. **Hydatid cysts** are rarely met with in the vertebræ or in the cord; they originate from the intra- or perispinal tissue. The majority are formed in the posterior mediastinum or in perivertebral muscles. They invade the spinal canal through the intervertebral foramina. Exceptionally the vertebræ and cord remain intact. Usually there is scoliosis and compression of the cord and roots; the destruction of the latter is very severe. Early laminectomy is urgent. (5) Tumors may also originate in the roots and in their meningeal coverings. Multiple fibromata are localized in the roots (neurofibromatosis) and they are most frequently found in the nerves of the cauda equina. The posterior roots are oftener involved than the anterior ones. Solitary fibromata are not accompanied by diffuse

neurofibromatosis; their point of departure is the dura. The most frequent seats of meningeal tumors are the dorsal region and the cauda equina.

The state of the nervous tissue in cases of slow compression by a tumor lying externally to the cord is as follows: The cord is flattened or presents a depression. It is pale or else congested. At the level of compression the myelin is broken up, the axis-cylinder disappears; instead of it neuroglia proliferates and around the blood vessels connective tissue prolifer-

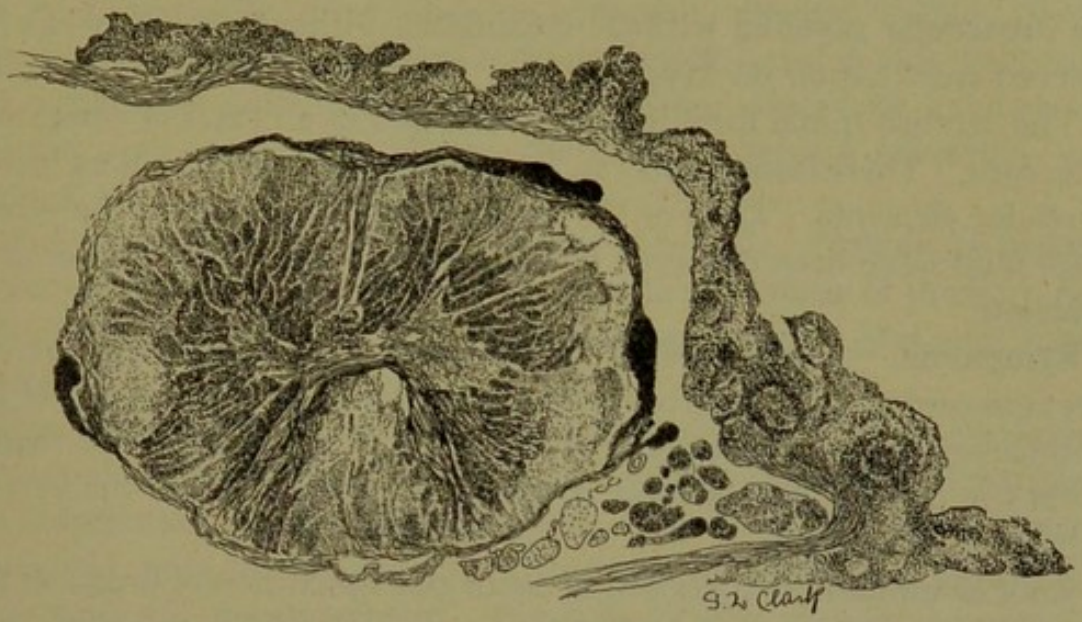


FIG. 101.—TUBERCULAR PACHYMENINGITIS. A CASE OF TUBERCULOMA OF THE CORD. (*Original.*)

ates; the affected segment is therefore in a state of sclerosis. Above and below this level ascending and descending degeneration develops. The cells of the gray matter at the point of pressure are either in a state of atrophy or completely absent. The meninges are thickened and adherent. Primary tumors of the cord and meninges, even the most malignant ones, do not produce metastasis. When they originate in the vertebræ, metastases are not rare.

In **Potts' disease** the destructive tubercular process of the bone (pus and fungosities) perforates the posterior vertebral ligament and reaches the dura; the latter becomes inflamed and proliferates (**pachymeningitis**), The tubercular masses covering the dura undergo softening, form small purulent collections at first, then later constitute a large abscess. On the other hand the tubercular masses may become organized, then sclerosed. The thickened dura is found then surrounded by a fibrous mass. Whatever the process may be, the roots and the cord undergo compression. The changes in the cord are identical with those described above. They

are generally localized at the level of the peridural inflammation with secondary degenerations. Inflammation of the pia-arachnoid is exceptional and may be due to a toxic infiltration along the roots. The involvement of the cord is explained in two ways: (1) mechanical action upon the cord, roots, vessels and lymphatics, and (2) the action of tuberculous toxins. An excellent illustration of the latter possibility is found in one of my cases (*J. of Nerv. and Ment. Dis.*, 1904), in which the cord was found to be entirely free in the vertebral canal and yet most extensive tubercular changes were observed in the cord. A primary parenchymatous **tubercular myelitis** without meningitis, without caseation has been observed (see chapter on Myelitis).

The cerebro-spinal fluid is always modified in all cases of compression of the cord. There is frequently an increase of albumen without an increase of cellular elements. Cells of the nature of the neoplasm in the cerebro-spinal fluid have been observed in the course of secondary cancer of the meninges.

Symptoms.—A compressing body, whatever its nature may be, may affect the cord and the roots or each individually. As to symptoms of bony involvement (in cases of tumors of the vertebræ), they are: local swelling, curvature, œdema, rise of local temperature, tenderness of the vertebræ and spontaneous pain.

Root Symptoms.—Irritation of roots and spinal nerves produces **pain,** which is unusually intense and of a neuralgic type; it is continuous, but presents paroxysms of exacerbation brought on upon the slightest movement. As pressure is at work, the nerves are in a state of neuritis. This is the reason of the accompanying **trophic** (eruptions) and objective **sensory disturbances** (anæsthesia) in the areas of their distribution. Gradually loss of power and atrophy develop. The patient then presents a **painful paraplegia.** The latter is frequently observed in carcinoma of the spinal column. When the motor symptoms become pronounced, the pain usually subsides. The seat of the pain is of an important localizing value. It is certain that the pain refers to the seat of the lesion or below it, but never above it.

Cord Symptoms.—They consist essentially of **motor and sensory paralysis.** Gradually but progressively a loss of power becomes established in that portion of the body which lies below the lesion. The paralysis may be **flaccid** at the beginning and become **spastic** later or be flaccid or else spastic through the entire course of the disease. **Spastic paraplegia** is the most frequent. The reflexes are abolished in the first and increased in the second form. In the latter case there may be also: ankle-clonus, Babinski's sign, Oppenheim's and paradoxical reflexes. The **objective**

sensibility is usually altered. There may be complete **anæsthesia** to all forms of sensations or a sensory dissociation of syringomyelic type; finally there may only be a diminution of sensations (**hypæsthesia**).

Babinski in 1899 and 1911 (*Rev. Neur.*) called attention to a form of spastic paraplegia without degeneration of the pyramidal tract. Clinically there is contracture of the **legs in flexion,** knee-jerks are not exaggerated, but the cutaneous reflexes are so much increased that stimulation of the skin of the paralyzed limb produces rapid flexion (defense movement). Since then the phenomenon has been observed in Potts' disease and it is due to a gradually progressing compression myelitis without marked degenerative changes. Consequently prompt removal of this compression may restore the patient to health. Sicard and Gutmann have recently reported such a case (*Rev. Neur.*, 1912). The defense reflex may be of practical value.

The **sphincters** are most of the time involved. **Trophic** disturbances are not infrequent. Bed-sores are formed in the points of pressure of the body (sacrum, gluteal regions, trochanter, malleoli). Muscular **atrophy** is frequent.

The **general symptoms** just described vary according to the segment of the cord involved. Moreover certain regions present **special symptoms.**

Compression of the Cervical Cord.—(a) When the **upper segment** is involved, **pain** will be present in the neck and shoulder, which at the same time will be anæsthetic. Paralysis of the muscles covering this region will produce an inability to rotate, flex or extend the head. All four extremities are in a state of spastic paralysis, the upper more than the lower. The sensations of the limbs are also involved. The special symptoms are: **slow pulse, paralysis of the phrenic nerve** (hiccough, vomiting, disturbance of respiration). The compression is at the level of the first four cervical segments which correspond to the first three cervical vertebræ.

(b) When the **lower cervical segment** is involved there are: pain in the arms, paralysis of all four extremities, muscular atrophy, objective sensory disturbances. The latter present the following distribution. When the lesion is at upper portion of the cervical enlargement the anæsthesia is at the level of the base of the neck. When the lesion is lower, the upper border of the anæsthesia descends to the upper limbs and is distributed in longitudinal bands parallel to the axis of the limbs. The special symptoms consist of **myosis and narrowness of the palpebral fissure.** When the compression is at the level of the fifth, sixth and seventh cervical roots, the atrophy will affect the muscles of the roots of the upper limbs (**Erb's type**): deltoid, biceps, brachialis anticus, supinator longus,

scapular muscles. When the eighth cervical and the first dorsal roots are under pressure, the atrophy will affect the muscles of the hands (**Klumpke's type**).

Compression of the Dorsal Cord.—The pain will be present around the trunk and the upper line of the anæsthesia will determine the level of the compression. The paralysis will affect the muscles of the trunk below the lesion and of the lower extremities. It may be flaccid, especially in complete destruction of the cord, or spastic. In the first case the reflexes are abolished, in the second exaggerated; the latter may be accompanied by ankle-clonus, Babinski's sign, Oppenheim's and paradoxical reflexes. The sphincters may be involved, and the more pronounced the more it approaches the lumbar enlargement.

Compression of the Lumbar Enlargement.—Pain in the lumbar region, in the abdomen below the umbilicus and especially along the crural and sciatic nerves; flaccid or spastic paralysis with lost or increased reflexes respectively; (the paralysis is flaccid in cases of complete interruption of the cord; spastic paralysis is in the incomplete lesions); **constant** involvement of the sphincters; anæsthesia in the paralyzed portions of the body—these are the symptoms observed. The last symptom deserves special mention. The upper border of the anæsthesia does not correspond to the level of the compression because of the oblique direction of the roots; it is therefore higher than the point of compression. The cremasteric reflex is lost. Achilles reflex is preserved. Atrophy is not observed in slight compression but marked in deep compression.

Compression of the Sacral Cord.—The pain is chiefly in the area of distribution of the sciatic nerve. Paralysis affects the gluteal muscles, those of the posterior aspect of the thighs, of the legs and of the feet. Deformities of the feet are constant and they depend upon the groups of muscles the most affected (equinovarus, or valgus). The knee-jerk which depends upon the integrity of the second, third and fourth lumbar segments are normal or increased. The Achilles' tendon reflex, which is controlled by the fifth lumbar and first sacral segments, is lost. The sphincters are involved. Anæsthesia occupies the postero-external aspect of the thigh, leg, foot, the gluteal and sacral regions. Muscular atrophy is present in advanced cases.

Compression of the Conus Medullaris and Cauda Equina.—This condition is described in the chapter on Diseases of the Conus.

Course, Termination, Prognosis.—The disease usually develops slowly and progressively, with vague painful and paretic symptoms. The clinical aspect will vary according to the localization of the tumor or of Pott's lesion. The most frequent form is that of spastic paraplegia, but

the disease may simulate any form of spinal cord lesion. When the tumor is of syphilitic nature, marked improvement in the symptoms is observed, if specific treatment is energetically administered. In Pott's disease the symptoms of caries of vertebræ may exist long before cord symptoms appear. Sometimes the cord symptoms appear long before deformity is observed. In Pott's disease the prognosis is more favorable when the motor symptoms are unaccompanied by marked sensory changes than when the latter are present. It is unfavorable when flaccidity of the limbs follows spasticity.

Death may ensue from bed-sores, ascending urinary infection or in case of a malignant tumor (cancer) from generalization of the latter; finally from some intercurrent affection. In Pott's disease the patient may die from extension of the tubercular process to the lungs. Recovery may sometimes follow in Pott's disease, the abscess discharging externally.

The **prognosis** depends greatly upon the segment of the cord involved. When the compression is in the cervical region, rapid death may follow. Tumors of the vertebræ which are almost always malignant, are more serious than tumors situated in the spinal canal. Among the latter the intra-dural are more benign and longer tolerated than the extra-dural. The prognosis in spinal tumors as a rule is very serious if surgery does not intervene.

Diagnosis.—In its **early period,** before the paralytic symptoms are marked, the diagnosis is quite difficult. The pain may be confounded with simple neuralgia (intercostal or sciatic) or lumbago. Charcot said that **bilateral pain** is frequently of spinal origin. A careful examination of the spine may reveal a deformity and lead to the diagnosis.

In cases of vertebral **cancer** and **Pott's disease** a deformity of the spine is almost always present. The first is mostly met with in old age; the deformity is round, not angular; the pain is the predominant symptom and cachexia is marked. Cancer of the spine is usually secondary to cancer of the viscera. The latter is observed in young people; the deformity is angular; pain is present on pressure; there is cutaneous hyperæsthesia; the spine is kept rigid; subjective pain is but slightly marked; tuberculosis of other organs is frequently present. In some cases of Potts' disease there is no deformity of the spine; the latter may be slightly or not at all perceptible. Alquier out of fifteen autopsies has found eight where involvement of the spine could not be ascertained. In such cases rigidity of the spinal column, localized pain and spinal hyperæsthesia to heat will help to recognize the disease. A flaccid monoplegia or a band of anæsthesia indicates root involvement, while spastic paraplegia or anæsthesia below the lesion indicates that the cord is being compressed. The above mentioned contrac-

ture in flexion with the defense reflex may be of assistance in determining the degree of involvement of the cord.

In the **paralytic period** the disease should be differentiated from tabes, myelitis, multiple sclerosis, multiple neuritis and hysteria. In **Tabes** the ocular symptoms and the ataxia will decide the diagnosis.

Myelitis (diffuse) develops very rapidly; the motor symptoms appear at the beginning, pain is usually absent. In the transverse form the onset is usually acute, the paralysis is marked and the objective sensory disturbances are early.

In **Multiple Sclerosis** the absence of sensory disturbances, the presence of tremor, nystagmus and the special speech are characteristic.

In **Multiple Neuritis** the muscular atrophy appears very early; the nerve trunks and the muscles are tender on pressure; there is no spasticity and there is usually a history of intoxication (alcohol, lead, etc.).

In **Hysteria** there may be rigidity of the spine with hyperæsthesia, but the sphincters are intact. There is no muscular atrophy. The sensory disturbances are marked. Finally the hysterical stigmata, the sudden onset of the paralysis after an emotion, will help to decide the question.

Treatment.—In Potts' disease absolute rest and immobilization of the spine with orthopedic appliances are of great benefit. At the same time general hygienic measures with abundant nutritious food, also administration of iodides, iron, arsenic and phosphates should be kept up. Great care must be taken of the patient's skin, as bed-sores are easily formed, also of the bladder, as urinary infection is frequently the cause of death. If syphilis is suspected, mercurials with iodides should be used.

As to surgical means, there are two methods: one consists of breaking the bones and putting them in place (redressement of Calot), the other is laminectomy. The first has given in some cases satisfactory results, but the majority of surgeons report disastrous consequences. A modification of this method, consisting of extension, followed immediately by fixation of the spinal column, has given better results. Laminectomy has been also successful in some isolated cases. In case of abscess of vertebræ which can be determined from gradually increasing spasticity and contracture of the paralyzed limbs, also from the fever, the abscess must be exposed as promptly as possible by **laminectomy** or by a **postero-lateral operation.** The latter appears to give more satisfactory results. In *Arch. f. Klin. Chirurgie*, 1909, Wassiliev reports brilliant results by this procedure and he cites very unfavorable results from laminectomy. If the operation is performed very early when pachymeningitis and cord symptoms have not yet made their appearance, there is hope for curing the patient. When the disease is old and the tuberculosis of the vertebræ is cured, laminectomy is indi-

cated. Finally the existence or non-existence of the defense reflex (see above) may assist in deciding the question of operative interference.

The wisest procedure is to apply first the non-operative treatment, and if there is no improvement in the paralytic symptoms, an operation should be attempted. Cases of spontaneous recovery have also been reported.

In cases of **compression by tumors** the following considerations may serve as a guide. Cases of primary and isolated spinal tumors without angular deformity and without flaccid paralysis necessitate immediate intervention, especially when there are only root symptoms. It is better to perform a large laminectomy over four or five vertebræ than to wait for cord symptoms which cannot always disappear. Tumors accompanied by grave deformities of the spine, tumors that show rapidly developing cord symptoms, present a grave surgical prognosis. In such cases an operation can be undertaken only when an exact localizing diagnosis is made. In such cases the operation will be only palliative. In cases of flaccd paraplegia with loss of reflexes no operation should be performed. As to the seat of operation, the latter should be performed in cases of intra-dural and extra-medullary tumors at the level of the highest nerve-root involved. In view of the oblique direction of the roots emanating from the spinal cord it is important to remember that in an adult in order to determine the number of the roots at the level of a certain spinous process it is necessary to **add** to the number of the corresponding vertebra: one in the cervical region; two in the upper dorsal region; three in the lower dorsal region (sixth to eleventh vertebræ); the lower portion of the eleventh dorsal and the interspinous space immediately below correspond to the last three lumbar pairs.

The spinous process of the twelfth dorsal and the space immediately below correspond to the sacral pairs.

Below the second lumbar vertebra the cord is no more involved, but the cauda equina is (see Fig. 96).

Cancerous vertebræ should of course never be operated upon. The immediate danger of operation lies in shock or hemorrhage or infection. The mortality depends much on the seat and the nature of the tumor. In the cervical region shock especially is to be feared by reason of proximity of the medulla. In the dorso-lumbar region where because of the frequency of diffuse infiltration of the meninges and roots extirpation of tumors is very laborious, hemorrhages are frequent. Moreover the presence of vesico-rectal trouble and of extensive anæsthesia facilitates infection; the mortality therefore is high. In the dorsal region tumors are usually benign and strictly limited; their removal is quite easy. The mortality is not so great as in the other localizations. Tumors of bony

origin present a high mortality, hydatid cysts still higher. Almost complete recovery follows removal of fibromata or sarcomata of the meninges. Removal of tumors of cauda equina has given but mediocre results. Before an operation is decided upon it is advisable to try for a short time a thorough course of antisyphilitic treatment. Severe pain in non-operable cases must be controlled by opium.

CHAPTER XX

MUSCULAR ATROPHIES

Classification

The chief forms of muscular atrophies are:

I. Progressive muscular atrophy of spinal origin (Aran-Duchenne).
Ia. Progressive muscular atrophy of infants.
II. Progressive muscular dystrophy (myopathy), which embraces the following subdivisions:
  (a) Pseudo-hypertrophic type (Duchenne).
  (b) Scapulo-humeral or juvenile type (Erb).
  (c) Facio-scapulo-humeral type (Landouzy-Dejerine).
III. Primary neuritic atrophy (Charcot-Marie-Hoffman-Tooth).
IV. Arthritic muscular atrophy.

I. Progressive Muscular Atrophy of Spinal Origin (Chronic Anterior Poliomyelitis)

(Aran-Duchenne's Type)

Pathology.—The main lesion consists of a gradual degeneration, atrophy and disappearance of the cells of the anterior cornua in the spinal cord. Under the microscope the cells appear small, their Nissl's bodies have partly or totally disappeared, the nuclei and nucleoli are either on one side or outside of the cells, the yellow pigment is increased in amount. The number of the cells is considerably reduced. The prolongations forming at the periphery of the cord, the anterior roots suffer secondarily: they undergo degeneration and atrophy and appear therefore slender.

The degeneration of the roots is continued into the peripheral nerves, but to a lesser degree. The nerve-terminations in the muscles are distinctly degenerated. The muscular tissue also undergoes changes: granular degeneration transforms the fibers into indistinguishable masses, which are gradually absorbed; atrophy is the result. The disease is therefore an affection of the lower motor neurons. In cases with bulbar symptoms the nuclei of motor nerves are involved. The eleventh and twelfth are most frequently affected. The ninth, tenth, seventh and motor portion of the fifth may also be involved.

The above changes in the gray matter are accompanied by proliferated neuroglia tissue. The blood vessels are dilated and thickened. A primary degeneration of the nervous elements of the gray matter is the essential characteristic of the disease.

Symptoms.—The onset is characteristic. In the majority of cases the **small muscles of the hands** are first affected. The thenar muscles, the superficial first and the deep next, are first to be involved. The ball of the thumb then becomes flattened. The other small muscles, the lumbricales, and the interossei follow. The spaces between the metacarpal bones are deep, the prominences of the bones become marked.

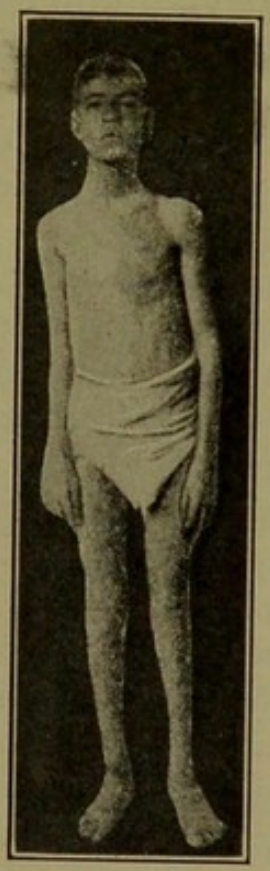


FIG. 102.

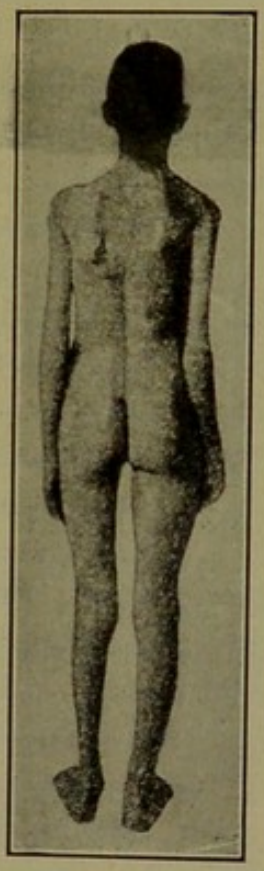


FIG. 103.

PROGRESSIVE MUSCULAR ATROPHY, FOLLOWING INFANTILE SPINAL PARALYSIS.

The first phalanges are in extension, while the other two are in flexion. The hand is **claw-like** (main en griffe). Gradually the atrophy spreads to the muscles of the hypothenar, of the forearm, arm, shoulder, neck, thorax and lower extremities (Figs. 102, 103, 104, 105, 106).

On the forearm the flexors are affected before the extensors, the extensors of the fingers before the extensors of the wrist. On the arm the atrophy of the deltoid muscle alters the round contour of the shoulder, the head of the humerus becomes evident. Atrophy of the scapular muscles brings out prominently the borders of shoulder blade; atrophy of the

serratus projects the posterior border of the scapula like a wing (Fig. 106). Atrophy of other muscles of the extremities of the neck and abdomen will also change the contour of the bones and produce corresponding functional disturbances. The muscles that frequently escape rapid wasting are: the latissimus dorsi, triceps of arm, highest portion of trapezius, sterno-mastoid.

The functional disturbances are first manifested in the hands. The patient's attention is attracted to an awkwardness in performing fine acts, such as writing, buttoning. When the atrophy advances, the patient becomes helpless and his attitude is quite characteristic when he attempts to do anything at all: he turns, tries to help himself with the entire body in an effort to do what the hands alone should have done. When the muscles of the neck are affected, the head cannot be held erect. Atrophy of intercostal muscles interferes with respiration, which then becomes exclusively diaphragmatic. Atrophy of the muscles of the lower extremities will interfere with standing and walking.

The other characteristic signs of the disease are: **fibrillary** or fascicular contractions of the affected muscles, also **reactions of degeneration.** The first can be observed almost continuously, and the least stroke intensifies them. They are probably due to an increased irritability of the cells of the anterior cornua in the cord.

The reflexes are usually diminished for want of muscular tissue. There are no changes in sensations or in the sphincters.

Course, Termination, Prognosis.—The description as given above is met with in typical cases, but the onset as well as the successive stages of the disease may vary. Although it is slow and progressive, nevertheless it may be arrested in its development at a certain period or for a certain time. It may last then many years, as long as fifteen or twenty. In other cases it may suddenly assume a subacute development and advance rapidly. Usually the patient dies from some intercurrent disease, as pulmonary tuberculosis. The disease may have also an ascending course and involve the nuclei of the medulla; the patient will die then from bulbar palsy.

The disease may also present an **atypical** onset: instead of beginning in the muscles of the hands, it may affect first the muscles of the arm or of any other portion of the body. The atrophy may begin in the lower extremities. In such cases the anterior tibial muscles are first affected and the atrophy gradually extends to the muscles of the thigh, etc. When the disease begins in the medulla, atrophy of the tongue appears first: difficulty of moving the tongue in every direction, flatness of its musculature, fibrillary tremor, difficulty in pronouncing words are the chief

symptoms. Other muscles follow: orbicularis oris, muscles of the soft palate and of the vocal cords. When this occurs, the speech becomes more and more indistinct, the voice is low, coughing is difficult, blowing with the lips is impossible. When the muscles of mastication (masseter, temporals and pterygoids) become affected, the patient cannot chew his food and the mouth does not close, swallowing is very difficult, food regurgitates through the nose and there is danger for food to enter the larynx; saliva dribbles constantly from the mouth. When the nucleus of the pneumogastric becomes involved, dyspnea and rapid heart action develop. When the disease begins in the medulla, the patient cannot live long and dies before atrophy appears in the limbs.

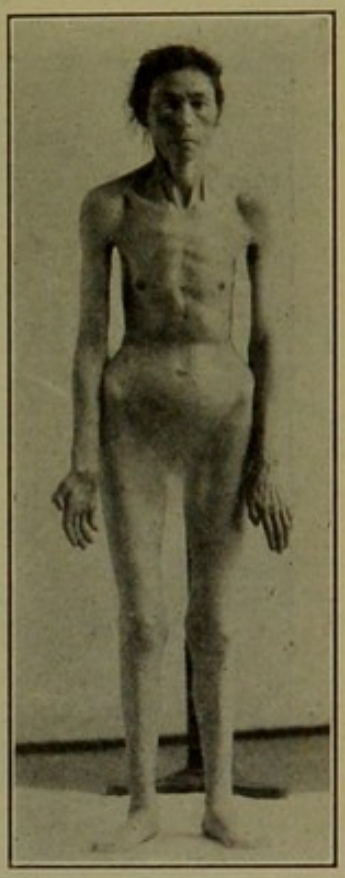


FIG. 104.—PROGRESSIVE MUSCULAR ATROPHY SHOWING INVOLVEMENT OF HANDS, ARMS, SHOULDERS, FACE AND LEGS.

The prognosis in all varieties of the disease irrespective of the mode of onset is always unfavorable.

Diagnosis.—Aran-Duchenne's disease is so typical in its onset, in its course and its symptomatology that in the majority of cases the diagnosis is not difficult. Thus it will be readily differentiated from muscular atrophies occurring in the course of other organic diseases, as myelitis, tabes, hemiplegia, etc. From **myopathies** it will be distinguished by the onset, fibrillary twitching and RD. In **syringomyelia** the sensory disturbances will decide the question.

Multiple Neuritis will be recognized by the presence of pain and paralysis, also by the absence of fibrillary contractions.

Hypertrophic cervical pachymeningitis presents a history of pain and the reflexes are usually increased.

Arthritic muscular atrophy affects the muscles only in the vicinity of a joint which was previously diseased.

Etiology.—Very little is known of the causes of this affection. Exposure to cold, trauma, exertion, have been reported as usual causes. Two cases came recently under my observation in one of which carrying daily large cakes of ice in the left hand for a period of three months was apparently the only accountable cause for the beginning of the atrophy in the same hand. The other patient sustained a blow with an iron bar over his shoulder. There was no erosion or any other apparent lesion. Shortly afterwards he noticed that he could not thread a needle and had difficulty

in buttoning his vest. Atrophy began to develop in the most characteristic manner.

Infectious diseases, intoxications, syphilis, have been also mentioned as causative factors. An attack of acute anterior poliomyelitis in childhood may become later in life the point of departure of progressive muscular atrophy (Figs. 102, 103).

The disease usually develops in middle life, more frequently in men than in women. In a few isolated cases it has been observed in several members of the same family (Ormerod, Holmes, Gee).

FIG. 105.—PROGRESSIVE MUSCULAR ATROPHY CONFINED TO UPPER ARMS, SHOULDERS AND THORAX.

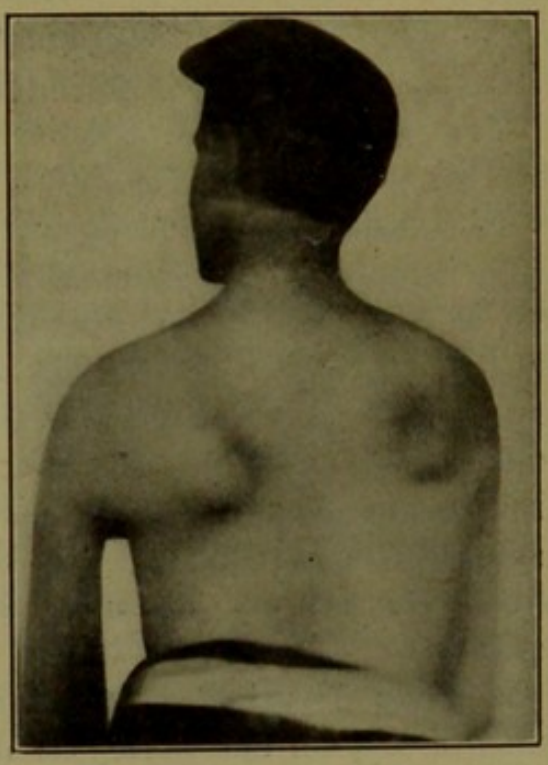


FIG. 106.—ATROPHY OF THE MUSCLES OF THE SCAPULA (Same individual as Fig. 105).

Treatment.—Massage and electricity (especially galvanism) are practically the only means of treatment. They may prevent rapid wasting. General hygiene, nutritious food and avoidance of stimulants are indispensable. Mercurials and iodides, tonics—arsenic, strychnine, phosphorus, may be useful in some cases.

Progressive Muscular Atrophy of Infants.—Werdnig (1891) and Hoffman (1893) described a variety of progressive muscular atrophy in infants which pathologically presents the picture of the disease just described, but clinically differs from it. The characteristic symptoms are as follows:

In the midst of good health the very young child begins to lose power. Soon develops a distinct paretic condition of the limbs and of the musculature

of the trunk, so that the child cannot sit up; the joints show a looseness; the muscular atrophy is generalized and symmetrical; there is some adiposity; the reflexes are absent, the limbs are flaccid; there are no sensory disturbances, no involvement of the sphincters or of the cranial nerves. What particularly characterizes this affection is its family character and its short duration, viz. from two to three years.

The disease is progressive and death takes place from some intercurrent disease, such as pneumonia, etc. The pathogenesis of the disease lies probably in a prenatal disturbance of the nutrition of the cells of the anterior cornua which eventually ends in their destruction. The disease is probably allied to Oppenheim's Myatonia congenita.

Amyotrophic Lateral Sclerosis

(Charcot's Disease)

Pathology.—The main lesion consists of atrophy of the cells of the anterior cornua of the cord and a sclerosis of the pyramidal tracts. This double lesion is found frequently in the corresponding portions of the

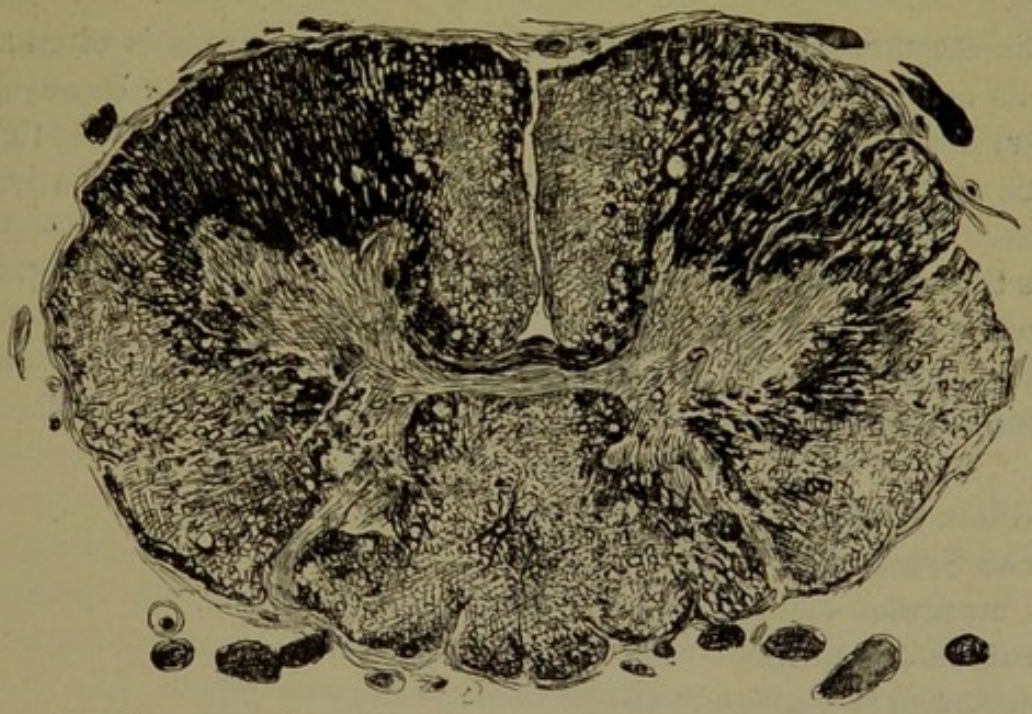


FIG. 107.—AMYOTROPHIC LATERAL SCLEROSIS. (*Original.*)

medulla, pons and brain. Thus a degeneration of the pyramidal tracts may be seen besides the cord in the subcortical tissue of the motor area, in the internal capsule, in the pons, in the medulla. The cells of the motor nuclei in the medulla may be affected at the time as the cells of anterior cornua. The nucleus of the hypoglossus is more frequently

affected; the facial, the pneumogastric and the motor portion of the trigeminus are not infrequently involved. The large pyramidal cells of the cortex of the motor area of the brain may be equally affected in advanced cases (Fig. 107).

The changes in the cord are more pronounced than in any other portion of the central nervous system. In view of the changes in the cells of the cornua, their prolongations, anterior roots and peripheral nerves must naturally suffer. Finally the muscles, in which the nerves terminate, also undergo degeneration and atrophy.

Taking a general view of the subject, one may say that in amyotrophic lateral sclerosis **two** systems of motor **neurones** are affected, viz. the **cortico-bulbo-spinal** and the **peripheral** or upper and lower. The first originates in the large pyramidal cells of the motor cortex, descends through the centrum ovale, internal capsule, crua, pons to terminate around the motor nuclei of the medulla, and especially around the cells of the anterior cornua in the cord. The second neurone consists of the cells of the anterior cornua (or their equivalents in the medulla, namely the cells of the motor nuclei) and of their prolongations, viz., anterior roots and peripheral nerves.

The degeneration of upper and lower motor neurons is often simultaneous and apparently independent of each other. The cause of this primary degeneration is still unknown. It is, however, certain that old organic diseases of the central nervous system may start the pathologic, process. Such a case I have observed (*Amer. Med.*, 1903).

As to the histological changes of the cells see the preceding chapter and of the fibers see the chapter on Primary Lateral Sclerosis.

Symptoms.—The main features of the disease are: **muscular atrophy** and **spasticity.** They may both begin simultaneously or one follows the other. As the nuclei of the medulla may also be affected, there will be in addition to the above symptoms also **bulbar** disturbances.

Cord Symptoms.—The lesion of the cells of the anterior cornua produces **muscular atrophy** and as the disease is chronic, the condition in every respect is that of Aran-Duchenne's Muscular Atrophy. A detailed description of it is given in the preceding chapter.

The lesion of the pyramidal tract produces **spasticity.** The latter is particularly observed in the lower extremities. The condition is that of spastic paraplegia. The gait is difficult because of the rigidity and the loss of power. The reflexes are increased. Ankle-clonus, Babinski's, Oppenheim's and paradoxical reflexes are also observed. When the upper extremities are involved, the arms are adducted to the trunk, the forearms pronated, the fingers in a claw-like position. The most frequent form of

the disease is: spasticity with weakness in the lower limbs and Aran-Duchenne's atrophy in the upper limbs, but there may be other varieties according to the portions of both systems of neurons most markedly involved.

Bulbar Symptoms.—They are identical to the labio-glossolaryngeal paralysis of Duchenne. A detailed description of the latter was given in the chapter on bulbar paralysis, in which its relation to amyotrophic lateral sclerosis was also discussed. (See also preceding chapter.)

Cerebral Symptoms.—In advanced cases in which the cortical cells and the subcortical white substance are involved, mental symptoms are observed. The patient is emotional and mentally feeble. There is a tendency to laughing and crying.

Course, Termination, Prognosis.—As the disease may affect at the beginning each system of neurones (see above) separately, the onset may be various. When the upper extremities are first affected, the muscular atrophy will be the initial and remain as the most conspicuous symptom. When the lower extremities are first involved, spastic paraplegia will be the most noticeable symptom.

In some cases the disease may begin in the medulla and remain as the most prominent symptom for a long time, as one of my cases shows (*New York Med. Jour.*, 1907).

The disease may last for years, if the bulbar symptoms are late to appear. The prognosis is invariably bad.

Diagnosis.—From **Progressive Muscular Atrophy** of spinal origin the disease will be differentiated by the addition of spasticity and modification of the reflexes. **Multiple Sclerosis** is recognized by its typical speech, nystagmus, tremor.

Amyotrophic lateral sclerosis with **bulbar onset** should be differentiated from diseases of the medulla (see this chapter).

Etiology.—Cold, trauma, exertion are mentioned as causes. It is a disease of middle life. In the case reported by me the boy was only fifteen, and the disease was far advanced. (*Amer. Med.*, 1903.)

Treatment.—It is only palliative. Proper hygienic and dietetic measures, and avoidance of stimulants should never be neglected. To relieve the spasticity, gentle massage and warm baths will be useful. Iodides and mercurials may be tried. Electricity and strychnia must be avoided.

II. Progressive Muscular Dystrophy. Myopathies

Pathology.—The lesion is in most cases confined to the muscular tissue. The nervous system is in the largest majority of cases intact.

In some cases atrophic (not degenerated) cells were found in the anterior cornua of the spinal cord. This condition may be secondary to the muscular changes. The peripheral nerves, also some fibers in anterior roots, have been found altered in a few cases. The muscles are pale and atrophied; their individual fibers are reduced in size and number; they become irregular and the striation disappears. The **connective tissue** between the fibers is proliferated to a considerable extent (hyperplasia)

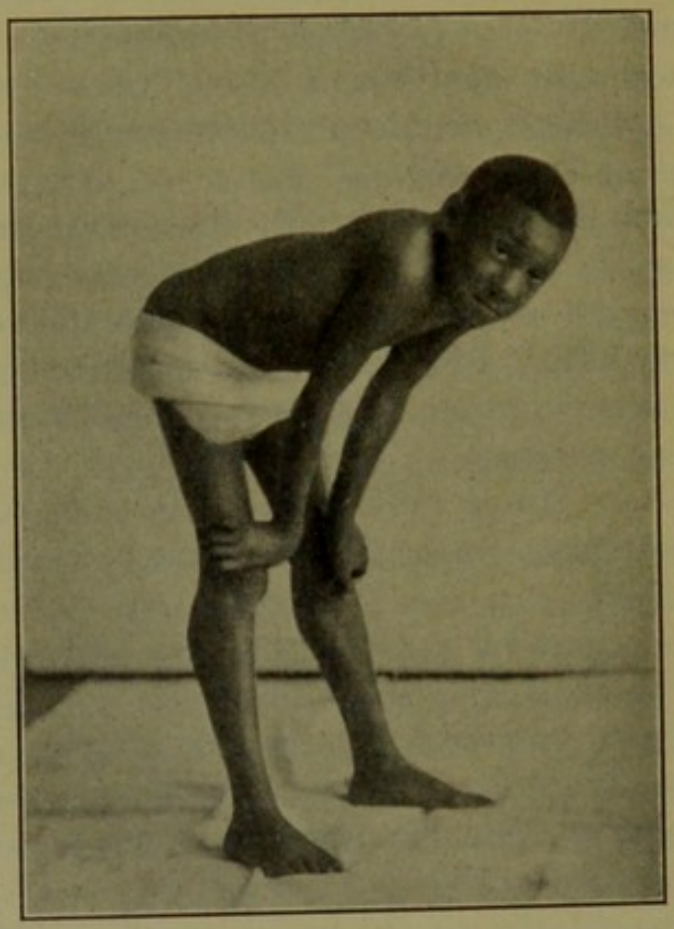


FIG. 108.—PSEUDO HYPERTROPHIC PARALYSIS. (Climbing on his four.)

and contains sometimes a large amount of fat cells. In some cases the muscular fibers instead of being atrophied are **hypertrophied.** In this form some fibers hypertrophy at first, but soon they atrophy and disappear, they are substituted by increased connective tissue or fat. Atrophic and hypertrophic varieties depend on the amount of connective tissue or fat respectively. The disease is a primary muscular affection independent of changes in the cord or nerves.

Symptoms.—(*a*) **First Variety: Pseudo-hypertrophic Type.**—It occurs usually in **young** children. The first sign of the oncoming trouble is noticed in walking. The child falls often and gets tired easily. Upon examination the size of the legs is found to be out of proportion with their

function; while the calf-muscles are enormous, their feebleness is striking. On palpation the muscles are either firm or soft according to whether connective or fat tissue respectively predominates. Of other muscles of the lower extremities the extensor of the knees and the gluteus maximus are particularly enlarged. As the atrophy spreads to the pelvis and abdomen, the patient has more and more difficulty in walking; in the latter act the pelvis moves markedly up and down; in rising from a seat the patient has to place first his hands on his thighs and help himself to get up. A very **characteristic sign** of this affection is the manner in which the patient leaves a recumbent position. He turns to either side with effort in his first attempt to sit up; then he tries to put himself in a kneeling position. After the latter is reached, he places his hands on his knees and then after several attempts he gradually gets up, supporting himself continuously with his hands, which slowly slide up the thighs until the erect position is obtained. (He climbs on his four, Fig. 108.)

The atrophy invades gradually the upper portions of the body. At this time **lordosis** of the lumbar spine and protrusion of the abdomen develop. The patient's gait is similar to that of a duck. As the disease is essentially progressive, the muscles of the thorax and of the upper extremities are gradually involved: the bones become prominent, the shoulder-blades move abnormally with each movement of the arms. The triceps of the arm is usually hypertrophied, the biceps is atrophied. The muscles of the hands usually escape.

The loss of power has this characteristic feature that it develops simultaneously and in proportion with the muscular wasting. The reflexes decrease also parallel with the degree of atrophy. Fibrillary contractions are absent. The electrical reactions are only diminished quantitatively, but there is no RD. Sensations are intact. The sphincters are not affected.

(*b*) **Second Variety: Scapulo-humeral or Juvenile Type (Erb).**—In this form the myopathy makes its first appearance in the muscles of the shoulder and upper arm, especially trapezius, serratus magnus, biceps and triceps. There is no hypertrophy. Gradually the atrophy extends to the muscles of the upper and lower extremities, also of thorax and pelvis. The disease occurs in early youth. Electrical reactions are only diminished but there is no RD. There are no fibrillary contractions.

(*c*) **Third Variety: Facio-scapulo-humeral Type (Landouzy-Dejerine).**—The atrophy affects first the muscles of the face and particularly the orbicularis oris. In a well-developed case the mouth is enlarged, the occlusion of the eyelids is incomplete; the facies is expressionless. The patient is unable to blow, whistle and pronounce labial letters.

Gradually the myopathy extends to the shoulders and arms. The rest of the musculature is invaded later in the course of the disease.

The affection makes its first appearance in adults. Electrical reactions are only diminished, but present no RD. Fibrillary contractions are absent.

Other Varieties of Myopathy.—The three forms of myopathy just described received their names from the mode of onset or from the predominant seat of the atrophy. Observations show that there may be other varieties, as for example **Zimmerlin's,** in which the myopathy commences in the upper musculature of the thorax and arm; **Eichorst's,** in which the femoro-tibial muscles are first affected. It is useless to multiply the names. Gowers in 1902 described a form to which he gave the name of **"Distal Myopathy."** It is characterized by atrophy confined to the muscles below the elbow and the knee.

Course, Termination, Prognosis.—Irrespective of the variations in its onset, the disease is essentially progressive in character. There are periods when the atrophy arrives at a certain stage of development and remains stationary for a certain time, but finally resumes its insidious course. As to its duration, observations show that the earlier in life the atrophy begins, the shorter is its duration. The pseudo-hypertrophic patients rarely reach above twenty. The ultimate results of all the cases are absolute loss of power and confinement to bed. Death usually results from some intercurrent disease (pneumonia, tuberculosis, etc.). Life is in danger when the respiratory muscles and the diaphragm are involved.

Diagnosis.—Generally speaking there is no special difficulty in making a diagnosis of myopathy by bearing in mind the essential features described above. There are, however, cases which present some obstacles in arriving at a positive opinion as to whether they belong to the myopathics or to progressive muscular atrophy of spinal origin. The **fibrillary contractions** and **reactions of degeneration** which used to be considered by the elder writers as pathognomonic of the latter, have been recently found to be not so constant. Moreover, there are undoubtedly cases in which some groups of muscles present the symptoms of one form, and other groups of muscles show symptoms of the other form of muscular atrophy (so-called **mixed type**). The pathological records of some cases also favor this view. The old border line can no more be considered as sharply defined in every case. There is a certain relation and affinity as to the origin between various forms of atrophy. However, for practical purposes it is a good plan to differentiate them according to the special symptoms described.

Etiology.—Little is known as to the causes of this affection, except

that it is met not infrequently in members of the same family. It is probably due to some congenital defect.

Treatment.—Proper hygienic and dietetic measures are beneficial. Locally massage and electricity are indicated. In administering the latter it should be borne in mind that muscles undergoing an atrophic process get easily fatigued. Violent electric contractions should therefore be avoided. Galvanism appears to have a better effect than faradism. As some cases of this affection present at the same time diminished or enlarged thyroid glands, administration of thyroid extracts or perhaps extracts of other ductless glands may be tried.

III. Primary Neuritic Atrophy (Charcot-Marie-Hoffman-Tooth.) Peroneal Type of Progressive Muscular Atrophy

Pathology.—The lesion consists of an involvement of the peripheral nerves, of the spinal ganglia, of the posterior roots and posterior columns in the cord and of the cells of the anterior cornua. The most pronounced changes are those of the posterior columns, especially of Brudach's. The latter is similar to that of tabes. Hoffman believed that the original lesion is in the peripheral nerves (**neuritis**), which has an ascending course and eventually involves the posterior roots and the posterior columns of the cord. Histologically the cells are in a state of chromatolysis and finally atrophy. The fibers undergo the same degenerative process with subsequent sclerosis, as in tabes. **The peripheral nerve-trunks show neuritis.** The **muscles** are **atrophied.**

Symptoms.—The disease begins in the majority of cases in the lower extremities, and especially in the peroneal group of muscles (extensors) and in the small muscles of the feet. The legs appear thin, emaciated. The contrast between the size of the legs, especially of their lower thirds, and the rest of the body is striking and quite characteristic. The gait is difficult (steppage). Foot-drop is evident. Deformities of the feet are very frequent (pes varus, equinus or equino-varus). Gradually the small muscles of the hands and of the forearms are invaded. A claw-like hand develops. The atrophy rarely involves the proximal ends of the extremities. It may extend and affect the muscles of the trunk and of the face, but this is rare (Fig. 109).

Fibrillary contractions are observed and **reactions** of **degeneration** are sometimes present. Diminished response to faradism and galvanism is common.

The **reflexes** are usually diminished or abolished.

Sensory disturbances (objective) are rare. Pain and paræsthesia may occur. In my case (see above) the disease began with excruciating

pain in the feet, but when the atrophy became pronounced, it gradually disappeared. **Trophic** disturbances (ulcerations) may occur.

The sphincters are intact.

Course, Termination, Prognosis.—It develops very slowly and may last an indefinite number of years. Life is not threatened. Remissions occur. Death usually occurs from some intercurrent disease (pulmonary or other).

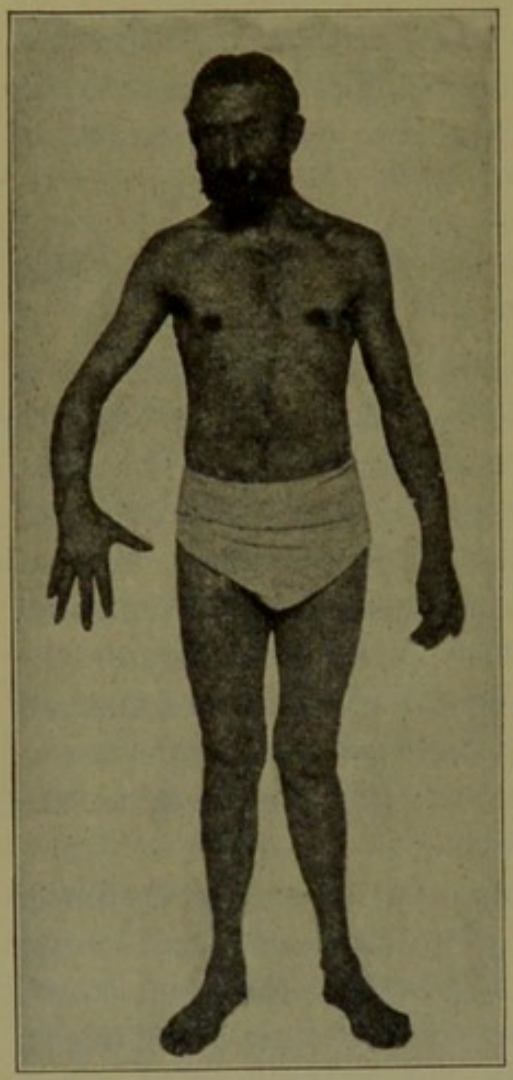


Fig. 109.—Primary Neuritic Atrophy.

Diagnosis.—In **multiple neuritis** the extensors of the legs are also frequently involved, but the sudden or rapid onset, the paralysis preceding the atrophy, the tenderness of the nerve-trunks, the etiology of the disease, will establish the diagnosis.

From **myopathies** the disease will be differentiated by the appearance of the atrophy in the distal ends of the limbs, by the fibrillary contractions and RD.

Dejerine and Sottas have recently described a disease presenting some analogy with primary neuritic atrophy. It is called **"Interstitial hypertrophic neuritis."** It is met with frequently in several members of the same family and it appears early in life. It is characterized by a **muscular atrophy** of the distal ends of the limbs, but it also presents a **tabetic symptom-group,** viz. ataxia, lancinating pain, Argyll-Robertson pupil. There is also a scoliosis or kyphosis. The peripheral nerves which are accessible to palpation are markedly **thickened** (hypertrophied). **Pathologically** it presents an interstitial neuritis in the limbs, also a certain degree of degeneration in the posterior columns of the cord.

Etiology.—The disease is of an hereditary character. Several members of the same family are frequently affected. Men suffer more than women. It is a disease of the second half of childhood. In one case reported by me (*Jour. Nerv. and Ment. Dis.*, 1903) the disease developed after a prolonged exposure to cold.

Treatment.—Massage and electricity, orthopedic appliances and tenotomy, are all the means we have at our command.

IV. Arthritic Muscular Atrophy.—It accompanies acute and chronic inflammations of the joints. It affects the muscles in the immediate vicinity of the joints and rarely extends to the entire limb in the acute and subacute forms of rheumatism. In chronic rheumatism the atrophy extends to the muscles lying at a distance from the diseased joint. In the first case there may be only one special muscle affected or else the group of muscles surrounding the joint. In the second case the atrophy becomes diffused and involves the entire limb. Generally speaking the degree of atrophy is in proportion with the duration of the arthritis and ankylosis of the joint. When several joints are involved, the generalized muscular atrophy may give the impression of Aran-Duchenne's amyotrophy. The differential diagnosis will show that the electrical reactions show only a quantitative diminution, but no reactions of degeneration. Some authors believe that the inflammation of the joint spreads to the neighboring nerves and the atrophy is then due to the neuritis. Others think that the atrophy is caused by ischæmia produced by the swollen joint. Others attribute the atrophy to a functional inactivity.

Finally a reflex action may play some rôle. There are some pathological evidences that the sympathetic system may be the underlying cause of arthritic muscular atrophy. The disease tends to disappear when the joint gets well.

Myatonia Congenita or Amyotonia Congenita

In 1900 Oppenheim called attention to a heretofore unknown condition which is characterized by a generalized muscular weakness without paralysis. It is congenital in nature. The cranial nerves are not involved. The intelligence and general health are intact. The condition has a tendeny to improve.

The symptoms are noticeable at an early age, viz. from birth. When the little patient is seated, the trunk bends forward, forming a marked kyphosis. Placed on his feet he is not only unable to stand alone, but, even when supported, his legs give way under him. That there is no paralysis can be seen from this fact, that when the patient is on his back he is able to move his arms and legs. The weakness (**atonia**) affects also the ligaments of various articulations, so that the latter can be placed in hyperextension and in all possible positions.

The muscles are soft but there is no atrophy. In the majority of cases the electrical reactions of the muscles are normal. In grave cases there is a diminished response or no response to electrical stimulation. The skin presents a certain thickness, reminding one of a myxœdematous

skin. The knee-jerks are usually lost. General sensations and special senses are normal. The sphincters are not involved.

The mentality is usually intact. The muscular atony is usually more marked in the lower extremities. but it may equally affect the thorax, neck and upper extremities. The muscles of the face, tongue, eyes, deglutition are not involved. The growth of the bones appears to be normal.

As to the nature of the condition little is known. The two autopsies placed on record (Spiller, *Univers. Penna. Med. Bull.*, 1905, and Baudoin, *Semaine Méd.*, 1907) reveal nothing characteristic for pathogenic in-

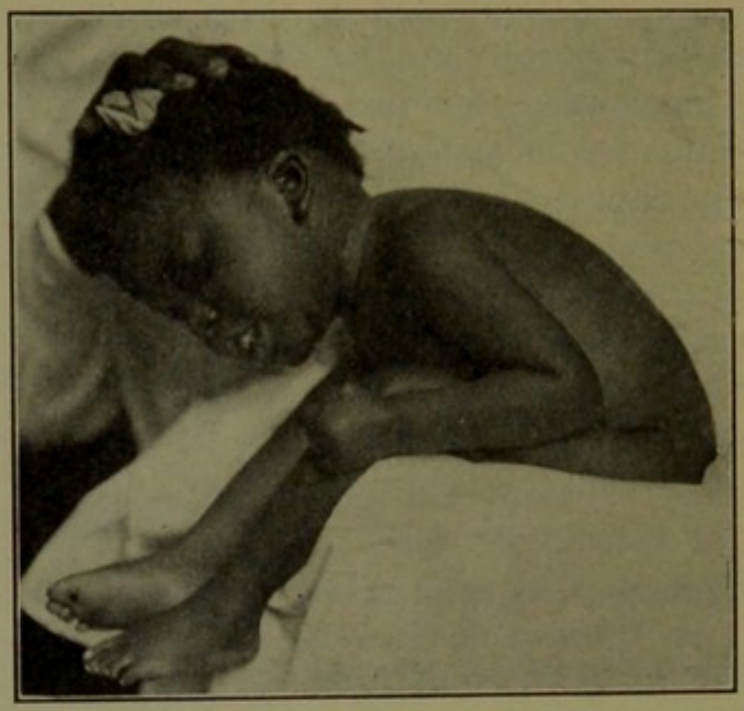


FIG. 110.—MYATONIA CONGENITA. When Sitting Trunk Falls Forward.

ferences. However, in some cases an increase of connective tissue and a considerable amount of fat between the muscle bundles were observed. Oppenheim believes that the disease is due to an arrest of development of the muscle-fibers. Some writers are inclined to believe in a disturbance of function of some ductless gland. Berti, among them, considers amyotonia congenita as a variety of congenital myxœdema. While Amyotonia congenita presents several features distinct from Myopathies, viz. the time of life at which it appears, the general involvement of all the muscles, absence of deep reflexes, the tendency to improvement, nevertheless the pathological findings which show that there is no essential difference in the two conditions, the not very infrequent occurrence of the same symptoms in both maladies—all these facts tend to show that Amyotonia congenita probably belongs to the great group of Myopathy, in which so many variations from the typical forms really exist.

The **treatment** consists of good hygienic and dietetic measures with massage and electricity. A natural tendency for improvement exists in this affection. In my two cases the improvement was remarkable.

Myotonia Atrophica

Rossolimo (*Nouv. Iconogr. de la Salpêtr.*, 1902) gave this name to an affection which was described before as Thomsen's disease with muscular atrophy by Noques, Tirol and Hoffman. It is characterized by muscular atrophy and a slow relaxation of muscles after voluntary contraction. The atrophy is characterized by its distribution, viz. it affects the muscles of the face (myopathic face), the sterno-mastoid, the vasti of the thighs and the extensors of the feet. The extensors and flexors of the forearm, the masseter and temporal muscles are sometimes affected. As to the relaxation of muscles after contraction the stronger the latter the longer is the former. The electrical reactions show a diminished response to faradism. The knee-jerks are absent. Several members of the same family have been found to be affected, males more frequently than females. It appears usually at the age between fifteen and thirty, although Grund has recently reported cases of myotonia atrophica at the age of 47 (Münch. Mediz. Wchnschr., April, 1913.) The disease is essentially progressive. Pathologically it resembles Myopathy as far as the muscles are concerned. Steinert found also degeneration of the posterior columns in the cord. Myotonia atrophica is probably one of the multiple varieties of Myopathy.

DISEASES OF SPINAL MENINGES

Spinal Meningitis.—There are three forms of this affection:

I. Acute spinal meningitis.
II. Chronic spinal meningitis.
III. Hypertrophic cervical pachymeningitis.

I. Acute Spinal Meningitis

This form of meningitis is usually a secondary affection. In the majority of cases it accompanies an acute cerebral meningitis. In some cases it may be primary and strictly localized to the membranes of the cord.

Pathology.—An incision of the membranes will reveal the presence of a serous or purulent exudate and adhesions between the pia, arachnoid and dura. The membranes are hyperemic. This condition may be localized or else extend over the entire cord. The exudate is particularly marked on the posterior aspect of the cord and it covers also the cord and the roots. In the **cerebro-spinal form** the lesion is confined only to the pia and arachnoid. In the **tubercular** form there may be

either isolated tubercular nodules or else infiltrations in the pia around the blood vessels. When the meningitis is secondary to a disease of the vertebræ (caries), the lesion may be confined to the dura, but it may also extend to the pia; purulent formation is found then between the spine and the dura. The periphery of the cord is always in a state of myelitis, more so than the cortical tissue in cases of cerebral meningitis.

Symptoms.—The onset is sudden. General malaise, fever and chills are the first signs. **Pain** along the spine is the most prominent symptom. The latter is extremely tender to touch and the patient immobilizes his spine for fear the least movement will bring on pain. The pain radiates around the thorax and to the limbs, also to the pharynx and larynx. It is continuous, but it presents also paroxysms of exacerbation. The entire surface of the body is hyperæsthetic. The neck and back are rigid. **Kernig's** sign is present. It consists of an inability to extend the legs when the patient is in a sitting position. The **reflexes** are increased. The sphincters are involved. Vaso-motor disturbances are frequently observed, viz. erythemata and sweating. In **cervical meningitis** the following important symptoms are observed: vaso-motor disturbances of the face, pupillary disturbances, vomiting, dyspnœa, dysphagia.

If the patient does not die, he enters into a phase of **paralysis,** which is an indication that the spinal cord became involved (meningo-myelitis). The limbs are then totally paralyzed, flaccidity is complete. The reflexes are lost, anæsthesia takes the place of hyperæsthesia and the sphincters are relaxed. For details see chapter on Myelitis.

Course, Prognosis.—While death may occur on the second or third day, viz. during the first phase of the disease, it may, however, be prolonged. In the majority of cases the termination is fatal, but recovery is possible, although very rare. Death is due to the involvement of the medulla. In recent years it has been shown through the study of cerebrospinal fluid that there are benign and curable (ambulatory type) forms of meningitis and that they are more frequent than they were supposed to be in former years.

Tubercular meningitis and meningo-myelitis may have an acute course, but as a rule they are slow and progressive. Infectious meningitis, primary or secondary, has generally a rapid course. The majority of the subacute form of meningo-myelitis are either tubercular or syphilitic.

Diagnosis.—From **acute myelitis** the disease will be differentiated by the **pain which precedes** the paralysis. The latter, together with anæsthesia, are much later symptoms in meningitis than in myelitis.

Tetanus will be recognized by trismus and paroxysms of contractions.

Meningeal hemorrhage, which also produces pain and rigidity, is not accompanied by fever.

Etiology.—Infection with a point of departure in the neighboring tissues is the chief cause of acute spinal meningitis. The necessary factor is a microörganism. Streptococcus and pneumococcus are the most frequent ones. Diseases of the spine, trauma, bed-sores, infectious diseases, septicemia, tuberculosis and syphilis, compression of the cord—are all causative factors in meningitis.

Treatment.—Applications of ice to the spine, cauterization of the latter, hot baths, administration of iodides and mercurials when syphilis is suspected, hypnotics, sedatives, also lumbar puncture, have been advised (see also Cerebral Meningitis).

II. Chronic Spinal Meningitis

It is a common secondary lesion in various diseases of the cord. Occasionally it may follow an acute meningitis. As a primary affection it is found in senility, in lead intoxication, in alcoholism. Pathologically it presents thickened membranes. The condition of the meninges secondary to cord diseases is given in each chapter. The symptoms of the primary chronic meningitis are analogous to those of the acute form except that they are less marked and may remain latent. There is pain in the back and in the neck. The rigidity of the spine is but slightly marked. Fever is absent.

As a special form of chronic meningitis the following was first described by Charcot and Joffroy:

III. Hypertrophic Cervical Pachymeningitis

Pathology.—The lesion consists of a thickening of the dura in the cervical region of the cord. The thickened inner layer of the dura adheres to the pia and the outer to the endosteum of the spinal column. The cord surrounded by the dense fibrous tissue is naturally compressed. The roots, the posterior especially, are equally compressed. The elements of the cord and the nerve-fibers of the roots undergo degeneration and atrophy. For histological details of the latter see any of the acute or chronic diseases of the spinal cord.

Symptoms.—The first phenomenon is **pain** in the neck. It is continuous and presents paroxysms of exacerbation. It radiates to the occipital region, shoulders and upper extremities. It never follows

the course of nerve-trunks, but it is diffuse. Sensations of tingling, of numbness, also trophic disturbances (herpes) are also present in the painful areas. The entire spinal column is tender and the neck is held rigid. Objective sensibility is altered, hyperæsthesia is present at first, but later when the nerves degenerate, anæsthesia will be observed. All these phenomena are due to compression of the posterior roots.

Gradually (when the anterior roots become involved) the painful phase of the disease gives place to the period of **paralysis** and **atrophy.** They appear first in the **upper extremities.** The atrophy affects mainly the muscles supplied by the median and ulnar nerves. Flexion and adduction of the forearm and hand are impaired. The overextension of the wrist, extension of the basal and flexion of the middle and last phalanges gives the hand a special position (**preacher's hand**) that is characteristic of the disease. The atrophy of the thenar and hypothenar muscles is marked. RD. is present.

Pain usually disappears at this period of the disease, but rigidity of the spine persists. Objective sensory disturbances, hyperæsthesia, hypæsthesia or anæsthesia are present in the affected areas. In a more advanced stage of the disease (when the cord itself begins to suffer) the lower extremities become involved. Spastic paralysis with increased knee-jerks, ankle-clonus, Babinski's sign, Oppenheim's and paradoxical reflexes will be present. But there will be **no** atrophy in the paralyzed muscles. At this phase of the disease the sphincters become involved and the objective sensory disturbances are more marked; the latter are **radicular,** viz. they follow the course of the nerve-trunks. Bed-sores may also develop. The cerebro-spinal fluid presents lymphocytosis.

Course, Termination, Prognosis.—The disease is very slow in development and lasts years. The painful period lasts usually several months. Death results either from the extension of the morbid process to the medulla (bulbar symptoms) or from an infection caused by extensive bed-sores, by purulent cystitis or else from an intercurrent disease (pulmonary tuberculosis, etc.). Arrests in the course of the disease, or even considerable amelioration (perhaps cure) of the symptoms have been reported.

Diagnosis.—Potts' disease, meningeal tumors, syringomyelia may sometimes be confounded with hypertrophic cervical pachymeningitis. The deformity and pain produced by pressure upon the cervical spine in Potts' disease are characteristic. Meningeal or vertebral tumors have their special signs (see these chapters). The diagnosis with syringo-myelia is sometimes difficult. The typical sensory dissociation especially will enable most of the time to diagnose syringomyelia.

Etiology.—Injuries, prolonged exposure to cold, lead intoxication, alcoholism, syphilis, are all considered as possible causes of the disease.

As a primary affection hypertrophic cervical pachymeningitis is rare. Most frequently it is symptomatic of syringomyelia, of meningo-myelitis of syphilitic or tubercular nature. It may participate in pachymeningitis of the base of the brain.

Treatment.—Local counter-irritation and cauterization, warm baths, internal administration of mercurials and iodides may be tried. Little can be expected from this treatment. Massage and electricity may improve the muscular atrophy.

CIRCUMSCRIBED SEROUS SPINAL MENINGES

This condition of the meninges has been described only recently. It consists of a circumscribed accumulation (pseudo-cyst) of cerebro-spinal fluid in the sub-arachnoid cellular tissue. It is probably due to an inflammation and subsequent adhesions of the meninges which thus produce an increased secretion of the fluid in a circumscribed space. This pseudo-cyst is found in the dorso-lumbar region on the posterior surface of the cord. As to the **Etiology,** traumatism, bacterial infection, a toxic process, are considered the main factors.

Symptoms.—Horsley insists upon unilateral pain at the onset. **Hyperæsthesia** is marked

Anæsthesia follows the hyperæsthesia. It may affect all forms of general sensibility (touch, pain and temperature). Pressure at the level of the roots is painful. The anæsthesia may vary daily as far as its distribution is concerned. Paraplegia sets in insidiously, first in one leg and then in the other. It is usually spastic but flaccidity may follow spasticity. The reflexes are increased, the toe phenomenon is present. Ankle-clonus is frequent. Atrophy may be present. The sphincters may be involved. Œdema has been observed.

The prognosis depends upon the early recognition of the affection and upon the promptness of surgical intervention. If abandoned, complete paraplegia with its usual complications (urinary infection, bed-sores, etc.) will develop. **Laminectomy** with puncture of the cyst, followed by washing it out with 1 : 2000 of bichloride is the only therapeutic procedure.

HEMORRHAGES OF SPINAL MENINGES

They may occur either on the *outer* or *inner* surface of the dura.

Extra-dural Hemorrhages.—They usually occur in traumatism, frac-

ture, dislocation of the spine, penetrating wounds, caries and carcinoma of the vertebræ and meningeal syphilis.

Intra-dural Hemorrhages.—The preceding factors may produce this form of hemorrhage but less frequently. Infectious diseases, acute and chronic inflammation of the meninges (especially syphilis), instrumental deliveries, hemorrhages in the cranial cavity—are all causes of spinal intra-dural hemorrhages.

Symptoms.—They are: pain in the spine radiating to the limbs, rigidity of the spinal column, paræsthesia, hyperæsthesia. Paralysis is the most characteristic symptom. It is incomplete at first, but in two to three days becomes complete. Anæsthesia is also slight in the beginning, but becomes more and more pronounced later.

The sphincters are usually involved. The knee-jerks are at first diminished, but later change according to the degree of compression.

The **course** of the disease depends upon the amount of blood in the spinal cavity. Absorption of the blood may take place in slight cases and the symptoms then gradually disappear; recovery may take place in a few weeks. Frequently, however, complications set in: secondary meningitis, infection from bladder, bed-sores, etc. The **prognosis** depends upon the seat of the hemorrhage. If it is in the cervical region, death may be rapid from bulbar symptoms. If hemorrhage is abundant, cerebral anemia and syncope may follow. Generally speaking, the disease lasts from two to three months and usually leaves motor, sensory and sphincter disturbances.

The **diagnosis** can be made chiefly from lumbar puncture.

The **treatment** consists of absolute rest in bed. The patient should be placed on his side in order to avoid congestion of the meninges and lungs. Application of ice and counter-irritation at the level of the suspected hemorrhage are the next indication. Pain must be relieved by the usual means. Finally repeated lumbar punctures are very important.

INTERMITTENT CLAUDICATION OF THE SPINAL CORD

In 1906 (*Reane Neurologique*) Dejerine called attention to the following symptom group. In the midst of apparently good health the patient notices that while walking, one of his legs gets more easily tired than the other, but after a rest this fatigue disappears. For a long time, sometimes years, he will complain of this unilateral weakness, but sooner or later the other leg will become similarly affected. Gradually the interval after which the weakness of the limbs appears, becomes shorter and

shorter and a typical spastic paraplegia develops. In the beginning of the intermittent spinal claudication disturbances of the sphincters are observed, either retention or incontinence, but especially imperative micturition, frequently also sexual impotence. The patellar tendon and Achilles reflexes are exaggerated, more particularly in the limb whose function is disturbed. Ankle-clonus may be absent. The toe phenomenon is frequent. The terminal spastic paraplegia may take place in months or in many years. The latter is the most frequent occurrence. In rare cases the condition may run an acute course, viz. the paraplegia develops shortly after the onset of the claudication.

The diagnosis is to be made from **intermittent claudication** of **peripheral origin.** In the latter affection the condition is at first also unilateral; the walking brings on rapidly a fatigue, but there is pain which is frequently very intense; it is a cramp that affects the entire limb. But the reflexes are normal, there are no disturbances of the sphincters. On the other hand we find here the limb cold, and after a walk it becomes cyanosed, the cyanosis disappears after a rest. The most important sign of the claudication of peripheral origin is the disappearance of normal pulsations in the **dorsalis pedis artery** at first and then in the posterior tibial artery; sometimes the pulsation cannot be felt in the popliteal and femoral arteries. Skiagraphy often shows an atheromatous condition of these arteries. Recoveries may occur because of the supplementary collateral circulation, but most frequently the condition ends in gangrene Its etiology is therefore an endarteritis and atheroma; it occurs in cases of alcoholism, gout and syphilis, or else it may follow an acute endarteritis after typhoid fever in young people.

A different condition is found in **intermittent spinal claudication.** Here the reflexes are exaggerated, the toe phenomenon is frequent and sphincter disturbances are present. Finally the arterial pulsations are normal and there are no vasomotor disturbances in the affected limbs; the patient feels no pain while walking but only a heaviness in the limbs. The **Pathogenesis** of the affection lies in a very slow process of arteritis which leads to a narrowness, of the lumen of the arteries supplying the segments of the dorso-lumbar region of the spinal cord.

As to the **etiology** of the affection, infectious disease, such as grippe (case of Dejerine and Poix) may be the cause. The most frequent etiological factor is **syphilis.** The anti-specific medications, such as Salvarsan, mercury and iodides, when promptly administered may improve the condition or at least delay for a very long time the oncoming terminal Spastic paraplegia.

CHAPTER XXI

SYPHILIS OF THE NERVOUS SYSTEM

THE microörganism discovered by Schaudinn and Hoffmann as being the cause of syphilitic lesions is known under the name of spirochæta pallida. The syphilitic poison, whatever its intimate nature may be, appears to have a special predilection for the nervous system. Its effect upon the latter may be manifested in **two different forms.** In one of them, to which tabes and paresis belong, the lesions were supposed to be until recently not the direct and immediate result of syphilitic infection, but a late and secondary development (degeneration), against which the anti-syphilitic treatment is powerless. They are the **"parasyphilitic affections"** of Fournier.

Our former view concerning the relationship of syphilis to tabes and paresis must now be modified. Noguchi and Moore (J. of Exper. Med., Feb. 1, 1913) have found the spirochætæ in the brains of 12 paretics, and Noguchi found them in the posterior columns of a tabetic. In the brain, they are seen more numerous in the cortical than in the white matter, they are scattered in groups between the cells and neuroglia fiber. They are rare in the vicinity of the blood-vessels and not found in the walls of the latter. These findings have been corroborated by Levaditi, Marie and Bankowsky (Presse Méd. No. 36, 1913). The parasyphilitic diseases are therefore very probably true syphilitic affections.

The other form with which we will be exclusively concerned here is characterized by distinctly specific lesions due to the direct effect of syphilis. They are amenable to antisyphilitic treatment, especially at the beginning.

Syphilis may affect the cerebro-spinal system at any period of its development. Generally speaking, however, cerebral syphilis occurs in the tertiary period, while spinal syphilis is an early occurrence, viz. in the first or second year, even as early as in a few months.

In a syphilitic individual a trauma, excesses, especially of alcohol, and exhaustion are apt to be the exciting causes for the development of syphilitic cerebro-spinal disturbances.

SYPHILIS OF THE BRAIN

Pathology.—Meninges, brain tissue and **blood vessels** are affected. In the **meninges** the specific **gummatous** formation, which is the characteristic lesion of syphilis, may be **diffuse** or **circumscribed.** Gummata may be of various sizes. As soon as they are formed, they become a source of irritation and inflammation to the neighboring tissue. They

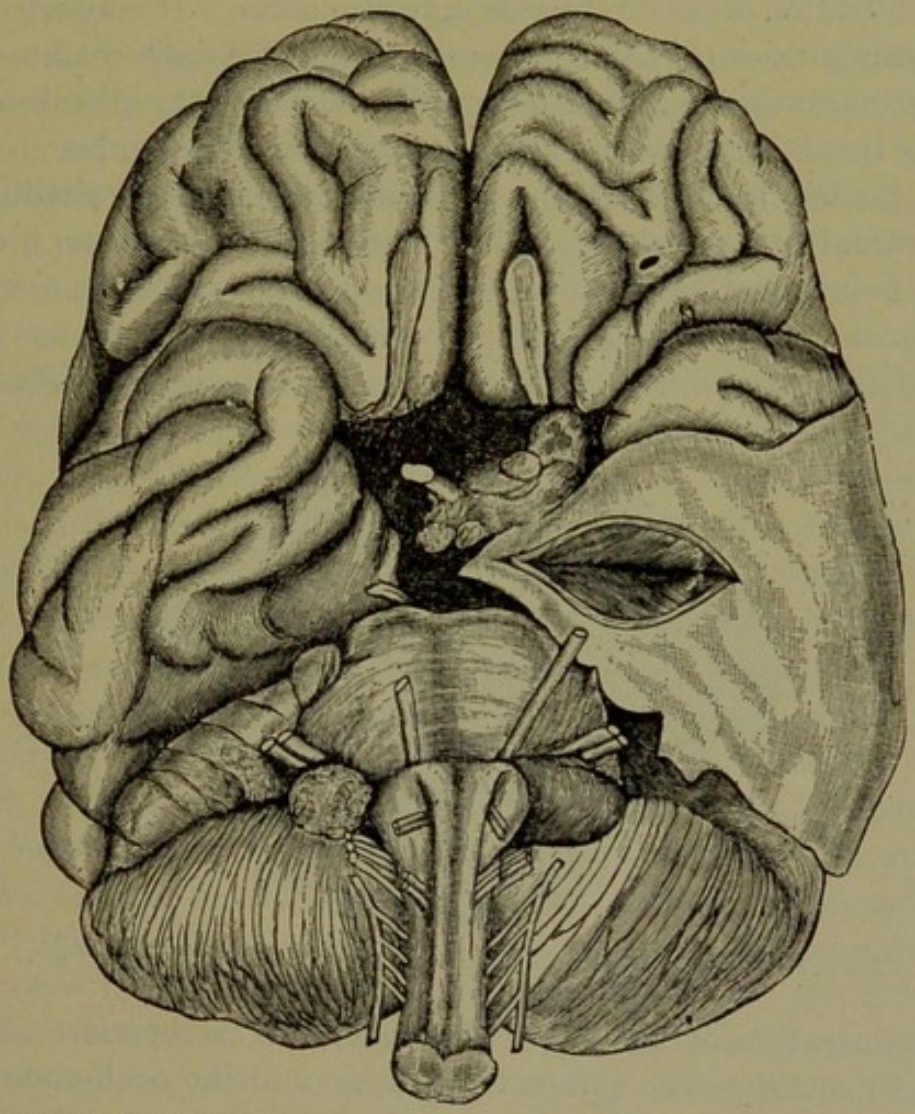


FIG. 111.—SYPHILITIC MENINGITIS OF THE BASE OF THE BRAIN. (*Flatau, Jacobson, Minor.*) On the left are seen strong adhesions between the dura, pia and underlying cerebral tissue.

extend into the latter along the sheaths of the blood vessels of the pia mater. Adhesions of the meninges and softening of the nervous tissue follow. The most frequent seat of gummata is the base of the brain from the chiasma to the pons. There the gumma starts from the pia and rarely involves the dura. The optic and oculomotor nerves, also the large basal arteries, especially the middle and anterior cerebral, are usually involved. When situated on the convexity, they are usually found in the frontal or parietal regions. The obliteration of the blood vessels will produce softening of the brain tissue. When the lesion is diffuse, the

meninges are covered with a thick gelatinous exsudate. Histologically a gumma consists of a granulation tissue formed by proliferation of connective tissue and endothelial cells. It is very vascular and presents caseation in some places. Round cells are in abundance along the adventitia of the blood vessels.

Gummata primarily developed in **the brain** are rare. Diffuse gummatous infiltration of cerebral tissue may also occur. It may affect all the three membranes or only the pia-arachnoid. The dura mater when involved becomes very thick and forms adhesions with the other membranes and brain tissue (gammatous pachymeningitis. The cortex is usually involved, the lesion will be then a syphilitic **meningo-encephalitis.**

The **cranial nerves,** which are frequently involved in basal meningitis, may also be affected independently. In such cases there is a round cell infiltration in the epineurium; thick processes are sent out between the bundles of the nerve-fibers. The latter being compressed undergo even-

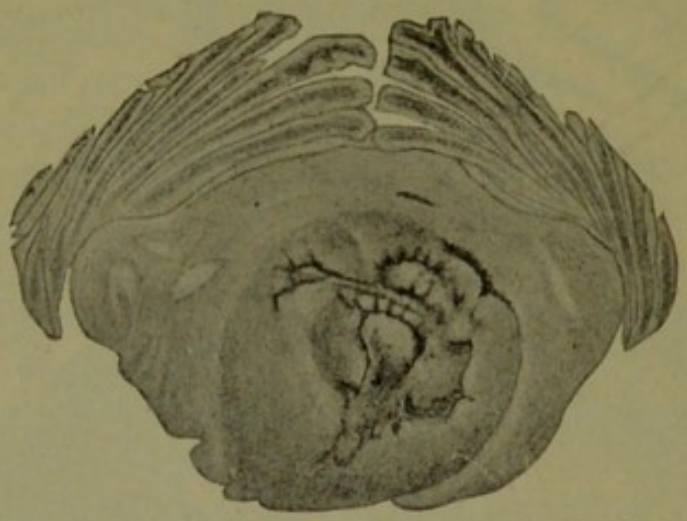


FIG. 112.—LARGE GUMMA IN THE PONS. (*Flatau, Jacobson, Minor.*)

tually degeneration or atrophy. The condition is therefore at first an interstitial neuritis. The chiasma, the optic and the oculo-motor nerves are most frequently affected. The fourth, fifth, sixth, seventh and eighth are next in frequency. The remaining four nerves are almost never involved.

Syphilis has a special predilection for the **blood vessels of the base** of the brain, although specific arteries may be encountered in any portion of the brain. The lesion consists of a small gummatous formation or infiltration in the walls of the blood vessels. Microscopically there is a cell hyperplasia in the lining endothelium of the vessel and the fenestrated membrane. The effect of the arterial lesion will be either thrombosis or rupture of the vessel. Softening of cerebral tissue or hemorrhage will be the consequence, provided collateral circulation is not established. Such is the case with occlusion of the terminal arteries in the internal

capsule, basal ganglia, crura, pons and medulla and the nuclei of the cranial nerves from the third to the twelfth inclusive.

In congenital syphilis the encephalitis is of the sclerotic form, rarely softening.

All the lesions enumerated may be observed in the same case. In fact **multiplicity of lesions** is frequent and characteristic of syphilis of the nervous system.

Symptoms.—The foregoing remarks indicate that the clinical picture will vary with the localization of the lesion. A syphilitic basal meningitis, a diffuse meningitis of the cortex, a circumscribed gumma in various portions of the brain, a generalized syphilitic arteritis—all these forms naturally have their special symptoms. Irrespective of the form or of a special localization, cerebral syphilis presents a **prodromal** period characterized by the following symptoms.

Headache is the most constant and the earliest phenomenon. Its essential feature is to present **exacerbations,** especially **in the evening** or at night, and to be deep seated in some part of the cranium. During the day it is dull or may disappear entirely. The headache yields with a remarkable facility to antispyhilitic remedies. **Vomiting** is quite frequent. It may occur without food in the stomach. **Vertigo** is also a common symptom. The general condition of the patient changes. He becomes apathetic, somnolent, languid, loses his appetite and in weight.

In cerebral syphilis, due to a **generalized** specific **arteritis,** the symptoms will be those apoplexy caused by arterial lesions, especially by arterio-sclerosis. Thrombosis is the usual occurrence. In such cases hemiplegia, aphasia develop slowly after a prodromal period and without loss of consciousness. The special character of these phenomena is that they improve rapidly and may even disappear under the influence of the specific treatment. In some cases there are brief and fugacious attacks of aphasia without paralysis of the extremities. Sometimes there is only some difficulty of articulating. In other cases the attack consists of peculiar sensations (tingling, numbness, etc.) on one side or in one limb.

Syphilitic arteriti may affect the large trunks of the convexity or at the base. In such cases the entire artery may become obliterated or else aneurisms may form. A rupture of such a vessel will produce a sudden coma and death. Generalized or focal epileptiform convulsions are not infrequent. If they are frequent and not controlled, psychic manifestations become evident, viz. slow cerebration, loss of inhibition, outbursts of anger.

In cerebral syphilis of **meningeal origin** the manifestations are different from those of the previous form. When the meningitis is acute,

the patient, after a period of intense headache, becomes stuporous. Hebetude is seen in his acts and speech. Coma may follow. In other cases the condition may be reverse; delirium, excitement, generalized convulsion take the place of the depression.

In **basal** meningitis, during this acute stage, very frequently palsies of cranial nerves develop. The third nerve palsy (ptosis, strabismus) is the most common occurrence; it may be total or partial; the first is rarer than the latter. When the optic nerve is involved, there may be amblyopia, complete blindness or hemianopsia. Ophthalmoscopic examination shows the following possibilities: optic neuritis, choked disc, optic atrophy. The fourth and sixth nerves are sometimes involved. Uhtoff collected only twenty-seven cases out of 150. The same can be said of the fifth nerve; the sensory portion is more frequently involved than the motor. The seventh and eighth are occasionally affected and usually together. The facial palsy is usually of the peripheral type. It may be associated with a hemiplegia or the opposite side (crossed paralysis). It occurs when the facial never is affected with other structures in the pons. The special feature of these palsies lies in their transitory character and in the fact that prompt administration of antisphilitic treatment is followed by marked improvement or even complete recovery.

In exceptional cases the acute stage instead of improving becomes prolonged: the patient enters rapidly into a profound coma and never regains consciousness. Among the less frequent symptoms of basal meningitis may be mentioned: polydipsia, polyuria and glycosuria.

In acute meningitis of the **convexity** epileptic convulsions will figure prominently instead of paralytic symptoms. They may be generalized or unilateral. Charcot believed that cortical epilepsy is one of the most frequent consequences of syphilis of the brain.

In **chronic syphilitic meningitis,** in addition to paralytic or epileptic phenomena, there will be psychic disturbances. They consist of mental feebleness associated with emotionality, amnesia, apathy with semi-somnolence, automatism, impairment of memory and in advanced cases of dementia. Delusions and hallucinations may also be present.

In cases of **circumscribed gummata** the symptoms depend upon their localization. Whether at the base, on the convexity or in the substance of the brain the symptomatology will be that of tumors of the brain (see this chapter).

Syphilitic Arteritis

While every case of syphilitic meningitis is associated with some degree of endarteritis, there are nevertheless cases which present symptoms of arteritis without meningitis.

They occur most frequently according to Gowers within the first two years after the infection and in young individuals.

Symptoms.—Headache exists for a long time before other symptoms. It is usually not very severe. It is diffuse, it disappears and reappears; it is increased by mental effort. Short attacks of **dizziness** are common. **Sleep** is usually poor. **Psychic** symptoms are especially conspicuous, viz., incapacity for work, mental fatigue, feebleness of intelligence, somnolence, periods of excitement, irritability.

Besides, a typical **neurasthenic** symptom-group is not infrequently observed in the course of syphilis. The symptoms are, generally speaking, those of neurasthenia from other causes, except the following features: In ordinary neurasthenia there are only paræsthesiæ, such as numbness, heaviness, etc. In syphilis there is pain, which is worse at night. Headache of neurasthenia is usually diurnal, in syphilis nocturnal. Antisyphilitic remedies will relieve the specific headache, but not the headache of neurasthenia. Finally the posterior Wasserman reaction in syphilis and the negative one in neurasthenia will decide the diagnosis.

Apoplectic seizures of various degrees occur; they may occur even during sleep. The seizure is usually transitory. Sudden impediment or loss of speech may occur very frequently. Transitory **hemiplegia** or **monoplegia** or else a **sensory disturbance** on one side of the body may occur and last even a few minutes.

The symptoms of endarteritis just enumerated are due to a general thickening of the intima, producing a partial occlusion of the blood vessels. These changes may eventually terminate in thrombosis of the diseased vessels and produce a permanent loss of function. Most serious symptoms occur when a basilar artery becomes occluded; a fatal termination is almost inevitable.

Syphilitic Hemiplegia

Closely associated with the subject of syphilitic arteritis is syphilitic hemiplegia. The latter is of very frequent occurrence and therefore of practical importance.

Etiology.—It affects particularly individuals who have not been treated or imperfectly treated, also in cases of severe infection. It occurs at any period of the disease, but especially between three and fifteen years after the infection. It may coincide with the secondary mainfestations of syphilis. Men are more frequently affected than women. As predisposing causes may be mentioned: mental and physical fatigue, violent emotions and various infectious diseases other than syphilis.

Hereditary syphilis plays a large part in syphilitic hemiplegia; it may occur as early as the age of five months and as late as thirty-three years. The classical infantile spastic hemiplegia has in a great many cases a syphilitic origin.

Pathology.—The chief lesion is **syphilitic arteritis.** The syphilitic virus has a special predilection for the cerebral arteries. The syphilitic process either narrows or obliterates completely the lumen of the blood vessel, producing either a temporary cerebral ischemia or softening, or else a destruction of the arterial wall followed by a hemorrhage. Among all the arteries of the brain, those of the base are the seat of predilection. The terminal branches are less frequently affected. In order of frequency the following arteries are involved: Sylvian, anterior cerebral, basilar trunk, posterior cerebral, finally the branches of the Sylvian artery. The main syphilitic lesion, gumma, may form in the arterial walls round and hard tuberosities, but more frequently the syphilitic process consists of plaques, of diffuse infiltration. The artery loses its transparency, the plaques form a thickened prominence within and without the artery. The lumen is gradually being **narrowed** so that it presents but a small fissure, or it may be totally obliterated by a secondary process, viz. **thrombosis.** In other cases the artery is dilated at a certain distance and thus **aneurisms** are formed. The latter are observed especially in the basilar and Sylvian arteries. Microscopically the following condition is found: thickening of the inner wall, leucocytic infiltration; the elastic inner membrane is fragmented, torn; the adventitia is always altered and the same leucocytic infiltration is observed. The muscular layer is the least involved.

The process that follows is the terminal state. It consists of chronic arteritis, of a sclerotic transformation: the entire vascular wall became a dense fibrous tissue. Such vessels are easily ruptured.

The cerebral tissue in view of the damage to the arteries undergoes either softening or suffers from hemorrhage. The first is the most frequent.

The lesions in hereditary syphilis are precisely the same as just described.

As to the spirochetæ, there are very few cases reported in which they were found in the nervous tissue.

Symptoms.—Premonitory symptoms may appear weeks, months or years before the hemiplegia.

Headache is constant, it is of nocturnal type and is ameliorated by specific treatment. At the same time the individual becomes irritable and depressed; all sorts of paræsthesiæ appear. Two special symptoms

make their appearance, viz. deafness or vertigo, also ophthalmic migraine. Following an effort and sometimes without cause an apoplectic attack occurs. It may be only slight with or without aphasic symptoms, or else it may be typical. Irrespective of the degree of involvement, the attack is usually transitory and lasts but a few days. On the other hand it may repeat itself in the same day or every few weeks or months. When later on the hemiplegia has a tendency to become permanent, it establishes itself by gradation: first the lower limb, a few days later the upper limb and then the face.

In another form syphilitic hemiplegia is associated with a palsy of cranial nerves, especially the third nerve.

Finally syphilitic hemiplegia may develop suddenly without premonitory symptoms similar to ordinary hemiplegias. Syphilitic hemiplegia, when definitely established, may present several clinical varieties according to the intensity of the motor disturbances and to the localization of the lesion. They do not differ from hemiplegias with other etiological factors (see chapter on Hemiplegia).

Course, Termination, Prognosis.—In cerebral syphilis with a generalized specific arteritis the attacks of hemiplegia or of cranial nerve palsies may be fugacious and brief; they may disappear and reappear or else disappear completely when under treatment. On the other hand because of repetition of attacks the damage done to the cerebral tissue or to the cranial nerves, especially in the meningitic forms of cerebral syphilis, may be so intense that the lesion remains permanent. Changes in the cranial nerves may become so profound (atrophy) that recovery is impossible. The same is observed in circumscribed gummata on the surface of the brain. Therapeutic intervention (salvarsan, mercury, iodides) undoubtedly modifies considerably, and sometimes favorably, the course of the disease, but it is powerless in cases of hemorrhages and softening in the cerebral tissue. The prognosis, generally speaking, is good when the disease is treated early, but it is grave in advanced cases. It is good also when the Wasserman reaction becomes negative. It is grave especially in cases with epilepsy, in thrombosis of the medulla and in cases with pronounced mental symptoms.

Cerebral syphilis, as a rule, is a serious disease.

Diagnosis.—The chief characteristics upon which a diagnosis of cerebral syphilis can be based are as follows:

1. Sudden onset of cerebral symptoms in an individual in the midst of apparently good health.
2. Headache of a special form (nocturnal exacerbation).
3. Palsies of cranial nerves.

4. Hemiplegia, monoplegia, focal or generalized epilepsy.

5. The course of the disease: disappearance and reappearance of symptoms, their brief duration; multiplicity of symptoms.

6. Disappearance or prompt amelioration of symptoms under the influence of salvarsan, mercury and iodides.

7. The exclusive presence of lymphocytes and increase of albumen in the cerebro-spinal fluid, positive Wasserman reaction in the cerebro-spinal fluid and in the blood (see chapter on Lumbar Puncture).

8. History of syphilitic infection.

As to the differential diagnosis with paresis and tabes see the respective chapters.

SYPHILIS OF THE SPINAL CORD

Spinal syphilis may present itself in the following forms: (1) chronic syphilitic meningitis; (2) meningo-myelitis; (3) acute syphilitic myelitis; (4) Erb's spinal paralysis; (5) gummatous tumor; (6) syphilitic disease of the vertebræ, which is very rare.

Pathology.—In the majority of cases of spinal syphilis the lesion is a **meningo-myelitis.**

As the point of departure in spinal syphilis is usually in the meninges, it is important to consider first the changes of the latter.

The most frequent lesion of the membranes is **pachymeningitis.** The thickened dura adheres to the pia-arachnoid and through the latter to the cord. It may be diffuse or circumscribed. The cervical region is most frequently affected. Histologically, in early stages the typical specific infiltration will be found (see Cerebral Syphilis), but in an advanced stage of the disease there is a fibrous meningitis consisting of a uniform sclerotic (connective) tissue. Solitary gummata are rare.

In **meningo-myelitis** (the most common occurrence) the following lesions are usually found.

Acute.—The pia-mater is infiltrated with round cells and is therefore thickened; its small blood vessels are the point of orgin for this infiltration. This can be traced in all the prolongations of the pia. In the gray matter of the cord the cell infiltration of the blood vessels is at its maximum. All the vessels, arteries and veins in the cord are similarly affected. Syphilis has a special predilection for blood vessels. Thickening of the walls of the latter causes a narrowness of their lumen, hence poverty of blood supply and softening of nervous tissue. Degeneration and atrophy of cells (especially in the anterior cornua) follow. In the white matter the nerve fibers at first lose their myelin and later disappear; they soon become substituted by proliferated neuroglia.

Chronic.—Cases of long standing are characterized by **sclerosis,** which affects the meninges and the cord. The entire section of the cord gives the impression of a fibrous tissue. Ascending and descending degenerations are seen parting from the original focus.

Symptoms.—In the majority of cases the meninges are first affected and the cord follows (**meningo-myelitis**). The meningeal symptoms precede often the symptoms of myelitis by days and weeks.

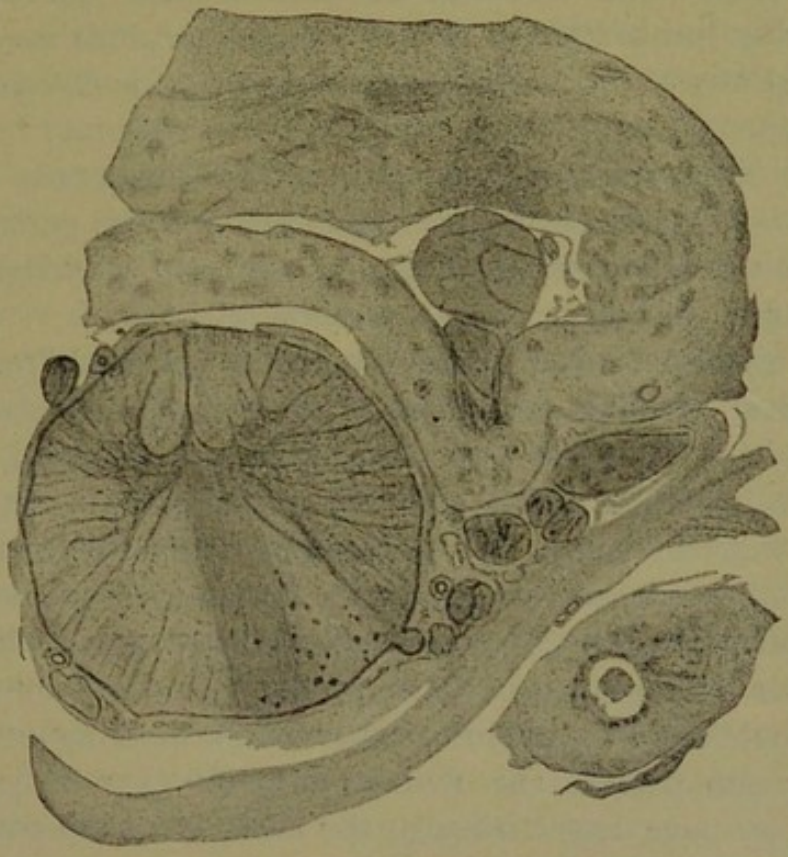


FIG. 113.—SYPHILITIC MENINGO-MYELITIS. (*Flatau, Jacobson, Minor.*)

Pain along the spine radiating toward the limbs is the first symptom. It is usually aggravated at night. It is due to involvement of the meninges and of the roots. Numbness and tingling (**paræsthesiæ**) in the limbs usually accompany the pain. Soon the cord becomes involved. Then develops **paralysis** in both or in one of the limbs, particularly in the lower, and usually in one more than in the other. It is **flaccid** at the beginning, but later spasticity develops. The **sensations** are usually disturbed. Anæsthesia or hypæsthesia may occur. Sometimes there is a sensory dissociation like in syringomyelia. The sphincters are always and very early affected: incontinence is rare. The sexual power is impaired. **Muscular atrophy** may occur, but this is exceptional.

The **characteristic feature** of syphilitic meningo-myelitis lies in the multiplicity of symptoms, in their unequal distribution on both sides of

the body, in their variability and instability, in their disappearance and reappearance, finally in their modification when the patient is under treatment. A. Thomas considers as one of characteristic signs of spinal syphilis what he calls "intermittent spasm of the blood vessels of the spinal cord." The latter is manifested in transitory, mobile disturbances in sensations, motion and function of the bladder. He considers these symptoms as the premonitory period of syphilitic paraplegia. (See also spinal intermittent claudication of Dejerine on page 334. According to the predominant localization of the lesion spinal syphilis may assume the form of almost any of the cord diseases: transverse myelitis, tabes, ataxic paraplegia, etc.

When the disease advances in spite of the treatment (sign of well-established and irreparable degenerative lesions), the patient is in the same condition as in chronic myelitis with an array of serious symptoms, such as bed-sores, incontinence, contractures, etc.

Meningo-myelitis of the **thoracic region** is the most frequent occurrence. **Cervical meningo-myelitis** is frequently associated with a basal meningitis. Here all four extremities are involved, and if the eighth cervical and first dorsal segments are affected, symptoms of the sympathetic will be present, viz. contraction of the pupil, enophthalmos, disturbance of secretion of the sweat glands. Meningo-myelitis of the **lumbo-sacral region** will present a flaccid paralysis with loss of all reflexes, severe sensory disturbances in the lower extremities, disturbances in the function of the bladder, rectum and genital organs. Meningo-myelitis may also present the Brown-Séquard's type of paralysis (see this chapter on page 283). Finally the **roots of cauda equina** may be affected with gummatous meningitis, producing a complex symptom-group which is most frequently unilateral (see page 291).

Erb's Spastic Spinal Paralysis.—This form is quite common. It is characterized pathologically by a primary degeneration of the postero-lateral tract, also ascending cerebellar tract. Shortly after the initial chancre (in one of my cases six months) gradually, but progressively, **paralysis** develops in the lower extremities. There are no marked meningeal or root symptoms observed in the preceding form. Pain, if present, is very slight. Girdle-sensation, numbness and a diminution of the general sensibility are usually present. The **gait appears** to be **spastic,** but it contrasts strikingly with the real flaccidity of the muscles (hypotonia). There is no muscular atrophy. The knee-jerks are exaggerated, ankle-clonus, Babinski's sign, Oppenheim's and paradoxical reflexes are elicited. The **sphincters** are early and constantly involved. The upper extremities are usually intact. There are no pupillary changes.

Course, Termination, Prognosis.—Spinal syphilis is rarely acute. The majority of cases are chronic. They may last years. Complete recovery is not frequent, although it may occur. Those cases in which evidences of well-established tract lesions are present bear a bad prognosis. It is also unfavorable in cases which show only slight improvement in spite of a prolonged energetic treatment. Under treatment acute symptoms may disappear, such as pain, bladder disturbance, but disturbances of reflexes, of gait, may persist. Death occurs from some intercurrent disease or from septic infection following cystitis or bed-sores.

Diagnosis.—The unequal distribution, the instability, the disappearance and reappearance of the symptoms, their prompt amelioration when under treatment, finally their disseminated character, are sufficiently typical in the majority of cases to make a diagnosis.

Acute Syphilitic Myelitis.—Acute myelitis may develop as a direct consequence of syphilitic infection.

Pathology.—Swelling of lymph sheaths in the vessels of the meninges and cord, rupture of small vessels and extravasation of blood corpuscles; degeneration of white and gray matter.

Symptoms.—Sudden or rapid onset with the following premonitory signs: paræsthesia and sharp pains in the lower limbs; stiffness of the back; cramps in the legs; retention of urine and feces; the reflexes are variable. Soon appear the following manifestations. Complete paraplegia. Profound sensory disturbances. In myelitis of the lumbar segment the tendon reflexes are lost, in myelitis of the dorsal region the reflexes may be increased. The function of the sphincters is very early involved. Wassermann test is strongly positive.

Course.—Improvement may occur rapidly after the treatment is instituted, but recurrences are frequent. In some cases it may terminate fatally in a short time.

CEREBRO-SPINAL SYPHILIS

A simultaneous involvement of the brain and the spinal cord is by far more frequent in syphilis of the nervous system than an isolated affection of each of the two portions of the central nervous system. In the majority of cases the cerebral disturbances are more marked than the spinal; the reversed condition may occasionally occur.

Not infrequently one series of symptoms disappears promptly when the treatment is energetic, or else some symptoms are so slight that they are overlooked. When the patient comes under observation, only the spinal or only the cerebral type may be present. A close investiga-

tion will reveal in a large number of cases the syphilitic invasion of the entire cerebro-spinal axis with predominance of either cerebral (more frequent) or spinal symptoms.

Syphilis of the Peripheral Nerves.—In spinal syphilis the roots are compressed by the thickened meninges through which they pass. The nerve-trunks in their course between the roots and their terminations may encounter syphilitic gummata in the tissues and undergo compression. But the peripheral nerves may be primarily affected by the specific poison, resulting in syphilitic endo- and perineuritis with obliteration of the blood vessels and subsequent degeneration of the nerve-fibers.

Symptoms.—They are: neuralgia, neuritis, multiple neuritis and root neuritis. Syphilitic neuralgia occurs in the early stages of the disease. Among all the cranial nerves the fifth is particularly a frequent localization. Of the cervical plexus the occipitales are often affected. The intercostal nerves are not rarely involved. Among the nerves of the lumbo-sacral plexus the sciatic suffers most frequently. We find here the same clinical manifestations as in neuralgia and neuritis of any origin; the same motor and sensory symptoms and changes of reflexes. Syphilis rarely affects all nerves with equal frequency. Some nerves are rarely involved. Thus, for example, the seventh nerve palsy is not a frequent occurrence (see my contribution in *Arch. of Diagnosis*, 1908).

For practical purposes it is important to bear in mind that if in any given case of peripheral nerve involvement of long standing, after having excluded all toxic or infectious causes, the condition persists, syphilis should be thought of and an appropriate treatment instituted.

Syphilis and Mental Diseases.—Mental disturbances may be encountered in the secondary or tertiary periods. Those of the **secondary period** are genuine psychoses of toxi-infectious nature in this sense, that they are due directly to the action of the specific poison and not to cerebral lesions caused by syphilis. They usually appear at a time when the eruption or any other acute symptoms such as mucous patches, adenopathies, etc., develop. The onset is usually sudden. Headache and insomnia appear first. Hebetude, stupor, somnolence, mental obtusion, lack of orientation, sometimes delirium and hallucination with delusions of persecution appear next. These are the phenomena of confusional insanity. They present no special features distinguishable from those encountered in similar states caused by intoxications or infections of any other origin, except with regard to the curative effect of the specific treatment.

The psychoses of the **tertiary** period differ from the preceding ones in this respect, that they occur long after the initial infection and do not coincide with eruptions or other secondary manifestations. They are due

to meningo-encephalitis, obliterative endarteritis and accompany usually motor symptoms, such as epileptic or apopletic attacks, palsies of cranial nerves, etc. They are the manifestations of cerebral syphilis. The mental phenomena of the latter with the gradually oncoming dementia as well as their exacerbations and amelioration when under treatment have been already considered. As to their relation to Paresis see this chapter.

The relation of **hereditary syphilis** to mentality is of great importance. With the advent of Wassermann reaction it was made evident that certain organic nervous diseases, especially in childhood, are the result of hereditary syphilis. It is admitted, for example, that juvenile paresis is due to hereditary syphilis. But apart from organic nervous diseases, parental syphilis can be considered also as the cause of various mental abnormalities, such as imbecility, idiocy, of various psychoses of the young. Such individuals not infrequently present in childhood or at puberty some external manifestations of syphilis, such as pupillary disorders, iritis, etc.

Treatment.—Salvarsan, mercury and iodides are the chief medications.

Mercury.—There are three methods by which mercury may be administered: (1) inunction; (2) intra-muscular injection; (3) ingestion.

Inunction is a very good method. In an adult ℥j of unguentum hydrargyri is rubbed in twice daily. The regions of the body having most cellular tissue are to be selected for the inunctions. The upper and inner surface of the arm and the upper and inner surface of the thigh are preferable. Each time a fresh area is selected. It is advisable to wash off with hot water and soap the surface to be rubbed into. Sulphur baths are advisable during the mercurial treatment, as thus the mercury is converted into sulphate of mercury which is easily absorbed. The number of rubbings depends on the intensity of the symptoms and upon the tolerance of the individual. I am in a habit to continue the inunctions for two weeks in succession, if there are no symptoms of intolerance. The latter consist of salivation, gastro-intestinal disorders, swelling of the gums, febrile state. Each inunction should last twenty or thirty minutes.

Intra-muscular injections are administered in soluble or insoluble forms of mercury. The latter are sufficient once a week, the former more frequently. By this method mercury is rapidly absorbed. It is indicated in urgent or very grave cases.

The gray oil is the most appropriate preparation among the insoluble ones; biniodide, benzoate and lactate of mercury, among the soluble ones. The dorsal muscles or the gluteal region are the places of choice. The injections must be made deeply into the muscular tissue and all possible antiseptic precautions must be taken during the manipulation.

Finally mercury may be administered by the **mouth** when inunction or intra-muscular injections cannot be used. Among all preparations I prefer bichloride of mercury in doses of gr. 1/32 t. i. d. for an adult.

Mercurial treatment should precede the iodides, particularly in cases with acute symptoms. The mixed treatment is appropriate when the disease becomes fully established and after the acute symptoms (headache, etc.) have been partly subdued by mercury. For the dosage of iodides see Treatment of Tabes. The action of the drugs is more prompt in cerebal than in spinal symptoms. In cases of circumscribed gummata, when the medical treatment has failed, operative procedures if accessible should be resorted to, and the removal of the tumor be followed by antisyphilitic treatment.

As an adjuvant to the above treatment may be mentioned: hydrotherapy, dietetic and hygienic measures.

Recently Ehrlich and Hatta have discovered a new arsenical preparation to which they gave the name of **606** and later of **salvarsan.** This has proven to be a powerful remedy which is capable to destroy the spirochætæ with great rapidity. Its usefulness in initial chancre and secondary manifestations is beyond question. In **cerebro-spinal syphilis** salvarsan has also been of considerable value but not to the same extent as in the preceding conditions. While in many cases, especially of cerebral syphilis, it succeeded in relieving the patient's condition with respect to the frequency and intensity of the short transient attacks, nevertheless in a great many cases it did not prevent recurrences.

The drug may be introduced into the body by deep injections in the muscles or directly into the circulation by intravenous method. The intravenous method presents these advantages, that there is no pain and the elimination is rapid, although it may be followed sometimes by a chill, rise of temperature, nausea and vomiting.

Dose and Method of Preparation.—Adult average dose is 0.4 grm. for women and 0.6 grm. for men. Dissolve the drug in 100 c.c. of hot sterile water, add drop by drop a 15 per cent. solution of sodium hydroxide (sterile and filtered) and agitate. A yellow precipitate will form. Continue adding the solution and agitate after each drop until solution becomes clear again. Then add sterile hot filtered water and bring total up to 300 c.c. Each 50 c.c. corresponds to 1 decigram of salvarsan. The injection is to be given at 100° F.

Very recently Ehrlich has recommended a modification of his first discovery. He calls the new preparation **neosalvarsan** or 914. It is given in doses of 0.9 which corresponds to 0.6 of salvarsan. The

solution is made by simple addition of the substance to 300 c.c. of filtered, distilled water at room temperature and injected at this temperature.

In treating with salvarsan it must be borne in mind that the dose may be repeated several times. This should be done when the symptoms are only slightly influenced or especially when Wassermann reaction remains or becomes again positive. It is always advisable to wait after each injection several weeks. In two of my recent cases I had administered three injections at intervals of four, six and eight weeks with progressive benefit.

Contraindications.—Diseases of the heart, kidneys, affections of the optic nerves, cachexia, alcoholism, advanced degenerative changes, are the main contraindications.

Unfavorable By-effects.—The following complications have been observed from the treatment with salvarsan.

Cranial nerve involvement. It seems that there is a special predilection of arsenic for the eighth nerve. Not only salvarsan but also other arsenical preparations show the same characteristic feature. The toxic effect particularly occurs after repeated injections. In another series of cases there may be a simultaneous paralysis of several cranial nerves, such as seventh, eighth and ocular nerves. In one of my cases a facial palsy of peripheral type occurred six weeks following a second injection of salvarsan. In another case I observed two days after the first injection a very marked myosis of the left pupil which remained unaltered two years later.

Heuser (*Mediz. Klinik*, 1911) reports a case of **iritis** in a few days after intra-muscular injection of 0.2 grm. followed by an intra-venous injection of 0.4 grm. of salvarsan, also a case of paralysis of vocal cords and soft palate after an injection of 0.5 grm., finally a case of choked disc with facial paralysis. In the first two cases there were also epileptiform convulsions. Heuser bases his belief in the **neurotropic** action of salvarsan also on the fact that arsenic could be detected in the urine (twenty-three cases) nine months and in the blood two months after the injection. Finger (*Berl. klin. Wchn.*, 1911) reports three cases of optic neuritis after salvarsan.

In a third group of cases **recurrences** of nerve disturbances have been observed after salvarsan injections. It is difficult to say whether the drug is directly or indirectly the cause of the recurrences. These special neuro-reactions are still debatable. They usually occur some time after the injections. It is possible that arsenic has a special irritative local effect on the spirochætæ.

Among other by-effects may be mentioned: cardiac arrhythmia; disturbed function of the bladder, especially retention; constipation; diarrhœa; jaundice (in one of my cases it appeared on the day following the first injection); disturbances of tendon reflexes; peroneus paralysis; epileptiform seizures. Fatalities have also been recorded. They may occur a few days or several weeks after the administration of the drug.

CHAPTER XXII

PARESIS

GENERAL PARALYSIS OF THE INSANE (DEMENTIA PARALYTICA)

It is a parasyphilitic disease characterized pathologically by a diffuse meningo-encephalitis and clinically by symptoms of progressive dementia.[1]

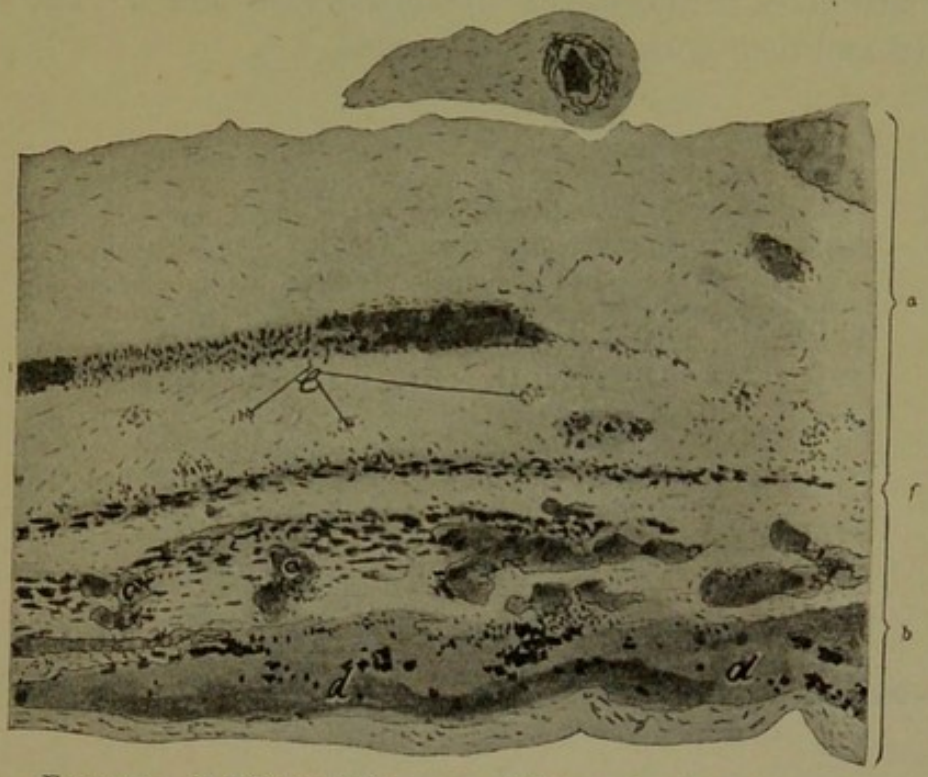


FIG. 114.—OLD CASE OF PARESIS. (*Flatau, Jacobson, Minor.*)

a, dura; *b*, pachymeningitis; *c*, capillaries; *d*, beginning small hematomata in the dura; *e*, leucocytic infiltration in walls of blood vessels and in tissue of dura; *f*, new membrane with pigment.

Pathology.—The **meninges, brain, skull** and **spinal cord** are involved.

Meningitis.—The pia-arachnoid is **inflamed, thickened** and **adherent**

[1] Our former view concerning the relationship of Syphilis to Paresis and Tabes must now be modified. Noguchi and Moore (*J. of Exper. Med.*, Feb. 1, 1913) have found the spirochætæ in the brains of 12 paretics, and Noguchi found them in the posterior columns of a tabetic. In the brain, they are seen more numerous in the cortical than in the white matter, they are scattered in groups between the cells and neuroglia fibers. They are rare in the vicinity of the blood vessels, and not found in the walls of the latter. These findings have been corroborated by Levaditi, Marie and Bankowski (*Presse médic.* No. 36, 1913). The parasyphilitic diseases are, therefore, very probably true syphilitic affections.

to the cortex. When attempt is made to detach it from the subjacent tissue, portions of the latter are torn off. The most favorite seat of the adhesions is the Rolandic area and the bases of the frontal convolutions. The walls of the blood vessels are infiltrated with round cells. Serous fluid in the ventricles and sub-arachnoid space is increased in amount. The space over the optic tracts and crura has a milky appearance.

Encephalitis.—Macroscopically the convolutions are thin and shrunken, the gray substances appear softened and the weight of the entire brain is diminished. Atrophy is particularly observed in the central convolutions, basal ganglia, medulla and pons. Softening which is due to obliterations of small arterioles is diffuse and irregularly distributed. The ventricles are dilated and their ependyma is congested.

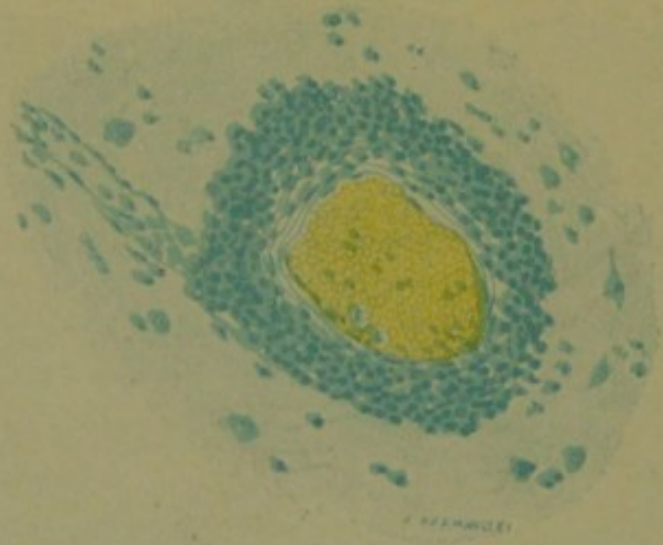


FIG. 115.—PARESIS. TRANSVERSE SECTION OF A CORTICAL BLOOD VESSEL, SHOWING LEUCOCYTIC INFILTRATION. (*Bouchard and Brissaud.*)

Histologically the cells, fibers, neuroglia and blood vessels are found altered.

The cortical **cells** are in a state of **degeneration.** In **acute** stage they are swollen, the chromatophilic substance disappears, the axis-cylinders and protoplasmic processes are enlarged. In **chronic** stage the cells are sclerosed, deformed and disintegrated. The neuro-fibrils, according to some, are pigmented and diminished in number.

The **nerve-fibers** undergo a gradual destruction. At first they become varicose and then lose their myelin, also the axis-cylinder. The cortical and sub-cortical fibers are particularly affected. The neuro-fibrils of the cortical cells are generally affected. The intra-cellular fibrils are more involved than the extra-cellular, also those of the small pyramidal cells more than those of the large pyramidal ones.

The **neuroglia** is in a state of hyperplasia.

The changes of the **blood vessels** consist of leucocytic infiltration of their adventitia and thickening of the other coats, so that eventually they become stenosed and completely obliterated. Similar alterations are found in the lymphatic perivascular spaces.

The above histological alterations, viz. atrophy of neurons and hyperplasia of vessels and connective tissue, are found not only in the cortex, but also in basal ganglia, pons, medulla and cerebellum, although to a lesser degree. Some of the cranial nerves show atrophy and

FIG. 116.—PARESIS. LONGITUDINAL SECTION OF A CORTICAL BLOOD VESSEL, SHOWING LEUCOCYTIC INFILTRATION. (*Bouchard and Brissaud.*)

degeneration. Optic neuritis and atrophy are quite frequent. The third and sixth are not rarely involved. Among other constant pathological lesions should be mentioned a sclerosis of the posterior and lateral columns of the spinal cord, the first more than the second (Anglade, Wyruboff, Klippel).

Less Constant Lesions.—Pachymeningitis (hemorrhagica); thickening of the skull with obliteration of the diploë and formation of exostoses; hyperemia and thickening of spinal meninges with adhesions to the vertebræ; proliferation of the neuroglia in the medulla and cord; atrophy of the cells of the anterior cornua in the cord and of the nuclei of the cranial

nerves (third, sixth, fifth, seventh, also olfactory nerve); inflammation of spinal nerves. The sympathetic nervous system has also been found involved in paresis.

The **peripheral nerves** show parenchymatous degeneration, atrophy and proliferation of connective tissue. These changes are more frequently seen in the lower than in the upper limbs.

The **cerebro-spinal fluid** presents the following characteristics: increase in the amount, lymphocytosis, increase of albumen (Nonne's Phase I), also positive Wassermann reaction (see also page 374).

Symptoms.—Three periods can be considered, viz. (1) initial period, (2) period of full development, (3) terminal period.

1. In the majority of cases the onset is very slow and imperceptible. Gradual changes take place in the **physical** and **intellectual** spheres before the typical symptoms begin to appear. The general appearance of the patient is altered: he is pale, the features are drawn and without expression. **Epileptic** seizures, generalized or unilateral, may occur long before the initial period; they may be motor or sensory; they may be of petit mal or grand mal character. Transient attacks of aphasia are not infrequent. **Palsies** of ocular muscles (strabismus, ptosis, inequality and irregularity of the pupils, diplopia) are common. Sudden **loss of power** in one limb, **increased reflexes,** various **neuralgias,** gastric and vesical **crises, insomnia** are also not infrequent. Various **trophic** or **vaso-motor** disturbances, as, for example, spontaneous fractures, falling out of the hair and nails, various **visceral** disturbances (vomiting, diarrhœa, palpitation, etc.) are observed.

Mentally the patient is changed. He is irritable, **depressed,** indifferent. His memory is weakened. His aptitude for work is diminished and when he makes an attempt, an unusual effort is required. It is difficult to hold his attention in a conversation or transaction. Briefly speaking, he presents symptoms usually found in **neurasthenia.**

Gradually these symptoms increase in intensity and others are added. Instead of being morose and apathetic the patient may be restless and excitable. The least contradiction angers him. He becomes egotistical and his moral sense is blunted: he has no obligations to his family, lies, deceives. Conventional laws are beginning to be ignored by him. Crimes against morality are of common occurrence.

Sometimes the patient shows in the initial stage an exalted intellectual activity. Instead of depression he presents a sense of **well-being.** There is a hyperamnesia, a remarkable power of forming ideas, of creating images, which is noticeable in acts and speech. He plans extravagantly, speaks extravagantly of his fortunes, of his properties. The same

phenomenon is observed in various vegetative functions; exaggerated appetite, sexual and alcoholic excesses are frequent.

These changes in the intellectual and moral spheres are forerunners of oncoming **dementia.** Physical signs begin to become conspicuous at this stage of the disease. An awkwardness in gait, in station, in doing fine work, in handling objects, a slight tremor of the hands, hesitation of speech, are beginning to be noticeable.

2. In the period of **full development** of the disease the physical and psychic symptoms are very marked.

Physical.—1. **Tremor** is constant. It is generalized, but more marked in the hands. It is fine, rapid and intentional. Instead of tremor, there may be only **jerky movements** of the fingers, which are noticeable upon a voluntary act. Tremor and instability are also present in the **tongue, lips** and **muscles of the face.** They are particularly noticeable in attempts to speak. The face when at rest gives the impression of immobile mask, but on the least emotion the facial muscles commence to contract excessively as if they awaken from a latent spasm. This mixture of paresis and spasm is quite characteristic of general paralysis of the insane. It demonstrates a disturbance of mimicry.

2. The **speech** is characteristic. It may be **tremulous** (**ataxic**) or **spasmodic.** In the first case the words are precipitated, then interrupted and then again continued, at the same time hesitated and repeated. In the second case the words and syllables are slowly pronounced and resemble those in multiple sclerosis. There is a dysarthria: the labials and gutturals are particularly affected.

3. The disturbance in **writing** is analogous to that of the speech: omission of letters, syllables, words, mistakes of grammer, repetition, poor spelling of letters, etc., are all typical. In an advanced period the writing is not distinguishable.

4. **Visual** disorders most frequently consist of myosis or mydriasis, irregularity and inequality of the pupils and reflex disturbance. Argyll-Robertson sign is frequent. A paradoxical pupil (response to light and not to accommodation), Piltz's sign (contraction of the pupil at the attempt to close eyelids) may occur. Ocular palsies, diplopia, nystagmus and changes in the eye grounds (optic atrophy) are not rare. According to Uhtoff optic atrophy is observed in 50 per cent. of paretics, while Galezowski's statistics show only 18 per cent.

5. **Muscular** weakness is contant. The face is mask-like, without expression. Sometimes there is loss of control over the facial muscles and the masticated food or liquids run out of the mouth. Paralysis, incomplete or transient, after an **apoplectic** seizure is frequent. Attacks

of monoplegia or hemiplegia, of aphasia, of blindness or deafness, may occur. In the majority of cases they are temporary. An attack of this nature may be accompanied by loss of consciousness. Occasionally and in severe cases an attack may leave the patient permanently hemiplegic. Spasmodic contractions are met with. **Epileptiform** seizures, while more frequent in the first period, may also occur at this stage. They may be generalized or focal. They may affect the sensory motor and vasomotor centers equally. Instead of convulsive attacks there may be fits of **petit mal.** Each attack of epileptiform convulsions leaves the paretic more exhausted than in ordinary cases of epilepsy. The **gait** is usually **ataxic;** it may be also spastic. Romberg's sign may also be present.

6. The **tendon reflexes** are altered in 90 per cent. of cases. In the majority they are exaggerated, in a small percentage of cases lost. Babinski sign is rare, while the paradoxical sign is frequent. This peculiarity lies in the fact that the pyramidal tract is only slightly involved. Babinski sign is usually the expression of a well-defined degenerative lesion, while the reflex described by me is present in the earliest changes of the motor pathway (*J. of Nervous and Ment. Dis.*, 1907). Ankle-clonus and Oppenheim's reflex are rare.

7. **Cutaneous sensibility** is usually diminished. Neuralgia, migraine, paræsthesias may be met with. The **special senses** may be affected. Anosmia, changes of taste, of hearing, are not rare.

8. **Trophic** disturbances, such as falling out of the nails, of the teeth, herpes zoster, bed-sores, arthropathies, spontaneous fractures, are sometimes observed.

Vasomotor symptoms, such as tinnitus aurium, pallor, coldness, gangrene of the extremities, are common. There is a tendency to formation of hematomata. **Hematoma of the ear** is not rare.

9. **Visceral Disturbances.**—They are: increased **digestive** function in the exalted state, diminished in the depressive state, gastro-enteritis; **pulmonary** involvement; **circulatory** disorders (aortitis); **genito-urinary** disturbances (impotence, sterility, suppression of menses); involvement of the **sphincters** of the bladder and rectum (retention or incontinence, frequent or imperative micturition).

10. The **secretions** may undergo changes. Excessive salivation and sweating have been observed.

11. The toxicity of **blood** and it **bactericidal** power are increased.

The **cerebro-spinal** fluid contains quite a perceptible amount of fluid and abundant **lymphocytes.** Wasserman reaction is positive and there is increase of albumen (Nonne's Phase I).

12. The **general nutrition** as a rule is lowered at first, then gradually improves and the patient becomes stout. Later on there is again a loss which finally ends in cachexia.

Psychic Symptoms.—Progressive enfeeblement of the mental faculties, viz. progressive dementia, which is accompanied by symptoms of other psychoses, is characteristic of paresis.

Amnesia predominates. In the initial stage the loss of memory affects recent events. In the second period it concerns also old events. The patients forget even their age, birthplace, etc.

Judgment, power of attention, will power are all markedly impaired. The patient is easily influenced, because of defect in the will power. As mental self-control is defective, the patient succumbs to the influence of impulse. Outbreaks of emotional nature are then seen. Theft, assaults, homicide, etc., are not rare. Performance of delicate acts is deficient.

A very important symptom is the **inability to recognize one's own infirmity** in spite of the exuberant spirit. Such a patient does not appreciate the fact that he is ill, that he fails in performing his work. The want of knowledge of this infirmity leads to formation of ideas of self-importance, of **expansive ideas.** The latter are very common in paresis. The expansion or exaltation is noticeable in psychic, motor, sensory and vegetative spheres. The patient believes himself as possessing millions of dollars, of properties, of friends, of enemies. He is famous, his name is mentioned everywhere; he is a prophet, a god, etc. He is restless, does not sleep, eats abundantly, shows a tendency to excesses of all kinds. Gradually the moral sense becomes obtunded. The patient is unable to form new connected ideas, unable to orient himself; he is indifferent and becomes **automatic** in his acts. The latter are foolish, childish. The further the dementia advances, the less he is capable to take care of himself; he is unclean about his person, indecent, uses profane language irrespective of the surroundings.

During the progressive development of the dementia other symptoms are observed, viz. delirium, confusion, delusions with or without hallucinations.

The **delusions** in paresis are usually vague, unsystematized, unstable. Sometimes they are systematized and fixed, similar to those of paranoia. They may be depressive, persecutory or else expansive. They may be also hypochondriacal. Hallucinations not infrequently accompany the delusions. **Hallucinations** of various senses are not rare in paresis,especially of sight and hearing. One of my patients had an olfactory hallucination: he smelled feces at the approach of food.

A delirious state with great excitement and restlessness occurs episodic-

ally during the entire course of paresis. A confusional state is present, particularly in the second stage of the disease.

The various delusions just mentioned may sometimes **simulate** other psychoses, but they never present the characteristic and well-defined features of the latter.

3. **Terminal Period.**—The physical and mental symptoms just described progress gradually toward a terminal stage, which is characterized by complete deterioration and relaxation of sphincters. The hebetude, dementia, difficulty of speech, pupillary symptoms, ataxia of locomotion, tremors, have all reached the climax. New phenomena develop. Paralysis of the vesical and rectal sphincters, paralysis of the pharynx, trophic disturbances, viz. bed-sores, render the paretic completely helpless and hasten his death. Apoplectiform and epileptiform attacks are frequent at this stage of the malady and may be the immediate cause of death. Not infrequently death occurs from an intercurrent disease (pneumonia or others).

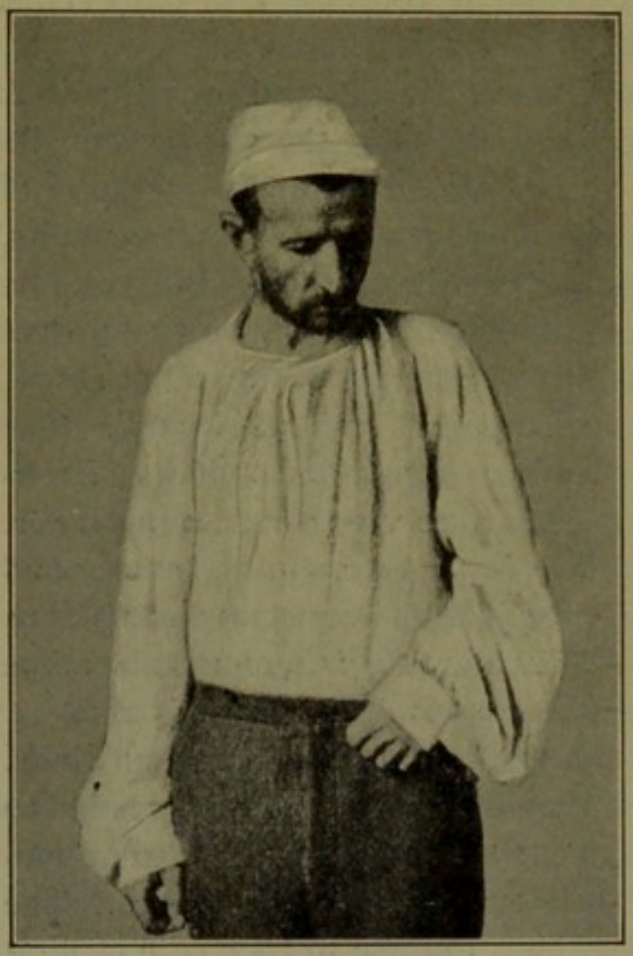


FIG. 117.—DEPRESSIVE FORM OF PARESIS. EXPRESSION SAD, PREOCCUPIED AND DEMENTED. (*Bouchard and Brissaud.*)

Forms.—(*a*) **Simple Demented Form.**—It is very frequent. It is characterized besides the typical physical symptoms by a gradually developing dementia from the beginning to the end and not accompanied by delusions. The dementia is usually not marked, so that the patient continues for a certain period his usual occupation. Epileptic seizures are very frequent in this form of paresis.

(*b*) **Depressive Form.**—Marked mental depression is characteristic. Delusions of persecutory or hypochondriacal nature are present. In some cases the latter are so pronounced that melancholia may be thought of, especially when the ideas of unpardonable sin or impending evil and tendency to suicide are predominating. However these delusive ideas are unstable and do not present **on a whole** the picture of genuine melancholia. In this form there is a marked tendency to obesity.

Remissions are not so frequent as in the following form.

(*c*) **Expansive Form.**—It is characterized by an exalted general attitude. Even in the absence of distinct delusions the patient's manner and expression betray a self-satisfaction. The paretic is happy, contented with everybody and everything. Sometimes the expansion may increase to such an extent as to cause marked restlessness and excitement, so as to simulate mania. However, taken as a whole, it is not a genuine mania. As to the delusions, they are of the exalted type. The patient is a multimillionaire, prophet, god, great actor, writer, etc. (see above). In spite of his great self-importance, he is very easily influenced and is submissive.

Fig. 118.—Expansive Form of Paresis. Expression Happy. (*Bouchard and Brissaud.*)

(*d*) **Circular Form.**—It is characterized by alternation of depressive and expansive states. It may be confounded with the manic-depressive psychoses. An examination for physical signs and repeated observation will decide the diagnosis.

(*e*) **Galloping Form.**—In rare cases rapid evolution of symptoms is observed. The mental manifestations are characterized by rapid deterioration of mind, loss of control of the sphincters, bedsores, exhaustion and death. It is possible that the symptoms of paresis in such cases had been long at work and because of mildness of the manifestations they were overlooked. Under the influence of some exciting cause, the symptoms suddenly assumed a rapid course and promptly reached a fatal termination.

(*f*) **Juvenile Paresis.**—It is occasionally observed. In one of my cases, a young man of eighteen, the symptoms began to show themselves at the age of twelve. Signs of mental enfeeblement appeared first. Speech disturbances of the typical paretic variety developed gradually two years later. At the time I saw him, he presented the characteristic fine tremor, paretic speech, ataxia, exaggerated knee-jerks, irregular and

unequal pupils and depression with hypochondriacal delusions without hallucination. He never had epileptic or apoplectic seizures. He is still living, but his dementia is getting progressively deeper. Wassermann reaction was strongly positive.

Course, Duration, Prognosis.—Paresis is essentially a **progressive** disease. Its course may be **slow** or **rapid.** The course of the simple demented form is extremely slow. The expansive form runs a more rapid course than the depressive form. Complications, such as apoplectiform or epileptiform seizures, visceral complications, hasten the course of the disease.

Paresis may present periods of arrest and improvement, so-called **remissions.** The latter consist of a partial or complete disappearance of the mental manifestations. These, so to speak, lucid intervals may occur at any of the three periods of the disease. They may last from a few days to weeks, months and even years. They are usually the result of a proper and early instituted treatment. **Remissions** sometimes follow a traumatism or a protracted suppuration. In the majority of cases the physical symptoms persist. I have seen cases in which very great improvement of the physical signs also occurred during remissions. The **duration** of paresis is from a few months to several years (five and ten years). The second stage is the longest. The **prognosis** is unfavorable. Death may result from the invariably progressive course of the disease, ending either with a disease of the lungs (tuberculosis, pneumonia), diarrhœa, bed-sores or extreme exhaustion. It may also end accidentally: suffocation from impaction of food in the larynx during an epileptic seizure; fractures of bones; finally suicide.

Sudden death has also been observed. In such cases either there is an element of alcoholism, or a cardiac pathological condition, or a cerebral hemorrhage. In some rare cases no perceptible cause could be found. Sudden death occurs more frequently in middle aged individuals than in elderly or young persons.

Diagnosis.—As the onset and various forms of paresis may simulate other diseases, the differential diagnostic points must be emphasized.

The **neurasthenoid** state of the first period will be distinguished from genuine neurasthenia by the following considerations:

A neurasthenic is capable of presenting a detailed account of his ills. He exaggerates his symptoms, it is true; nevertheless they are recited in the most connected manner. The patient repeats his recital often, but this repetition is not because of a genuine amnesia, but because he is anxious to analyze his troubles and to draw his physician's special attention to them. The neurasthenic appreciates his illness keenly.

The paretic is incapable of giving a connected history of his trouble. He frequently interrupts his speech and forgets what he said previously. When reminded he does not attach any importance to it. Frequently the expression of his face does not correspond to the subject of his conversation: he may show a smile while speaking of death. A true neurasthenic appreciates keenly his trouble, while the paretic is incapable of recognizing properly the ills of which he speaks. In a true neurasthenic mental operations are readily exhausted but not disturbed or modified. The paretic presents a genuinely diminished intelligence. Want of power of criticism and of judgment, grave omissions, errors in daily acts, change of moral personality—are all met with in paresis but not in neurasthenia. Besides, the cerebro-spinal fluid presents certain characteristics in paresis (see page 375), which are not present in neurasthenia. Wassermann test on blood or on cerebro-spinal fluid is positive in paresis, but not in ordinary neurasthenia.

The delirious state, the confusional state of the paretics may be confounded with similar states occurring in intoxications. These are the so-called cases of **pseudo-paresis** (alcoholic, saturnine and others). If the diagnosis is difficult at the beginning, a subsequent study of the cases will clear it up. The delirious, confusional states are only transient and the obnubilation of intelligence is not as profound in intoxications as in paresis.

The **alcoholic** subject usually suffers from hallucinations of a terrifying nature which become accentuated at night. He usually appears brutal and stupid, but not demented. The paretic presents a reverse condition. The alcoholic appears sad and preoccupied, the paretic indifferent and apathetic. If an alcoholic develops expansive delusions, they have to be brought out and can be ascertained only on a skillful questioning of the patient. The paretic does it unsolicited. The speech of the paretic is ataxic, full of repetition of syllables and words. The speech of the alcoholic is merely embarrassed, thick and tremulous. Withdrawal of intoxicants is followed by a progressive amelioration; in paresis the symptoms are essentially progressive. Lymphocytosis is absent and Wassermann test is negative in alcoholism.

Symptoms of **lead intoxication** (encephalopathy) may resemble paresis especially by the psychic manifestations. In both affections a neurasthenic state and mental attitude suggest an oncoming dementia, but in encephalopathy the dementia seems to dominate the entire situation from the onset. The physical signs, such as cachectic appearance, difficulty of speech of a gross nature, motor disorder of paralytic nature affecting chiefly the extensor groups of muscles, abdominal colic, severe

headache, the blue line on the gums, interstitial nephritis, arterio-sclerosis —are all found in lead intoxication. Finally, the regressive evolution of symptoms after an appropriate treatment and removal from the toxic influence is characteristic of lead intoxication.

Lymphocytosis, increase of albumen in the cerebro-spinal fluid, positive Wassermann reaction, are characteristic of paresis, but absent in lead encephalopathy.

Diffuse **cerebro-spinal syphilis** cannot always be easily differentiated from paresis. In both affections a neurasthenic symptom-group exists. Cerebro-spinal syphilis produces a profound neurasthenic state which resembles appreciably paresis, especially in the initial stage of the latter. The diagnostic difficulty is especially great, when in both affections Wassermann and Noguchi reactions are positive and lymphocytosis is present.

In cerebro-spinal syphilis the onset is usually acute, in paresis slow and insidious. In the first a marked diminution of intellectual functions is present at the onset, in the latter the intellectual defect is less marked at first. In the first there is a continuous severe headache which is worse at night, in the latter this is not the case. In the first there is intellectual inertia, profound apathy, somnolence and a special dulness in the facial expression. Such a picture is not seen in paresis. There is never in cerebro-spinal syphilis the optimistic carelessness and general exaltation in the motor and sensory spheres which are observed in the expansive form of paresis. Alongside of the apathy and loss of memory the reasoning power may be totally preserved in the first, but this is not observed in paresis.

Cerebro-spinal syphilis is usually accompanied by local symptoms. Very early are observed ocular changes, such as optic atrophy, contraction of visual fields, palsy of other cranial nerves; symptoms of spinal cord involvement, such as exaggerated knee-jerks, toe phenomenon, ankle-clonus, sensory changes, involvement of sphincters. The course of cerebro-spinal syphilis is characterized by great variability and mobility of the symptoms which become either exaggerated or ameliorated. In paresis the symptoms are invariably progressive. The antisyphilitic treatment has a remarkably rapid effect on the symptoms of cerebro-spinal syphilis. Such a course of events is not observed in paresis. The richness of symptoms in syphilis is such that it is possible in almost every case to reveal some particular feature indicative of the existence of a localized focus in the brain and spinal cord. The incomplete, unequal and regressive character of cerebro-spinal syphilis is different from the

global and progressive character of paresis. Variability and capriciousness of the symptoms are typical of syphilis.

Paresis should also be differentiated from tabes, from multiple sclerosis, tumors of the brain. The special symptoms of these affections will aid in the diagnosis.

Etiology.—The chief **determining** cause is **syphilis.** It is not in the acute stage of syphilis that paresis develops, but years after the initial infection (from five to fifteen years). Since the discovery of Wassermann's reaction and in view of the findings of Spirochætæ in paretic brains by Noguchi and Moore (page 353), paresis has proven to be without reservation a syphilitic affection. Plaut (*Allg. Zeist. f. Psych. u. Neur.*, 1909), for example, obtained in every one of his cases of paresis without a single exception a positive Wassermann reaction. In a number of cases in which a history of syphilitic infection cannot be obtained, the Wasserman test is of utmost value. While formerly such cases kept us in doubt as to syphilis being the only cause of paresis, now with Wasserman's discovery all hesitation must be set aside. Juvenile paresis showing a positive Wassermann reaction proves that **congenital** syphilis plays also a powerful rôle. The following peculiarities concerning the relation of the reaction to Paresis deserves special mention. (1) If the blood serum does not give a positive reaction, the cerebro-spinal fluid will be negative. If the cerebro-spinal fluid gives a positive result, the serum does the same. (2) If the serum gives a positive result, but the cerebro-spinal fluid a negative, the disease is not paresis, but cerebro-spinal syphilis. (3) The reaction of the cerebro-spinal fluid is more important in paresis than that of blood serum. (4) Mercury, iodides, salvarsan may render Wassermann reaction negative for a certain time.

The **predisposing** factors are: undue and prolonged mental efforts, worry and anxiety; venereal and alcoholic excesses; traumata of the head; sunstroke. Men are more frequently affected than women. The age between thirty and forty-five is the most favorite. Juvenile paresis may occur as early as twelve years of age. As to the countries, the higher the degree of civilization, the larger the number of cases. The question of neuropathic hereditary influences is not entirely settled. However, not infrequently a history of some neurosis or insanity is traced in the antecedents of paretics.

Treatment.—As soon as paresis is recognized or even suspected in its initial period, an antisyphilitic treatment should be instituted (see Treatment of Syphilis of the Nervous System). However, in spite of the fact that paresis is a syphilitic disease, not much can be expected from the specific medications. The value of Ehrlich's new remedy, salvarsan,

has been sufficiently tested to warrant some definite conclusions. If it does any good at all, it improves sometimes the general health, but it has no effect whatever upon the course of paresis. It may be given in conjunction with mercury and iodides. The treatment should be instituted as early as possible. However, I have seen favorable results in prolonged treatment with iodides given progressively in very large doses (300 or 500 grains daily). In view of the fact that some authors observed unfavorable results from iodides, the increase of the dose should be made very cautiously and upon the least sign of intolerance the drug should be discontinued (see also Treatment of Tabes). My favorable results are meant only from the standpoint of some acute symptoms, such as headache, increase of tremor, insomnia, disturbance of hearing or of sight, also ataxia. It is worth while mentioning the fact that administration of salvarsan renders the Wassermann reaction negative. In spite of this the course of paresis is not altered.

The hygienic and dietetic measures are more important. As soon as possible a paretic should be removed from his usual occupations. A quiet life (better in a sanitarium or in the country), avoidance of excitement and worriment, total abstention from stimulants, including tea and coffee, regularity in meals and sleep, a diet free from articles which are likely to produce fermentation, finally hydrotherapy (brief douches, baths, etc.) are all extremely beneficial. Any disturbance of the gastro-intestinal tract, of renal function, of sleep, should be immediately combated. Sexual intercourse must be avoided.

If this plan of treatment is strictly carried out, **remissions** will occur early in the course of the disease and if a remission is not considered a cure (which is frequently done otherwise) and the above general treatment kept up with the same regularity and energy, the remissions may be prolonged for months or years.

EARLY PARESIS

Early Paresis.—The recognition of paresis in its earliest stages is of utmost importance both from a practical and medico-legal standpoints. At this stage the symptoms may be so slight that they are easily overlooked, proper measures are neglected and deplorable consequences follow. The patient is permitted to be at large, to travel, to transact business, to govern. It is only when grave errors are committed that attention is drawn to the patient's condition.

Psychic Status.—The most delicate and the highest mental functions are invaded the earliest. They are therefore particularly noticeable in persons of culture. It is in the fulfilment of fine acts necessitating the

most delicate discrimination that lapses and omissions are observed. Memory is affected early in the disease. It is slight and observed only at intervals in words and acts. Soon the patient shows omissions in his daily work; his writing shows lapses and repetition in letters, syllables and then in words. The amnesia leads to neglect in his own appearance. Punctuality of habits is neglected and the patient becomes indifferent. Usual reservation in language is replaced by loquacity and unreasonableness. He is conscious of the defect of his memory but he is not disturbed by it in the least. He does not try to avoid errors. He even ridicules his own mistakes. This indifference toward the amnesia is characteristic of early paresis.

At the same time changes appear in the character, disposition and moral personality. The previously cheerful person becomes depressed, irritable, apprehensive. He loses interest in everybody; is neglectful of his duties, of his home. He resembles then a neurasthenic, but the resemblance is only apparent (see differential diagnosis).

In other cases a totally opposite condition is observed. The usually conservative man has become exuberant in actions and spirits; he makes ambitious schemes, spends money in an unusual way. This exaltation is manifested not only in the intellectual and motor spheres, but also in the affective sphere. The paretic is self-contented, optimistic and he shows a tendency to excesses. Immorality is the natural consequence; sexual perversion is not an infrequent phenomenon. The alterations of the moral personality occurs often long before the intelligence is seriously affected.

The exuberance is only superficial, it lacks in depth. With all his agitation the early paretic is not capable to accomplish much. The contrast between what he appears to be able to do or between what he promises to do and what he actually does is striking. He is easily fatigued. He is always ready to undertake work, but is unable to persist in it.

A characteristic feature is the **inability** of the patient **to realize his infirmity** in spite of his exuberant spirit. He does not appreciate the fact that he cannot continue his usual occupation. Another interesting phenomenon is the **early defect of criticism.** When the patient is confronted with the most evident manifestations of his affection, he does not pay the slightest attention and rather ridicules them.

To **sum up,** defective memory, weakened faculty of attention, of judgment, of criticism, perversion of moral sense, of disposition, radical change in conduct, inversion of affective faculties—all constitute characteristic psychic manifestations of the early period of paresis.

Somatic Symptoms.—**Apoplectic seizures, epileptic seizures,** attacks of **aphasia**—are the three groups of somatic manifestations that not infrequently announce the oncoming paresis.

The **apoplexy** may consist only of sudden mental hebetude, or of a genuine loss of consciousness followed by monoplegia, hemiplegia with or without aphasia. These phenomena are usually brief and temporary.

Epileptic seizures are mostly of **petit mal** form. They may be also of **grand mal** form, generalized or local. In the latter case (local) they may be of the sensory variety: sudden paræsthesias, such as tingling or burning on one hand, on one side of the face.

Aphasia may occur without hemiplegia. The attack is sudden and transitory, but it has a tendency to recur.

Pupils.—In fifty out of fifty-nine patients studied by me from the standpoint of early paresis the pupils were found **irregular** and especially **unequal**. Sluggishness of light reflex is another frequent symptom. Ocular palsies are not very infrequent, but they are usually temporary.

Reflexes.—The knee-jerks are mostly exaggerated. Babinski's sign is rare. Paradoxical reflex is not infrequent.

Speech and tremor are both rarely altered in early paresis.

Early paresis presents a specially important chapter from the **medicolegal** standpoint. By reason of gradually developing moral and affective perversions which announce the onset of the disease, the initial period of paresis is the real medicolegal period. It is then that misdemeanors are frequently misinterpreted and that the individuals are sent to prison. Early paretics not infrequently marry. The new life the patient leads in association with sexual excesses aggravates the condition. Early paretics are able to carry on their former habits and other acts concerning their usual occupation, but they do it automatically. The slight lapses of memory, the digression in their conduct apart from their regular work, the changes of disposition and sentiments—are usually all attributed to fatigue or absent-mindedness. They are permitted to have liberty of action and to continue in responsible positions until misconduct of grave nature and consequences occurs. It is at this period that illegal acts and crimes are most frequently committed. Its recognition and proper interpretation is a matter of grave importance. Civil capacity and legal responsibility should be considered as having no existence as soon as the earliest signs of paresis begin to make their appearance.

CHAPTER XXIII

LUMBAR PUNCTURE AND CEREBRO-SPINAL FLUID

Lumbar puncture is a necessary procedure at bedside. It gives an opportunity for study of the cerebro-spinal fluid besides the therapeutic effect procured by extraction of a certain amount of the latter and by injection of soluble drugs or specific sera into it.

Anatomical Points.—The arachnoid sac which does not contain the spinal cord, but contains only the nerves of the cauda-equina, extends normally from the second lumbar vertebra to the second sacral vertebra. It is between these two extreme points that a puncture of the sac can be made. As, in children especially, the cord reaches lower down, it is advisable in every case not to puncture between the second and third lumbar vertebræ. The most reliable place is the space between the fourth and fifth lumbar vertebræ. A transverse line drawn between the crests of iliac bones passes exactly at the level of the spinous process of fourth lumbar vertebra. Immediately beneath is the fourth lumbar space. It is there that the puncture is made.

Technic.—The position to be given the patient is variable: some prefer a lateral position, others prefer a sitting position. The latter presents certain inconveniences, namely a too rapid issue of the fluid or fatigue of the patient. When the patient lies on his side, he is placed as near as possible the edge of the bed; the head is slightly raised on a pillow, the thighs are flexed as strongly as possible over the pelvis so that a marked posterior convexity of the lumbar spine is obtained and thus a maximum separation of the two vertebræ is produced. The skin is then washed thoroughly and painted with tincture of iodin. Rigorous antiseptic precautions must be observed by the operator with regard to his hands and the instrument to be used. Local anesthesia is not necessary. The trocar or a needle of 9–10 cm. in length and 0.8 mm. in diameter must be used. It must be fine but at the same time solid. In muscular or insane individuals a very strong needle is necessary. The operator then places the index of the left hand on the space to be punctured. The needle is then seized firmly with the right hand and plunged at a half of a centimeter laterally to the place outlined with the finger of the left hand. Slowly but progressively the needle is advanced toward the median line and slightly upward. At a distance of about 4 to 6 cm. in an adult and

of 1 to 3 cm. in a child the needle reaches the spinal canal and perforates the arachnoid sac. The cerebro-spinal fluid appears immediately at the outer end of the needle. When the desired amount is collected in a sterile tube, the needle is withdrawn abruptly, the puncture of the skin is obliterated with collodion. It is not always easy to enter the spinal canal. Frequently the needle strikes against the vertebræ; the needle is then slightly withdrawn and with a slight deviation from the original direction will eventually penetrate the canal. Sometimes it is necessary to withdraw the needle entirely and renew the operation. It may happen that instead of cerebro-spinal fluid blood appears at the outer end of the needle. The blood frequently rapidly disappears and clear fluid makes its appearance. If the blood persists, the needle must be withdrawn and a new puncture made; the blood was probably due to a puncture of a small intradural vein. Sometimes toward the end of the operation the patient is seized with **cramps** in the thighs; they are due to compression of some nerve filaments of the cauda equina. They do not last long.

Accidents.—Lumbar puncture is, generally speaking, an innocuous operation. However, untoward symptoms have been observed. Vertigo, headache, nausea, vomiting, pain in the back, convulsions may occur, but they do not last longer than a few hours. Occasional death has also been observed especially in cases of cerebral tumors and when a too large amount of cerebro-spinal fluid was withdrawn. Sicard has formulated the following precautions to be observed in lumbar punctures.

(1) Avoid lumbar puncture in brain neoplasms in which headache, nausea, vertigo increase when the patient is in dorsal position.

(2) Before the puncture keep the patient in bed for 24 hours.

(3) Always use the lateral position for the operation.

(4) After the puncture keep the patient in dorsal position during 48 hours with the head but slightly elevated.

(5) Except in special cases, withdraw only 4 to 8 c.c. of the fluid without aspiration.

(6) Use a very fine needle 0.8 or 0.9 mm. to reduce to a minimum the meningeal injury.

In cases of brain tumor observe the following rules:

(1) Before the puncture, observe horizontal position in bed, the head but slightly raised, for 48 hours.

(2) Puncture in lateral position, the head slightly lowered (Trendelenburg's position).

(3) After the puncture keep the same position with the head still lower for 24 hours; then horizontal position for 48 hours.

CEREBRO-SPINAL FLUID

The study of cerebro-spinal fluid offers a great deal of information from a diagnostic standpoint.

Cerebro-spinal fluid closed up in the ventricles and in the subarachnoid spaces is secreted by the choroid plexuses.

Physical Properties.—Normally the fluid is perfectly clear. In pathological conditions it is generally **turbid, purulent** or lacteous, or else in exceptional cases also clear. In **tubercular** conditions it is floccular and coagulates in large lumps.

The **color** may change. When it is red, intra-meningeal hemorrhages are to be suspected. Whether the hemorrhage is of cerebral, bulbar or meningeal origin, or else in cases of contusions or fractures of the skull, the cerebro-spinal fluid will present either a frankly hemorrhagic color or a yellowish or greenish discoloration. Sometimes however the bloody fluid may originate from the tissues through which the needle passes before it reaches the spinal canal. The following differential features have been emphasized by Tuffier.

(1) The blood from an accidental cause coagulates; when it is from the cerebro-spinal fluid it does not coagulate, but presents a sediment at the bottom of the tube.

(2) If the bloody fluid is centrifugated and presents a clear aspect, the hemorrhage was accidental and due to the puncture of the external tissues. On the contrary, if the fluid remains colored, the blood existed in the subarachnoid cavity.

In the course of acute cerebro-spinal meningitis the cerebro-spinal fluid may be of yellowish color. The latter is probably due to small meningeal hemorrhages.

Tension.—In cerebral tumors, in meningitis, in syphilis (secondary period), in epilepsy there is increased tension.

Chemical Properties.

Reaction.—Normally it is alkaline.

Albumen.—In normal condition albumen is either absent or it is present in extremely small quantities. In pathological states there is increase of albumen. In Paresis, Tabes, in Syphilis of the Nervous system albumen content is increased. The majority of acute states present hyper-albuminosis, such as tubercular meningitis, cerebro-spinal meningitis, meningeal symptoms accompanying infectious diseases and intoxications (lead). When the quantity of albumen in above **0.25 or 0.30 grm.** it is an indication of **meningeal reaction.**

Glucose.—Normally there is 0.50 gm. of sugar. Diminished quantity is indicative of meningitis, especially cerebro-spinal and tubercular.

Chlorids.—Normally there is 7 grm. of chloride of sodium. Diminished quantity is found in meningeal conditions, especially in the tubercular form.

Cytological characteristics.—In normal condition the cerebro-spinal fluid contains very few cellular elements. There may be a few rare lymphocytes, but no polynuclear cells. As soon as the meninges are inflamed, rapidly a **lecocytosis** develops. Lymphocytes and polynuclear are the two forms that figure prominently in pathological conditions. Speaking generally, according to Sicard **polynucleosis** is an indication of a grave pathological condition, lymphocytosis of a less serious state or of a chronic condition. But to this rule there are many exceptions.

Polynucleosis is observed in the acute stage of epidemic cerebro-spinal meningitis, sometimes also in the beginning of tubercular meningitis. The number of cells is in relation with the intensity of the disease. When the latter improves, the polynuclears are gradually substituted by lymphocytes. Polynucleosis accompanies meningeal infections of a strongly microbic nature (except tubercular), viz. **epidemic cerebro-spinal, other forms of meningitis, cranial traumatism, otitic complications. Tubercular meningitis** is essentially a **lymphocytic** condition.

Syphilis at any period (except the chancre) presents essentially a lymphocytosis. In acquired syphilis, specific meningitis acute or chronic, specific meningo-encephalitis, specific neuritis, also in hereditary syphilis —lymphocytosis is characteristic. In Parasyphilitic diseases, tabes and Paresis, lymphocytosis is constant. In Paresis during the paroxysms a temporary polynucleosis is observed. The same lymphocytic formula is observed in acute anterior Poliomyelitis, in Landry's paralysis, in Herpes Zoster, in Meningo-myelitis.

Total absence of leucocytic reaction is observed in softening and hemorrhage of the brain, cerebral tumors, syringomyelia, disseminated sclerosis, myopathies, neuritis, provided there is no syphilitic factor.

Chorea presents not infrequently lymphocytosis because not infrequently it is associated with a meningeal reaction.

Bacteriology.—Microörganisms may be discovered in the cerebro-spinal fluid either by **direct examination, culture** or **inoculation.** Great varieties of microbes have been found: streptococcus, pneumococcus, Eberth's, Löffler's and Pfeiffer's bacilli and coli-bacillus. The most interesting ones are: **meningococcus** of Wechselbaum and **tubercle bacillus.** The first is the specific microörganism of the epidemic cerebro-spinal

meningitis, the second of tubercular meningitis (see the respective chapters).

Wassermann Reaction.—In 1901 Bordet and Gengou discovered the fundamental principle known as "absorption or deviation of the complement." They introduced the terms **antigen** and **antibodies** to designate: the former—a substance which injected into an animal will produce an immune serum; the other—an antagonizing substance which is necessary for the immunizing action of the serum. There is a complement in every normal serum which is specific for each animal. Moreover if the blood corpuscles of one animal are injected into another of a different species, these corpuscles disappear with the production in the serum of a specific hemolysin. If the antibody or the complement is removed, the specific hemolytic action of the serum is lost. If the antibody is linked up to the antigen in the presence of the complement, both the antibody and complement become inactivated. The method of determining if the blood serum or cerebro-spinal fluid contains the antigen or the antibody is known under the name of deviation of the complement. The determination of the presence of syphilitic antibodies and antigen constitutes the basis of Wassermann's test.

Wassermann reaction is regarded at present one of the most valuable assets in medicine. It enables to decide difficult and perplexing diagnoses in obscure cases. It is extremely useful in Syphilis and Parasyphilis of the nervous system. It is the consensus of opinion that when it is positive, it means syphilis, but when the reaction is negative, it does not absolutely exclude syphilis, as some factors may influence the reaction. There is at a present sufficiently large number of facts proving that in latent tertiary syphilis the Wassermann reaction is the more rarely observed the more energetic is the treatment with mercury. A positive reaction changes into a negative one under the influence of mercury. Bizzozero (*Mediz. Klinik*, 1910) made investigations with reference to iodides. In a series of cases he observed that when under the influence of iodides the morbid symptoms improve, the positive Wassermann reaction equally becomes negative. Nichols and Craig (*J. Am. Med. Ass'n.*, 1911) have shown that after ingestion of considerable amounts of alcohol within twenty-four hours of making the test a positive serum may be rendered negative, and in some cases the serum will strongly give a negative reaction for as long as three days after the administration of alcohol. A careful inquiry therefore should always be made regarding the recent use of alcohol before the Wassermann test is made.

On the preceding pages the importance of the leucocytic formula in the cerebro-spinal fluid was strongly emphasized. It is interesting to ob-

serve that there is no parallelism between the leucocytic reaction and Wassermann reaction. The first, for example, may be very marked and the Wassermann test negative for the cerebro-spinal fluid.

Diagnostic Significance of Four Reactions in Organic Nervous Diseases.—Wassermann reaction in blood serum and cerebro-spinal fluid, also lymphocytosis and increase of albumen contents in the cerebro-spinal fluid appear to have an important value for differential diagnosis. Nonne (*Deutsch. Zeitschr. f. Nerv.*, 1911) has recently made a complete study of these four reactions in 167 tabetics, 179 paretics, ninety-seven individuals with cerebro-spinal syphilis, sixty-eight cases of multiple sclerosis, thirty-eight cases of brain tumors, fourteen cases of spinal tumors. His important conclusions are as follows:

1. **Lymphocytosis** and **increase of albumen** (Phase 1). There is almost constant presence of lymphocytosis in syphilis and **parasyphilis**; it is less abundant in parasyphilis than in syphilis of the nervous system.

In a few isolated cases there is absence of lymphocytosis. In eleven cases of tabes the latter was absent; four of these cases were stationary, seven in incipient period.

Among the syphilitic forms, specific hemiplegia is most frequently without lymphocytosis and without increase of albumen. The spinal forms of syphilis always present lymphocytosis and Phase I.

In organic nervous diseases other than of syphilitic or parasyphilitic character these two reactions are slight. However, in two cases of **Multiple Sclerosis** the first reaction alone was present.

In **brain tumors** the two reactions are usually absent or very slight.

In **tumors of the cord** seven times out of fourteen Phase I was present, but lymphocytosis absent.

In syphilis without involvement of the nervous system the two reactions are observed in 20 to 30 per cent. The two reactions have a practical bearing: they help to ascertain whether the nervous system is involved. In neurasthenics, alcoholics, epileptics, whether they are syphilitic or not, the amount of albumen is normal. The increase of albumen has no absolute value for a differential diagnosis of syphilitic and parasyphilitic diseases of the nervous system, but it permits to differentiate functional from organic nervous diseases. Lymphocytosis has no absolute value for differentiation of syphilitic and parasyphilitic affectious.

II. **Wassermann Reaction with Blood Serum and Cerebro-spinal Fluid.**—(a) A positive **serum reaction** is, with very rare exceptions, an indication of syphilis. Nonne found it in 60 to 70 per cent. of tabetics, in 90 to 95 per cent. of paretics, in 80 per cent. of syphilitic arteritis. He be-

lieves that a positive serum reaction controls the prognosis in syphilitic individuals. He believes that a negative Wassermann is almost a certain indication that there is no paresis. The degree of the positive Wassermann has a certain significance. A very strong positive reaction is more frequently observed in paresis than in tabes or cerebro-spinal syphilis.

(b) A positive reaction with the **cerebro-spinal fluid** is observed in 100 per cent. of paresis. The frequency is less marked in tabes and still less in cerebro-spinal syphilis. The test with cerebro-spinal fluid is more important in paresis than with blood serum. When serum gives a positive result, but the cerebro-spinal fluid a negative, the disease is not paresis.

With these four reactions errors of diagnosis may be easily avoided.

THERAPEUTIC INDICATIONS WITH REGARD TO CEREBRO-SPINAL FLUID.

A. **Lumbar Puncture and Withdrawal of Fluid.**—The withdrawal of cerebro-spinal fluid, which should not be above 20 to 30 c.c., is indicated in the following conditions.

In congenital **Hydrocephalus** some favorable results had been reported. Permanent or temporary improvement in **uremia** has been observed. In **tumors** of the cerebrum or cerebellum relief of headache, vomiting and œdema of the papilla has been reported by very competent observers. However, the greatest care must be exercised in such cases and only very small quantities may be withdrawn, as rapid decompression may produce a fatal syncope. **Epileptic seisures** have been ameliorated in intensity and frequency from occasional withdrawal of small amounts of cerebro-spinal fluid.

The main indication for lumbar puncture is in **Meningitis.** In acute meningeal conditions, in tubercular meningitis, amelioration of symptoms has been noticed. The most favorable results have been obtained in non-tubercular forms, cerebro-spinal, serous, purulent and syphilitic forms of meningitis.

In some cases of serous meningitis excellent results have been obtained after one or several punctures. In **Syphilitic headache** good results have been obtained. Lumbar puncture has a distinct therapeutic value in meningitis of bacterial origin but non-tubercular.

B. **Injections of therapeutic agents** into the sub-arachnoid cavity.

The sub-arachnoid method of injection is superior to the subcutaneous one in view of the greater absorbent power of the sub-arachnoid cavity. This fact is being utilized for introduction into the latter of drugs and sera. Antitetanic serum, antimeningococcus serum—have both proven

to be of great utility. Sulphate of magnesia is also injected into the sub-arachnoid cavity in cases of tetanus with satisfactory results. Cocain, eucain, novocain, stovain, have been injected for analgesic purposes; they produce a rapid, complete and sufficiently durable anæsthesia to permit operative procedures on the lower extremities and some abdominal viscera. Certain inconveniences have been observed, viz. vertigo, headache, nausea—of which all may last several days.

Finally, this method has been utilized to relieve pain occurring in tabes, sciatica, lumbago (see these Chapters). Sicard advised the use of the **epidural** method for injection of analgesic drugs. This method is easier and is not followed by the unpleasant symptoms of the first (see for details page 443).

CHAPTER XXIV

DISEASES OF THE PERIPHERAL NERVOUS SYSTEM

NEURITIS (INFLAMMATION OF NERVES)

Pathology.—An inflammation of a peripheral nerve may be confined to the connective tissue surrounding the nerve (**perineuritis**) or lying between the individual bundles of the nerves (**interstitial neuritis**) or else to the nerve fiber itself (**parenchymatous neuritis**). In the majority of cases these forms are combined.

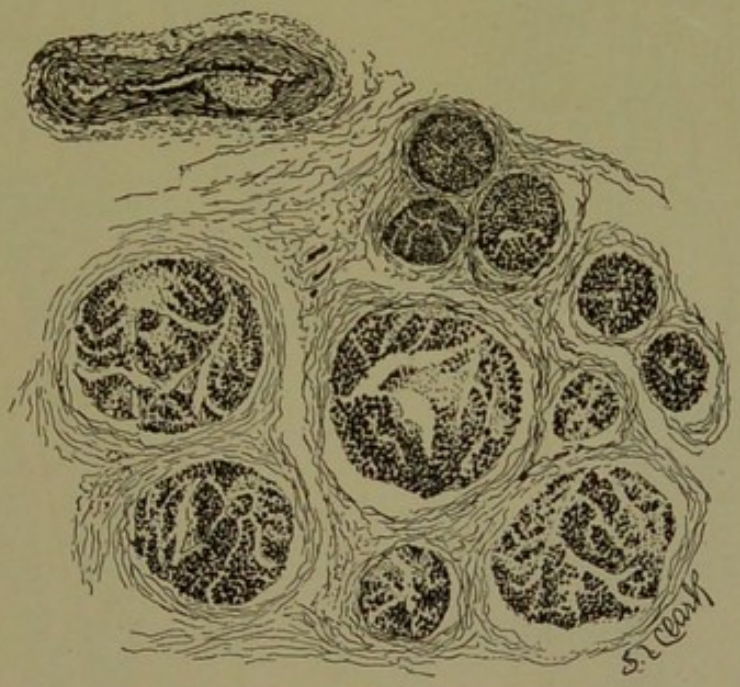


FIG. 119.—NEURITIS OF SCIATIC NERVE. (*Original.*)

Examination shows a tumefaction of the tissue surrounding the nerve. Dilatation of the blood vessels, extravasation of blood and sero-fibrinous exsudation infiltrating the sheath, leucocytes around the vessels are the first changes. Gradually these alterations extend to the interstitial tissue which proliferates. The exsudation within the sheath produces pressure on the nerve-fiber itself. In the latter the myelin becomes broken up into fine particles. When the disease advances, the axis-cylinder itself takes part in the morbid process. It undergoes degeneration and in more pronounced cases it atrophies and disappears. In cases of primary parenchymatous neuritis, viz. without the involvement

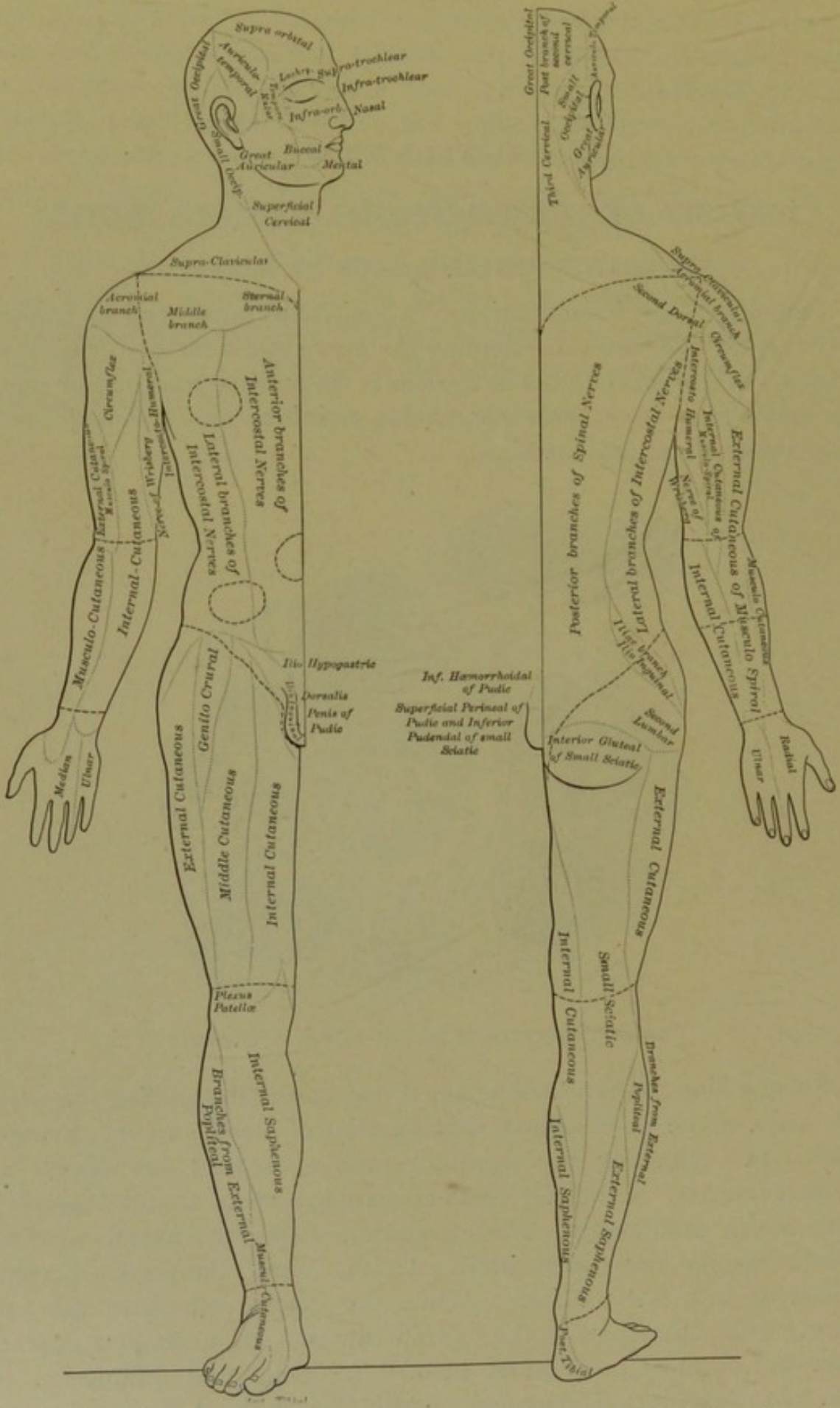


FIG. 120.—DISTRIBUTION OF SENSORY NERVES IN THE SKIN. (*Flower.*)

of the interstitial tissue, and which occurs as a result of a toxic influence, there is an exsudation with leucocytes around the blood vessels, segmentation of the myelin and breaking up of the axis-cylinder, swelling of the media of the blood vessels and proliferation in the adventitia.

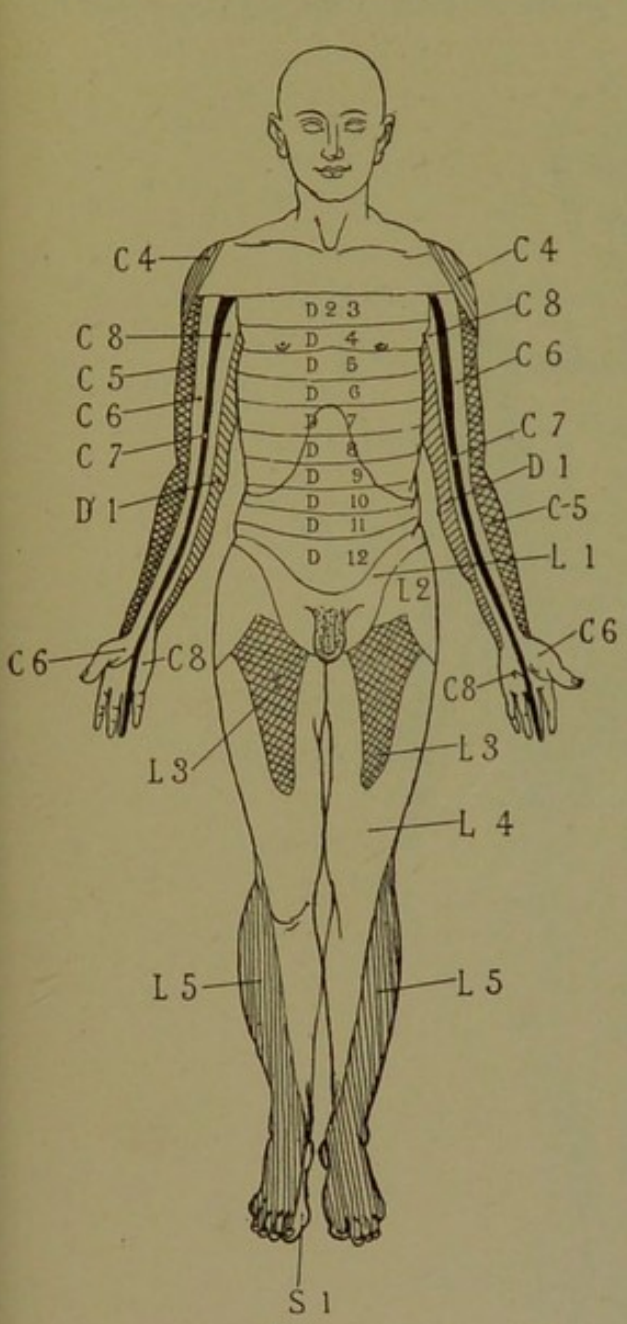


Fig. 121.

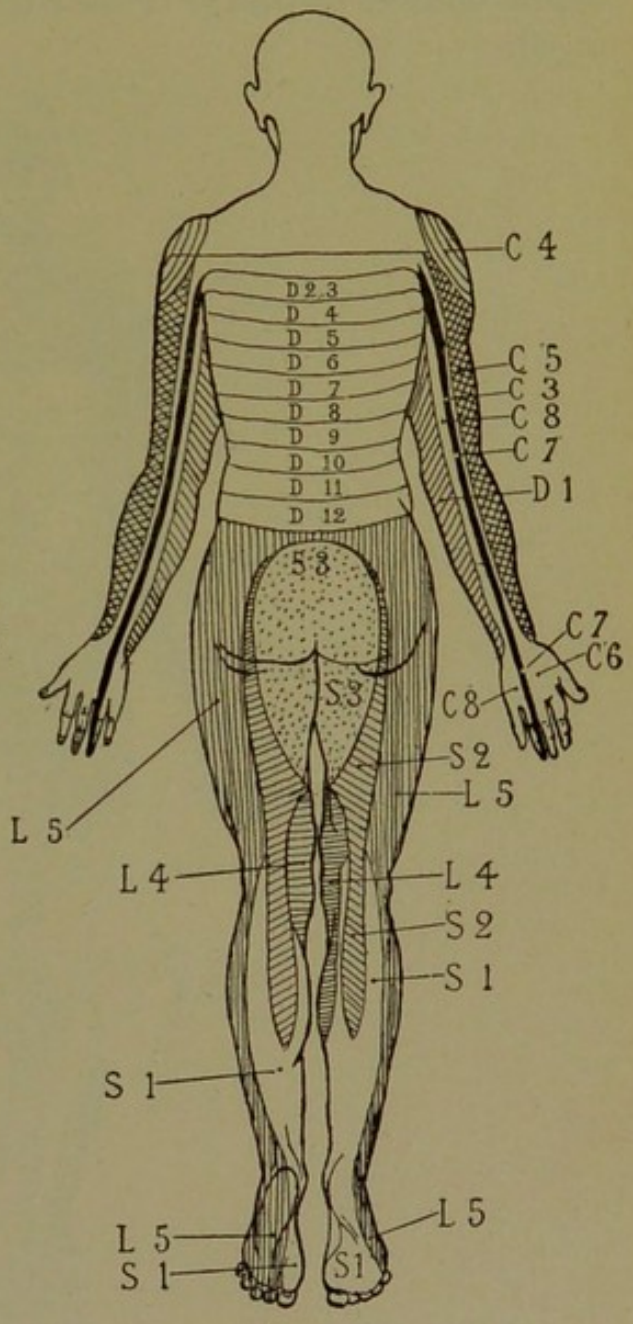


Fig. 122.

Sensory Radicular Distribution. (*Bouchard and Brissaud.*)
Letters *C*, *D*, *L* and *S* indicate respectively cervical, dorsal, lumbar and sacral roots.

In the form described by Gombault under the name of **periaxile segmentary neuritis** only some segments of the nerve-fibers are affected and the lesion consists of fragmentation of the myelin sheath, the axis-cylnders remaining intact. It is observed in **toxic** or **infectious,** also **traumatic** neuritis.

Recovery in neuritis corresponds to a process of **regeneration.** The latter consists of longitudinal division and sub-division of the portions of the axis-cylinder which remained intact and of gradual growth of the new filaments until they reach the tissues innervated by the nerve before its destruction; at this stage of their development they become covered by myelin.

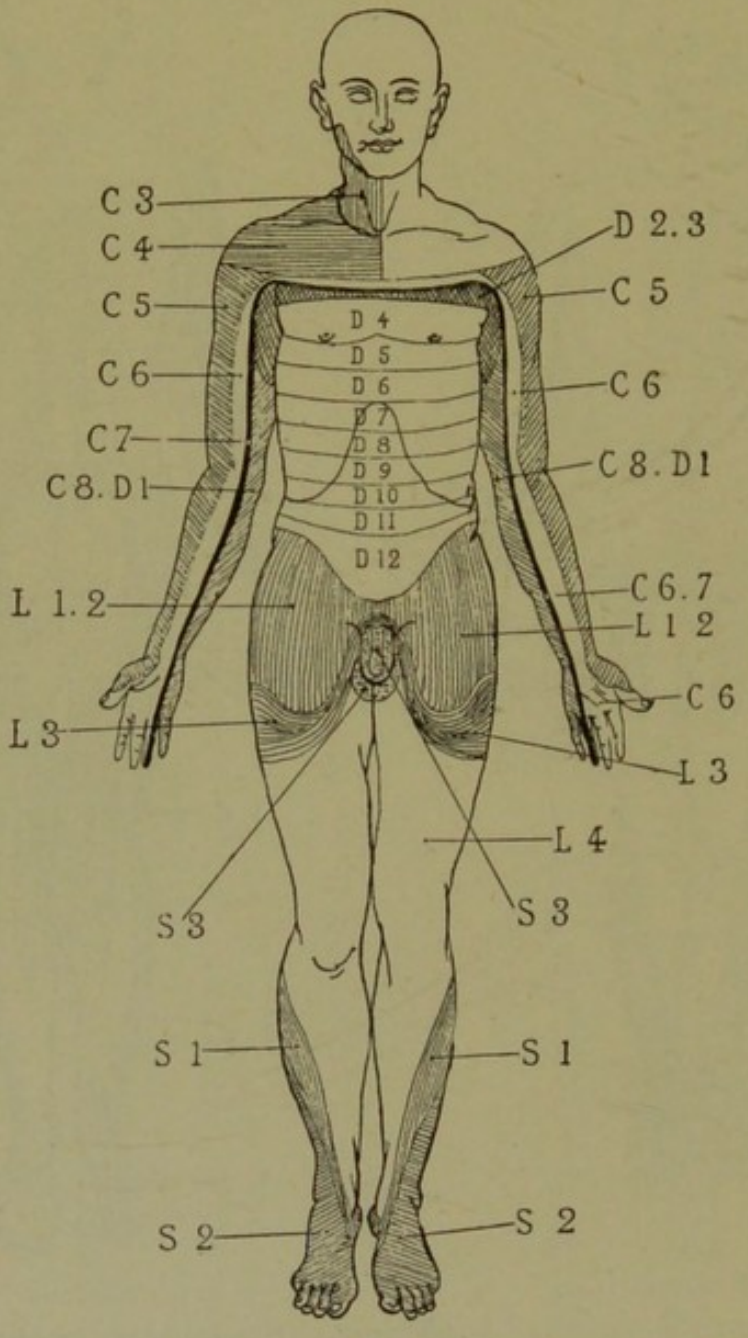


FIG. 123.

Letters *C*, *D*, *L* and *S* indicate respectively cervical, dorsal, lumbar and sacral roots.

Etiology.—Infections, intoxications and **local** factors are the chief causes of peripheral neuritis. Microbes or their toxins in infectious diseases frequently produce a multiple neuritis, but they may also be the cause of a localized neuritis. An infected wound is likely to set up an

inflammation of a nerve. Trauma sets up an inflammation, which may involve also a nerve. An inflammation of a finger may extend to a nerve or nerves of the arm (**ascending neuritis**). In such cases the neuritis is an upward extension of an inflammatory or septic process along a nerve or nerves. Suppurating joint or acute synovitis, diseases of bones (fracture,

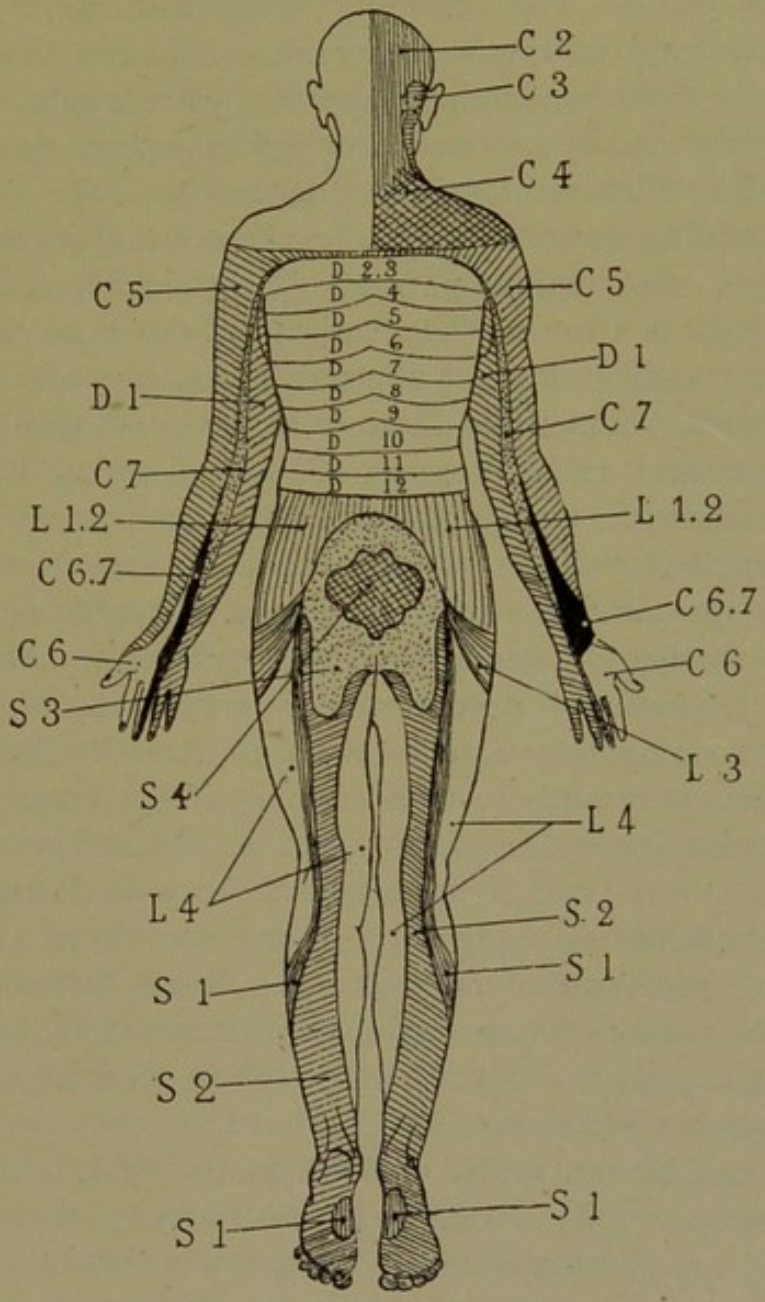


FIG. 124.

Letters *C*, *D*, *L* and *S* indicate respectively cervical, dorsal, lumbar and sacral roots.

tumor, caries), dislocations may produce a neuritis in the immediate neighborhood. Diphtheria, syphilis, cancer, tuberculosis may set up a toxic neuritis. Neuritis due, so to speak, to cold is often of toxic or infectious nature. Intoxications with alcohol, metals, carbonic acid, may produce a localized neuritis. In the course of diabetes, gout, chronic rheu-

matism, not infrequently a neuritis (sciatica for example) is observed; autointoxication is probably the immediate cause. Finally neuritis may be the result of localized ischemia or anemia caused by partial or complete blocking of an artery such as embolus.

Symptoms.—In diseases of peripheral nerves sensory disturbances are very conspicuous. The adjoining plates will render considerable assistance in the study of various sensory areas (Figs. 120, 121, 122, 123 and 124). The main sign of an inflammation of a nerve is **pain.** It is usually intense and aggravated by movement, or anything producing a congestion of the limb. The slightest touch or pressure upon the nerve produces pain. In **ascending neuritis** there is an excruciating burning pain along the nerve-trunk, also various paræsthesias, as formication, numbness, etc. The objective sensory disturbances in the area of distribution of the nerve consists at first of **hyperæsthesia,** but later of **hypæsthesia** or **anæsthesia.** Sometimes there is a perverted sensibility, such as cold taken for heat and *vice versa*. Motor disturbances usually accompany the sensory. Twitching and tremor are present at the beginning, but when the condition becomes chronic, paralysis of the muscles innervated by the affected nerve may ensue. Trophic disturbances of the skin are frequent (œdema, cyanosis, erythema). Atrophy of the muscles with reactions of degeneration is very frequent. Sometimes the muscular atrophy may assume the type Aran-Duchenne (see this chapter). In a case recently reported by Long (*Nouv. Iconogr. de Salpêtr.*, 1912) the atrophy first appeared in the left hand, three years later in the right and after twelve years it was a typical Aran-Duchenne form. Microscopically the following changes were found: atrophy of a large number of nerve-fibers, proliferation of connective tissue between the fibers, hypertrophy of vasa nervosum; atrophy of muscular fibers; the spinal cord was intact except a few isolated cells in anterior cornua of the cervical enlargement which were found altered.

A neuritis may be only **motor** or **sensory,** and then the symptoms are either only motor or only sensory. In the majority of cases it is of a mixed type. In lead intoxication the motor phenomena of neuritis are almost exclusively present. In syphilis sensory symptoms alone may be present for a long time. An insolated neuritis of a sensory filament of a nerve-trunk was reported by me in *J. of Am. Med. Ass'n*, 1909. It was a record of four patients in whom the lower cutaneous branch of the musculo-spinal nerve was involved. The symptoms were exclusively sensory (subjective and objective); motor and trophic disturbances were absent.

Course, Termination, Prognosis.—Neuritis usually lasts a long time—

from a few weeks to a few months; it may also last years. Its prognosis is, as a rule, good. It depends upon the cause. Recoveries are frequent. Rapid improvement predicts recovery. Neuritis of rheumatism and gout usually lasts a very long time. As a rule neuritis of long duration presents a bad prognosis as to complete recovery. The best outlook is in neuritis of traumatic origin.

Diagnosis.—Marked changes in objective sensations, especially anæsthesia over the area of distribution of the nerve, paralysis with atrophy and reaction of degeneration, are the chief and certain symptoms of neuritis.

Treatment.—**Rest** of the affected limb is the first indication. Ideal rest is obtained when the limb is immobilized by appropriate means. This procedure gives considerable relief from pain. In addition to immobilization local applications of extreme cold or extreme heat are advisable. A very good procedure is to place the affected part in very hot water for about half an hour or even an hour, two or three times a day. In two cases the only relief could be obtained from a continuous immersion of the arm in hot water. Counter-irritation by fly-blister and sedative applications may be useful in relieving pain. In **ascending neuritis** the pain may be so intense that relief can be obtained only by opening the wound and removing the cause of irritation; if the latter does not help, nerve section or even amputation are the only means. Internally coal-tar products, sedatives should be given. Morphine should be resorted to as a last resort. Massage can be used only when the acute symptoms, and especially pain, have subsided. Massage and electricity are advisable for combating the atrophy.

When this treatment has failed and the pain is intense, surgical intervention, as nerve-stretching or nerve-section, is indicated.

Neuritis of Ischemic Origin.—A total or incomplete obliteration of a blood vessel may produce irritation and cause a degeneration of a nerve and subsequently a paralysis. In such cases there is an insufficient blood supply to the nerve and muscles either by compression (ligature, surgical apparatus, tumors, enlarged glands) or by embolus, thrombus, obliterative arteritis. Cases of this kind have been reported by Broca, Charcot, Delherme, Babinski, Liston, Mally, Volkmann and others. The symptoms are: paralysis and atrophy with the reactions of degeneration. Pathologically there will be degeneration of the nerve, if the ischemia is complete and durable. In incomplete interruption of circulation the paresis of the limb disappears as soon as the obstacle is removed.

Hypertrophic Interstitial Neuritis of Dejerine.—See chapter on Muscular Atrophies.

NEUROMA

Neuroma tumors may be isolated and single or multiple. They usually grow in the sheath of a nerve-trunk. They may be soft (lipomata, myxomata) or hard (fibro-neuromata). The former are benign, the latter malignant. The **first** are usually single, they cause pain along the nerve, they are tender to touch; they can easily be removed.

The **malignant** form (fibro-sarcoma) has a special predilection for the sciatic nerve. It has a progressive course and destroys the nerve, producing besides pain also paralysis and atrophy of muscles. Operation is the only remedy, but the prognosis is not certain.

Multiple neuromata are fibrous tumors situated either in the skin or on the nerve-trunks. They are usually **diffuse**. They are known under the name of **Recklinghausen's** disease. When they are located on the nerves they occupy the endo-neurium; degeneration of the nerve-fibers follows. Pain is present and paralysis gradually develops. Not infrequently the cranial nerves are the seat of these tumors. When the spinal roots are affected, the tumors may be found also in the cord. The subcutaneous tumors are usually soft and consist of plexiform bunches of nerve-fibers described by Verneuil. Other symptoms: (1) pigmentation of the skin in brownish patches usually situated on the trunk and upper portions of the limbs; (2) mental manifestations, such as depression and diminution of intelligence. The **prognosis** is uncertain (Fig. 117).

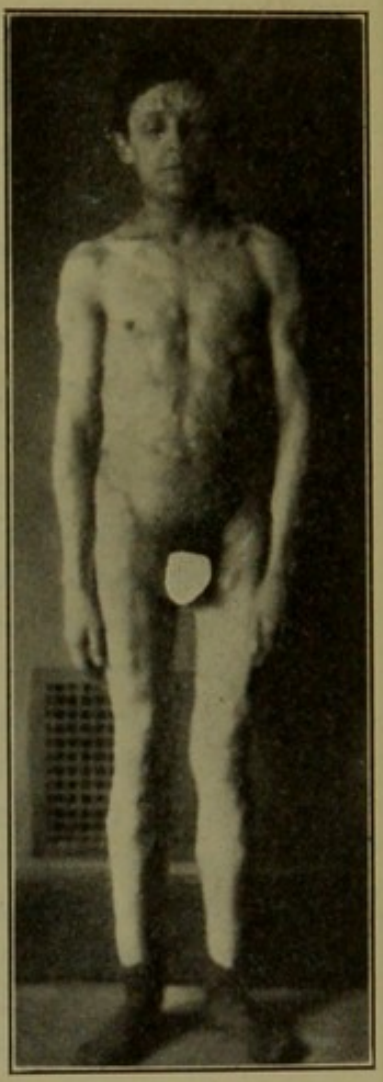


FIG 125.—NEUROFIBROMATOSIS (*v. Recklinghausen's disease.*)

Neuroma following amputation consists of a bulbous mass developing on the proximal end surface of a divided nerve. It is due to a rapid growth of the sectional axis-cylinders and proliferation of the surrounding connective tissue. It is usually very painful to pressure and thus it prevents from using an artificial limb. Twitching of the muscles is also observed and such a neuroma may be the point of departure for epileptic seizures. Removal of such tumors is a necessity but recurrences are frequent.

MULTIPLE NEURITIS

Pathology.—The lesion described in the preceding chapter is the same when several nerves are simultaneously affected. The special feature of polyneuritis lies in the fact that the atrophic state of the nerves is more marked than the inflammatory, and that perineuritic and interstitial changes are extremely slight, while the nerve-fibers are markedly altered. Another peculiarity is found in changes in the central nervous system. Thus poliomyelitic foci in the cells of the anterior cornua, degenerative condition of the posterior columns (see my case in *Amer. Medic.*, 1905), inflammation of the bulbar nuclei, have been observed

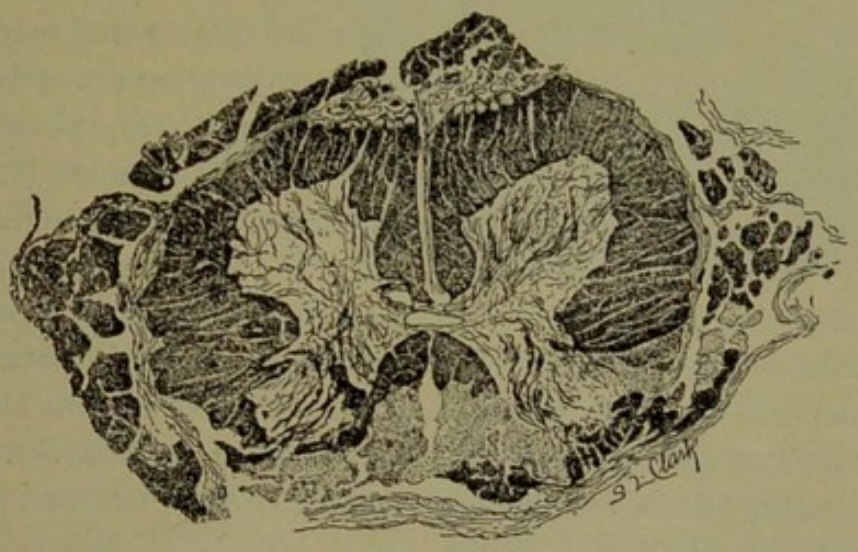


FIG. 126.—LEAD MULTIPLE NEURITIS, SHOWING INVOLVEMENT OF THE POSTERIOR COLUMNS OF THE CORD. (*Original.*)

sometimes. Evidently the chemical poison which is the chief cause of multiple neuritis affects almost all parts of the nervous system but to a different extent in various cases: sometimes the peripheral nerves, motor or sensory or both, at another time the nerves and the cord.

Etiology.—**Intoxications** are the most frequent causes. Alcohol occupies the first place. Lead, mercury, arsenic, phosphorus, carbonic acid gas, intoxications of alimentary origin (ptomain) are also frequent causes. **Infectious diseases** play a great rôle. Typhoid fever, diphtheria, small-pox, erysipelas, pneumonia, scarlet fever, grippe, dysentery and tuberculosis are not infrequently accompanied or followed by multiple neuritis. In these cases the toxins are the immediate cause. In leprosy and beriberi the neuritis is due to a direct action of microörganism.

Syphilis, malaria, diabetes, puerperal state, tobacco and arteriosclerosis are less frequent causes.

The interstitial hypertrophic neuritis of Dejerine and Sottas is described in a separate chapter.

Symptoms.—Irrespective of their causes all forms of polyneuritis present a common clinical picture, which will be described here; the special symptoms characteristic of each variety will be mentioned later.

General Symptoms.—In the **subacute form** there is usually noticed a rapidly progressing loss of power in the lower extremities. The **paralysis** is more marked in the lower part of the limb than in the upper. When the disease advances, the upper extremities become affected. As the **extensors** are particularly involved, various abnormal attitudes of the limbs are acquired. **Wrist-drop** and **foot-drop** are characteristic of polyneuritis. The gait is typical: being unable to flex the foot, the patient is obliged at each step to flex the thigh on the pelvis in an exaggerated manner. This raising of the thigh with the toes of the foot hanging down is analogous to the gait of horses. It is called **"steppage gait."** The paralysis is flaccid. While it affects most frequently the extensor muscles, it may nevertheless attack any muscle or group of muscles. The tendon reflexes are abolished or markedly diminished.

Gradually the muscles of the thorax and abdomen become affected and when the bulbar nerves are invaded the condition is alarming.

The electrical reactions of the paralyzed muscles present the following changes. At first there is a diminution of response to faradic and galvanic currents. Later on the faradic contractility is abolished and reactions of degeneration are manifest.

Muscular atrophy follows the paralysis, and its beginning corresponds to a destruction of the axis-cylinders of the nerves distributed in the muscles. It is very frequent in polyneuritis, and it may make its appearance at any stage of development of the paralytic symptoms.

Sensory disturbances are very common. They consist of all sorts of abnormal sensations, as tingling, burning, numbness, etc.; of spontaneous pain or of pain produced by the slightest pressure or movements of the limb; of objective modifications of all forms of sensations (hypæsthesia, anæsthenia, hypalgesia, analgesia); of loss of muscular sense (patient is unable to appreciate the position of the affected limbs).

Vaso-motor disturbances are occasionally observed, especially in polyneuritis of external origin (traumatism, compression, tumors). They are ulcers, eruptions, œdema, hyperhidrosis of hands and feet.

The **sphincters** are usually intact.

In the course of multiple neuritis paralysis of **cranial nerves** may occur. The oculo-motor, the abducens, the optic, the facial, the pneumogastric are sometimes involved. Strabismus, diplopia loss of light reflex, optic

neuritis, amaurosis, facial palsy, paralysis of the vocal cords, tachycardia, difficulty of respiration—are all observed occasionally.

The **acute form** of multiple neuritis differs somewhat from the preceding form. The onset is here sudden, accompanied by chills, fever and headache. Rapidly the symptoms develop. Paralysis, loss of reflexes, objective sensory disturbances, etc., appear in the same succession as in the previous form.

FIG. 127.
DOUBLE WRIST-DROP IN A CASE OF LEAD PALSY.

Course, Termination, Prognosis.—In the **sub-acute** form recovery follows the majority of cases. The paralysis, the atrophy, the sensory disturbances gradually improve, the electrical reactions become normal. In some cases the paralysis persists, retraction of tendons and immobilization of joints take place and the patient is permanently crippled. In exceptional cases the respiratory muscles are paralyzed and death may ensue from pulmonary complications. The **acute form** (not to be confounded with Landry's type of polyneuritis) bears practically the same prognosis as the subacute.

Special Features

A. Alcoholic Polyneuritis.—This is the most frequent of all forms of multiple neuritis and more in women than in men. The disturbances are both **motor** and **sensory.** The **latter** are very conspicuous; they may be pronounced while the motor power is only slightly disturbed. The objective and especially the subjective sensory disorders described in the preceding chapter are particularly well developed. The patient complains early of unusually severe pain, which may be lancinating, tearing or pulling. The least pressure shows exquisite tenderness. The pain leads to insomnia, to mental depression.

Cramps in the calves of the legs, muscular twitchings occur frequently. Intestinal or gastric pain occurring in paroxysms is common. **Muscular hyperæsthesia** is particularly marked; the least pressure, even the **contact** of bed-clothes is intolerable. **Cutaneous anæsthesia** in the limbs is usually only in their distal portions.

The **paralysis,** gait, reflexes are as described in the general symptomatology. However in alcoholic neuritis the bilateral paralysis of the extremities has a tendency to **generalization;** it gains the proximal ends of the limbs, the thorax, the abdomen. The muscular **atrophy** is pronounced.

The paralysis and atrophy produce deformities of the limbs. When the muscles of the back are involved, there is lordosis. When the glutei are involved, the patient is unable to rise from his seat. In paralysis of the abdominal muscles the expelling power of the bladder and rectum is weakened. If the muscles of the neck are affected, the head cannot be moved voluntarily. Involvement of the intercostal muscles interferes with respiration. Very rarely the muscles of the face are involved. Tremor of the tongue and weakness of the muscles participating in articulation of words may give the impression of paresis.

Among the **vaso-motor** disturbances œdema of the lower limbs is very common. Profuse sweating is frequent.

Ocular disturbances are frequent. Retro-bulbar optic neuritis, central scotomata, strabismus, ptosis and sluggish pupillary reflex to light—are all met with in alcoholic polyneuritis.

Finally **psychic** disturbances are sometimes observed. Besides various manifestations which are usually encountered in alcoholism, such as delirium, confusion, stupor and hallucinations, there is a special symptom-group which sometimes accompanies polyneuritis. This is the so-called **Korsakoff's psychosis.** It consists chiefly of confusion with illusions of identity, loss of orientation, of memory. Delirium and hallucinations may also be present.

The amnesia covers the period for recent events and for the time of the illness. The various lapses of memory are filled by the patient with a great variety of **fabrications** or **confabulations.** The patient described events and occurrences which never took place and does it with minutest details.

The amnesia for recent events is considerably facilitated by an inability to associate or synthetize new facts. The disturbance in association of ideas is limited to new perceptions and conceptions. The voluntary psychic activity is diminished, but the patient preserves the old intellectual acquisitions and his judgment on those old events is intact.

The affection lasts from a few weeks to several years. It terminates either in death, or in a more or less complete recovery, or else in dementia.

The **course** of alcoholic polyneuritis is usually subacute, lasts several months or even longer and in a large number of cases results in recovery. In some cases the recovery is incomplete: deformities of the extremities

remain permanent. Pulmonary tuberculosis is not infrequently the cause of death in the protracted cases. The **prognosis** is serious, as bulbar symptoms may develop at any time and hasten death and on the other hand deformities render the patient powerless. The acute cases have ordinarily an unfavorable prognosis. In a case that I had under my observation for eight months there were besides the involvement of the limbs also bulbar symptoms (difficulty of deglutition and of respiration, tachycardia) and mental disturbances; the patient nevertheless made a perfect recovery. Recurrences of attacks is another special feature of the alcoholic multiple neuritis.

B. Lead Neuritis (Lead Palsy).—Pathologically it presents the periaxile segmentary form of Gombault (see Pathology). The **paralysis** presents a strictly localized character. The upper extremities are the seat of predilection for lead palsy. The latter is bilateral and symmetrical. It may present the **antribrachial** type, **Aran-Duchenne's type,** also the **scapulo-humeral type** (see Muscular atrophies, also my contribution in *New York Med. Jour.*, 1906). The first is the most common. Gradually and insidiously the extensors of the fingers, **except the long supinator and the long abductor of the thumb,** are paralyzed. This leads to a special attitude of the hand. The latter is in pronation and flexion (wrist-drop), also the fingers are slightly flexed.

Muscular atrophy with **reactions** of degeneration, also loss of reflexes develop quite early. Cyanosis of the hands is frequent.

Sensory disturbances are usually absent. In some cases the onset of paralysis is preceded by some pain or paræsthesia.

Optic neuritis, amblyopia, contraction of the visual field are sometimes observed.

In rare cases lead palsy may become generalized. It may invade the lower extremities and even the trunk.

When the lower extremities are affected, the peroneal muscles are paralyzed, but the **tibialis anticus** is usually spared.

In acute cases of lead intoxication the paralysis may become generalized. Paralysis of the diaphragm and intercostal muscles causes dyspnœa and the condition becomes alarming. Ordinarily the disease has a chronic **course.** In a large majority of cases the symptoms improve and even disappear completely. Recurrences are not infrequent. The **prognosis** of lead neuritis is as a rule favorable, but the damage done by lead on other organs and tissues may be so great that life is in danger, as for example, in cases with renal complications. In making the **diagnosis,** some aid may be obtained from other symptoms of lead poisoning, such as the blue line on the gums, history of colic, encephalopathy, tremor. The distinction

between wrist-drop from lead neuritis and that from musculo-spiral palsy lies in the retention of power of the long supinator muscle in the first affection.

Schoenfeld (Mediz. Klinik, May, 1913) has recently called attention to the importance of examination of blood in lead poisoning. He points out as a characteristic finding the basophil granulation in the red corpuscles and the change in tint of these basophil substances under the action of certain stains. These blood findings are also useful to control the progress toward recovery; in 18 of 40 patients thus kept under observation the blood returned to normal after the patients had changed their occupation. Negative findings exclude lead intoxication.

C. Arsenical Neuritis (Arsenical Paralysis).—This form is a mixed type of neuritis: **motor and sensory.** It affects the four extremities, the lower before the upper, and while the extensors are particularly involved, the paralysis **affects also the flexor muscles** (unlike alcoholic and lead palsies). Ataxia is frequently observed, and this in addition to the loss of reflexes, gives the impression of tabes (arsenical **pseudotabes** of Dana). Psychic disturbances are sometimes observed. Muscular atrophy with reactions of degeneration, sensory and trophic disturbances are identical with those of other forms of polyneuritis. Subjective sensory disturbances are usually conspicuous and persistent. They are: severe tearing pain in the limbs, burning in the toes and soles of the feet. The skin and muscles are very tender to pressure. This hyperalgesia is frequently associated with anæsthesia to touch, pain and temperature. The atrophic paralysis may spread and involve not only the proximal ends of the limbs, but also the diaphragm (the intercostal muscles usually escape). Tremors and spasmodic contractions have been observed. Loss of vibratory sense or osseous sensibility in the lower extremities was observed by Byrnes (*J. Amer. Med. Ass'n.*, 1909). Among other symptoms of arsenical neuritis may be mentioned: pigmentation of the skin, vesicular eruption, erythematous and œdematous condition of the soles and of the palms. Epileptiform convulsions have occasionally been observed. The prognosis in arsenical neuritis is usually favorable. In 1900 an epidemic of arsenical neuritis occurred in Manchester in beer drinkers. The analysis of the beer discovered the presence of arsenic. The arsenic was derived from sulphuric acid employed in preparation of glucose used in brewing cheap beer.

Arsenical neuritis may occur from medicinal use of arsenic. Thus it was observed in patients suffering from chorea who used arsenic.

D. Diphtheritic Neuritis (Diphtheritic Paralysis).—It usually occurs during convalescence from diphtheria. It affects first the **palate** and develops slowly. At the beginning there is only some disturbance in

phonation and deglutition, but gradually the difficulty becomes greater. The palatine velum hangs down and does not contract during phonation. The mucous membrane is anæsthetic. The **food passes through the nose.** As the paralysis extends to the pharynx and larynx, the epiglottis does not close the larynx; food penetrates into the respiratory passages and produces pneumonia or else direct asphyxiation. This is also aided by **anæsthesia of the larynx.** The **phonation** is nasal, labial letters are pronounced indistinctly. When the paralysis reaches the recurrent laryngeal nerve, **hoarseness** and **aphonia** will be the result. The **eye muscles** are infrequently involved. Strabismus with diplopia, ptosis, disturbed pupillary reflexes (paralysis of accommodation with preservation of light reflex), amblyopia are all observed.

When the paralysis shows a tendency to generalization, the lower extremities become affected after the palate, pharynx, larynx and eyes. Like in the preceding forms the antero-external group of the leg muscles are paralyzed. **Steppage** gait with **foot-drop** is present. Ataxia, loss of muscular sense, loss of reflexes, intense pain gives the impression of tabes (pseudo-tabes). The muscles of the upper extremities, also of the trunk, may follow those of the lower. When atrophy develops, it is usually rapid and reactions of degeneration are present.

The muscles of the tongue, lips and face may be affected. When the pneumogastric and the phrenic nerves are involved, cardio-pulmonary disturbances are present.

The **course** of the disease depends upon its intensity and localization of the poison. When bulbar symptoms are present, death usually ensues. Dyspnœa, syncope, asphyxia, aspiration pneumonia are all the causes of death. Generally speaking the **prognosis** is favorable, especially when the paralysis is confined to the palate. In the latter case the duration is about two to three weeks. When the paralysis is generalized, it lasts several months. The special feature of diphtheritic palsies lies in **rapid development** and the **instability** of the motor symptoms, which disappear in one place to appear in another and then again reappear in the first.

E. Carbonic Gas Neuritis.—In cases of intoxication with carbon dioxide the following special symptoms are present: **anæsthesia** of the extremities; **paralysis** at first of antero-lateral muscles of the legs and later of the muscles of the upper extremities; **preservation** or **increase** of tendon reflexes; **trophic** and **vaso-motor** disturbances; **mental disturbances** (particularly amnesia). Recovery follows almost in every case. Mental feebleness and anæsthesia remain a long time, even in the most favorable cases.

F. Mercurial Neuritis is manifested by paresis or paralysis of the antero-external groups of muscles of the legs and sometimes also of the upper extremities, by anæsthesia in certain areas and hyperæsthesia in others, finally by enfeeblement of the mental faculties.

G. Puerperal Neuritis may occur either during pregnancy or after labor. In the first case usually the nerves of the lower limbs are involved, in the second case the nerves of the upper limbs are affected. Here the neuritis presents certain peculiarities in its localization. It affects especially the terminal branches of the median and ulnar nerves, and mostly on the right side. Sometimes it becomes generalized and then involves the lower limbs. Sensory disturbances are slight. The disease is probably infectious in nature in cases which occur after labor. In neuritis occurring during pregnancy the etiological factor is probably disturbed metabolism. It usually ends in recovery.

H. Beriberi or Kakke.—Under this name is known a form of endemic or epidemic multiple neuritis, considered **infectious** in origin, and met with in South America, Japan, Philippines, China. Its microörganism has not yet been discovered. It is supposed to enter the body through the gastro-intestinal tract. The disease has been observed in connection with prolonged consumption of mouldy rice.

A very recent experimental study by Strong and Crowell (*Philippine Jour. of Science*, 1912) shows that there is no evidence which could suggest that beriberi is an infectious disease, that beriberi in the Philippine Islands is due to a prolonged consumption of a diet which lacks certain substances necessary for the normal physiologic needs of the human body. For prevention and cure of beriberi it is necessary that man shall be supplied with a liberal nutritious diet suitable to the physiologic needs of the body. As to the question of rice, although a rice containing 0.4 per cent. of phosphorus will prevent the appearance of polyneuritis in fowls, nevertheless from these two authors' experiments it is evident that beriberi in man may be produced by rice containing 0.37 per cent. of phosphorus pentoxide. The higher the phosphorus content of rice the less is the liability of that rice to produce beriberi.

The following are the chief symptoms of the disease. As an early manifestation must be mentioned a **cardiac** syndrome consisting of smallness and rapidity of the pulse, dilatation of the heart, systolic murmur. Attacks of cardiac insufficiency are not rare **Œdema** is another frequent early symptom. It affects the lower extremities, abdomen, neck and face, also the viscera; pericarditis is not rare; increase of cerebro-spinal fluid in the spinal canal has also been observed. Albuminuria is usually absent. Absence of fever is the rule. The **nervous symptoms** which usually

develop after an acute onset consisting of nausea, vomiting, oppression, are: (1) paralysis of the lower extremities and especially of the antero-external group of muscles; the gait, reflexes, attitude are same as in other forms of polyneuritis. The upper extremities, thorax, may also become involved, (2) anæsthesia of the affected limbs and spontaneous pain, (3) muscular atrophy, (4) mental disturbances similar to those of alcoholic polyneuritis, (5) respiratory disturbances. The latter symptom, also the above-mentioned cardiac condition, are due to the involvement of the vagus and phrenic nerves. **Rapid development** of cardiac and pulmonary disturbances is characteristic.

Beriberi has been observed under three varieties: (1) the humid or hydropic form in which the œdema is most conspicuous; (2) the dry or paralytic form in which there is no œdema but paralysis of the muscles is the most prominent symptom; (3) mixed form in which the symptomatology is complete. In all forms of the disease the cardiac symptoms are invariably present. The **duration** of the disease is from a few weeks to several months.

The **prognosis** is grave, although recovery is possible.

Death usually occurs from the cardiac failure or from some intercurrent disease. The **treatment** does not differ from that of other forms of multiple neuritis. Special attention, however, should be given to the condition of the heart. Lumbar puncture may be useful in view of the increased tension of the cerebro-spinal fluid.

I. Lepra Neuritis (Nerve Leprosy).—In this form of leprosy the changes are found almost exclusively in the peripheral nerve trunks and their cutaneous branches. Changes in the central nervous system, if present, are secondary to the general leprous infection. Thickening of the nerves (and sometimes nodules) is very frequent in the early period of the disease, but in the advanced stage atrophy is usual. The leprosy bacillus is found in the connective tissue between and around the nerve-fibers. This tissue proliferates and through pressure on the nerve-fiber itself produces its atrophy (Figs. 128 and 129).

Clinically trophic disturbances are observed. The joints of the phalanges are commonly affected. Ulcerations with subsequent necrosis are characteristic. The phalanges gradually fall off. The process may begin at the nail and then progressively involve the entire digit. The skin presents patches of discoloration of bronze color. Small areas of thickened skin on the ears and face have been observed (due to a local accumulation of bacilli).

In the majority of cases the hands present a claw-like appearance with marked atrophy of Aran-Duchenne's type (see this chapter). Reac-

tions of degeneration are present. The atrophy may not be confined to the extremities but also involve the muscles of the trunk and face. Objective sensory losses are usually found over the distal portions of the limbs. Syringomyelic sensory dissociation is common, but at the beginning there is a long period of hyperæsthesia with pain in the extremities. The relationship between syringomyelia and leprosy is according to competent authorities a very intimate one.

Prognosis is, generally speaking, unfavorable, although the disease may last several years. The disease may be also self-limited.

The **treatment** is mostly hygienic. Alteratives, also strychnia, may be tried.

J. Senile Neuritis has been described particularly by Oppenheim. He observed a slight paralysis of the distal ends of the limbs, which develop very gradually. Pain and hyperæsthesia may or may not be present. The degenerative state of the peripheral nerves is probably due to the deficient blood supply caused by atheromatous arteries characteristic of old age.

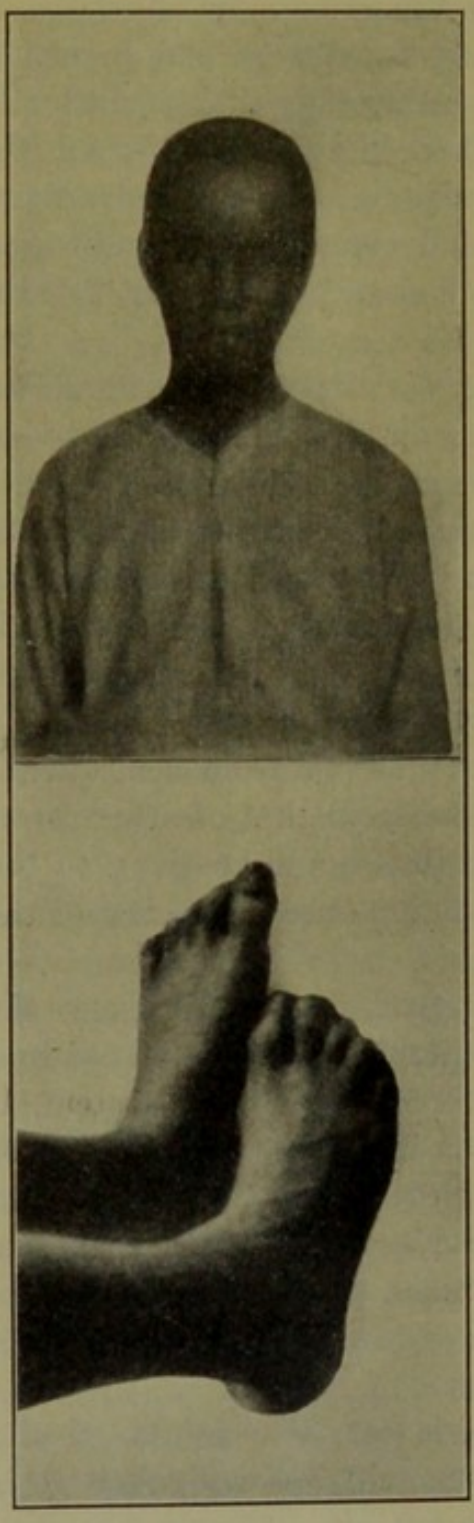


FIG. 128.—LEPROSY. Mutilated toes, ulcerations, self-amputations, constrictions around phalanges. Patches of discoloration of skin of the face and feet.

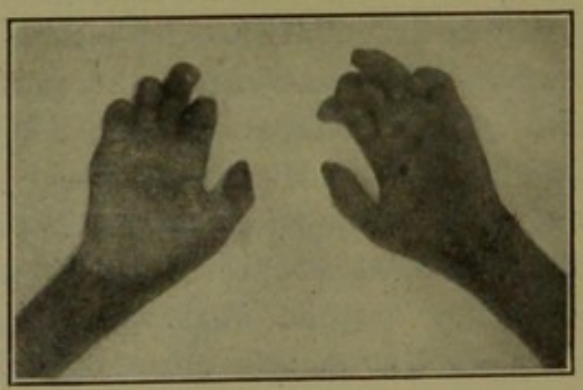


FIG. 129.—LEPROSY ON HANDS.

Diagnosis of Multiple Neuritis.—The above described general symptoms of polyneuritis and the special symptoms of each individual form

are sufficiently characteristic for establishing the diagnosis. There are, however, a few maladies which sometimes resemble closely polyneuritis. They are: **Acute myelitis, anterior poliomyelitis, tabes.**

When the paralysis due to **myelitis** is **spastic,** the disease cannot be confounded with polyneuritis, as the state of the tonicity of the muscles, the reflexes, the gait and the condition of the sphincters are radically different in the two affections.

The **flaccid** form of **myelitis** presents some difficulty of differentiation. Here the **anæsthesia** is much pronounced and affects almost symmetrically the entire lower extremities (in polyneuritis the objective sensory disturbances are slight); the involvement of the bladder and rectum, also sexual disturbances, are constant (in polyneuritis they are usually absent); bed-sores, also ulcerations of the heels are constant (absent in polyneuritis).

In **acute anterior poliomyelitis** the paralysis affects chiefly the roots of the limbs while the distal ends are not involved (in polyneuritis the condition is reversed); the cranial nerves are rarely affected (in polyneuritis frequently); fibrillary contractions are present (absent in polyneuritis); sensory disturbances are absent (present in polyneuritis); the distribution of paralysis, of atrophy and electrical contractility is symmetrical (irregular in polyneuritis).

In **tabes** the etiology is different from that of polyneuritis. In the former the presence of Argyll-Robertson's pupils, of disturbances of the sphincters, of optic atrophy, gastric crises, exaggerated superficial abdominal reflexes, thoracic bands of anæsthesia, finally the absence of tenderness of the nerve-trunks (so characteristic of polyneuritis) will decide the diagnosis. Lymphocytosis of the cerebro-spinal fluid is present in tabes, absent in polyneuritis.

Treatment.—The first indication is to remove the cause. In cases of intoxication with alcohol the patient should be isolated; in poisoning with lead or mercury the patient is advised to discontinue his occupation. In diphtheritic polyneuritis administration of antitoxin may be of benefit. In syphilitic cases mercury and iodides are indicated. Iodides are also beneficial in lead neuritis. In rheumatic cases salicylates, iodides, diuretics, diaphoretics are of use. When **pain** is present, it will be relieved by rest in bed, hot applications or hot baths, also by coal-tar products (aspirin, phenacetin, etc.), sedatives (bromides). Morphia should be avoided. When the acute stage has subsided, massage and electricity will be very useful for combating the oncoming atrophy and perhaps for relieving the pain. As soon as pain has subsided, the patient should be allowed to use his limbs and this is aided by daily

massage, electricity and systematic exercises especially in cases of incoördination. When permanent contractures with deformities are present, surgical intervention is necessary.

General hygiene, good nutritious food are essential, and in case of emaciation, iron and tonics should be added.

ACUTE ASCENDING PARALYSIS (LANDRY'S PARALYSIS)

Pathology.—The findings in Landry's paralysis are not constant. Frequently are seen changes in the spinal cord. In such cases there may be disseminated inflammatory foci (myelitis), or a certain degree of anterior poliomyelitis or else changes in the pyramidal tracts. The cells of the anterior cornua appear smaller, their contour is irregular. Their chromatolysis is marked. The most pronounced changes in the majority of cases are seen in the lumbar enlargement, which corresponds clinically to the earliest involvement of the lower limbs. The axis cylinders and their myelin sheaths are in a state of degeneration. The neuroglia cells are smaller and in a state of degeneration; sometimes hyperplasia is present. The blood vessels may be normal or more frequently present a leucocytic infiltration; in the latter case rupture may take place; punctiform hemorrhages are not rare. The roots, especially the anterior ones, and the peripheral nerves are involved. Disintegration of myeline and subsequently atrophy of the axis cylinders are characteristic. When the meninges are involved, they present all degrees of inflammation. The alterations just described are not all present in every case. In some cases there are only central changes. In some cases there are evidences of neuritis. In other cases there may be a combination of spinal and neuritic lesions with or without involvement of the medulla. The observed alterations are variable as to their localization and intensity. Acute ascending paralysis is an affection of the peripheral motor neurone involving separately or simultaneously every constituent portion of the neurone. Finally in a certain number of cases no lesion whatever has been discovered.

The cerebro-spinal fluid presents also a variability in findings. The leucocytic formula may be either normal or exaggerated. Lymphocytosis or polymorphonucleosis have been found. However it may be said with some degree of certainty that abundant leucocytosis is in favor of central origin (spinal cord) of the disease, while absence of leucocytosis is in favor of its neuritic origin.

Etiology.—All the evidences point to an **infectious** origin of the malady. It may develop in the course of various infectious diseases or independently. By means of injections of cultures in animals typical

Landry's paralysis had been reproduced experimentally (Thoinot and Masselin). The microörganism has not yet been discovered. A recent case reported by Bolten (*Berl. klin. Wchn.*, Jan., 1911) seems to confirm the contention that Landry's paralysis is the result of intoxication of the spinal cord, the toxin interfering with the function of the spinal and bulbar centers. The toxin seems to affect the motor neurone alone. On this basis 600 c.c. of cerebro-spinal fluid were withdrawn and substituted by 540 c.c. of non-toxic fluid, viz. salt solution. The patient recovered.

Among other etiological factors may be mentioned: cold or extreme heat. Men are more affected than women, adults more frequently than children or aged individuals.

Symptoms.—The clinical picture may be either that of poliomyelitis or polyneuritis. There is almost always a prodromal stage lasting from hours to weeks. General malaise, pain in the back and various paræsthesias (tingling, numbness) in the limbs usually precede. The **onset** is always rapid or even sudden. The disease in **typical** cases begins with **paralysis** of the lower extremities. Rapidly the upper extremities and the trunk become invaded. They are followed by the muscles of the head, neck, the tongue, palate, larynx and pharynx. Difficulty of deglutition and of respiration, slowness or else acceleration of the pulse appear. The patient dies from asphyxia. Ocular palsies, as well as facial palsy, have been observed in some cases.

The **paralysis is flaccid.** The **reflexes,** deep and superficial, are **abolished.** Sensations are very slightly or not at all disturbed. The sphincters are usually intact. The muscles are usually normal and no reactions of degeneration are present. Fever is, as a rule, absent.

In **atypical** cases the onset of paralysis may be preceded by chills, headache, malaise. The paralysis of the lower extremities is gradually followed by involvement of the sphincters and of the upper extremities and sometimes of the medulla. Among the atypical cases can also be mentioned those in which the paralysis begins with the upper extremities.

In another series of cases there may be also the typical picture of multiple neuritis with pain and tenderness of the nerve-trunks, etc.

Course, Termination, Prognosis.—In the majority of cases the termination is fatal. Recovery rarely occurs. In the latter case the symptoms gradually improve. The patient may recover, particularly when the bulbar symptoms disappear. In such cases muscular atrophy will be observed; it makes its appearance at the end of a few weeks. The atrophy is then diffuse. Reactions of degeneration are absent; there is only a diminution of response to electric stimulation. In fatal cases the duration is only of a few days (three to eight).

Diagnosis.—The rapidity of invasion, the mode of development of the paralysis, viz. the ascending course, are characteristic of the disease. From acute anterior poliomyelitis it will be differentiated by the rapid course and bulbar involvement. Besides, the prodromal symptoms such as malaise, gastro-intestinal disturbances, etc., are more pronounced in acute poliomyelitis. Asymmetry in the involvement of muscles on both sides of the body is characteristic of poliomyelitis. Rapid improvement in some muscles within a few days, early atrophy and RD are typical only of poliomyelitis.

Treatment.—At the appearance of the first symptoms the chief indication is elimination of the infectious element. This can be accomplished to some extent by proper elimination. Diuretics, purgatives and diaphoretics should be administered. Cauterization of the spinal column is advisable. Internally ergot, and in specific cases, mercury may be of some use. If the patient survives, and paralysis with atrophy becomes chronic, massage and electricity are useful.

PERIODIC PARALYSIS

Westphal in 1885 first called attention to an affection which is analogous to polyneuritis. It is characterized by attacks of paralysis in the intervals of which the patient is perfectly normal. The attacks may last from several hours to two days. The palsy affects all voluntary muscles except those of the face, tongue, pharynx, mouth, eyes, also those of the sphincters. The lower as well as the upper extremities may be equally involved, although the latter more frequently than the first. The paralysis develops rapidly and it is **flaccid.** It is usually preceded by a tingling sensation and even some pain. As a rule the lower extremities are first attacked, then follow the trunk and arms. Sometimes some muscles of the neck are affected: the patient can move his head when it lies on a pillow, but he cannot raise it. The muscles innervated by the cranial nerves are intact. The sphincters are intact and if constipation is present, it is due to a paralysis of the abdominal muscles. The return of power is done in a manner reverse to the development of paralysis. Thus the arm first and the legs last regain their motility. The reflexes are abolished or markedly diminished. The electrical contractility of muscles and nerves is markedly diminished or totally abolished, although the muscles may contract when the nerves are being stimulated. Sensations and pupillary reflexes are normal. A very frequent symptom is **sweating** during the attacks which may last between fifteen minutes and two days.

The attacks may be severe or complete and mild or incomplete. In the latter case there is only paresis of the muscles.

As to less frequent symptoms, there may be disturbance of circulation: dilatation of the heart; weak and irregular pulse; pallor of the face; excessive thirst; congestion of the conjunctivæ and of the respiratory tract, slight spasm of the affected muscles. Consciousness is preserved.

The **etiology** is not definite. The hereditary character of the disease is well known. Holtzapple reports seventeen cases in four generations. The exciting causes may be either rest or exertion. In the first case the attacks occur after a night's sleep or after the patient is seated and rests a certain time. In the second case violent exertion brings on an attack. In one of my patients, girl of fourteen, an attack occurred after a long walk or dance; she would develop paralysis first in the arms and then in the legs; it lasted from twenty-five minutes to eight hours. Her uncle was similarly affected. Holtzapple observed attacks after indulgence in food.

The **prognosis** as a rule is favorable to life, because the attacks become less frequent with advancing age. Holtzapple, however, observed six deaths during attacks. Difficult respiration during an attack is of serious moment.

The **pathogenesis** of the disease is unknown. A diminished secretion of urea was found in several members of the same family. Necropsies have been negative. The trouble probably lies in a developmental defect of the muscles themselves.

Treatment.—Avoidance of overexertion, of stimulants, of over-feeding is indicated. In my case mentioned above I succeeded in shortening the attacks with the administration of sodium bromide in gr. x doses every two hours. Massage was also used in my case. The results were satisfactory.

DISEASES OF INDIVIDUAL NERVES

I. Paralysis of Cranial Nerves

A. Paralysis of the First Nerve (Olfactory). Symptoms.—Loss or impairment of the sense of smell (**anosmia**) is the prominent symptom. As the taste is dependable to a large extent upon the integrity of smell, the former will be involved when anosmia is present. When the olfactory nerve is in a state of irritation, hallucinations of smell (parosmia) may occur. Subjective sensations of smell may occur as an aura preceding epileptic seizures in cases of tumor of the uncinate portion of temporo-sphenoidal lobe.

Etiology.—Diseases of the nasal cavities (in the upper portion of which are distributed the fine olfactory nerves), viz. tumors, catarrh, diseases of the bony structure, meningitis at the base, trauma of the nose,

fracture at the base of the skull in the anterior fossa—are all apt to irritate or destroy the filaments of the olfactory nerve.

Treatment.—Removal of the cause is the sole therapeutic indication.

B. Diseases of the Second Nerve (Optic) are discussed in the chapter on Diseases of the Brain.

Optic Neuritis and **optic atrophy** were mentioned and their significance emphasized in intra-cranial diseases (especially tumors, abscess of the brain, basilar meningitis, choked disc in cerebellar tumors), in tabes, insular sclerosis, Freidreich's ataxia, paresis. It may be added here that optic neuritis may be encountered also in acute febrile diseases, toxic conditions (see polyneuritis), anæmia, diseases of orbital region, Bright's disease, gout. Optic atrophy may also develop in syphilis, malaria, diabetes, abuse of tobacco, narcotics or other drugs (quinine).

In **optic neuritis** the disc is swollen and hyperemic. When the veins become greatly distended and œdema develops because of the interference with the return circulation, the condition is **"choked disc."** Vision may remain intact in optic neuritis and even in choked disc. On the other hand, there may be various degrees of impairment of vision or complete loss of vision.

Loss of vision (amaurosis) may occur in the course of infectious diseases and develop very rapidly. Widal, Joltrain and Weill (*Presse. Méd.*, 1909) report a case of amaurosis in typhoid fever accompanied by œdema of both papillæ and hypertension of the cerebro-spinal fluid. Two lumbar punctures produced complete recovery from the eye symptoms.

Optic atrophy may be primary like in tabes or multiple sclerosis, or secondary following optic neuritis. In primary optic atrophy the loss of vision is usually gradual. Besides, there may be loss of vision only in one portion of the visual field, either centrally or peripherally.

Neuritis and atrophy of the optic nerve may be the result not only of organic diseases of the nervous system (see above), but also of localized disturbances in the nerve itself, such as embolism of the central artery, hemorrhage and trauma.

The **pupillary reactions** are frequently affected in diseases of the optic nerve; impairment or loss of light reflexes is common.

Diseases of the **optic chiasm** were discussed in the chapter on **Hemianopsia.**

C. Paralysis of Third, Fourth and Sixth Nerves (Opthalmoplegia.)

Symptoms.—They are **general** and **special** to each of the three nerves.

General.—**Strabismus** is very conspicuous. It consists of a deviation of the eye globe. In the view of the paralysis the excursion of the latter is limited. For example, if the external rectus (sixth nerve) is involved, the

eye globe will move in every direction except externally. Under **secondary deviation** is understood a deviation of the normal eye when the paralyzed eye endeavors to fix an object. This secondary deviation is always greater than the paralytic deviation.

The consequence of strabismus is **double vision** (**diplopia**) because the image of an object will be formed in both retinæ at various levels. Diplopia produces not infrequently vertigo and headache. Vertigo may disappear when the vision of the diseased eye is entirely removed or else corrected by some means.

Special Symptoms. (*a*) **Paralysis of the Third Nerve** (**Oculomotor.**)—It may be **complete** or **partial.**

In **complete** palsy the levator palpebræ superioris, the superior, inferior and internal recti, the inferior oblique, the sphincter of the pupil and the ciliary muscles are all involved. **Ptosis** is the striking symptom. The upper eye-lid is lowered. The eye globe is deviated externally and downward. The pupil is dilated, does not react to light or to accommodation. **Diplopia** is marked: one image is placed higher than the other.

In **partial** paralysis any one of the muscles may be affected. When the internal rectus alone is paralyzed, there is divergent strabismus: the images in both eyes are at the same level. When the inferior rectus is paralyzed, the eye looks upward and outward, the head is lowered. In paralysis of the superior rectus, the head is raised and thrown backward. When the inferior oblique is involved, the eye is directed downward and inward.

In **nuclear lesions** of the third nerve the internal muscles may escape, but the external are paralyzed.

(*b*) **Paralysis of the Fourth Nerve** (**Pathetic**).—The superior oblique is involved. The eye globe looks upward and inward. Diplopia exists only in the lower part of the visual field.

(*c*) **Paralysis of the Sixth Nerve** (**Abducens**).—The external rectus is involved. The eye globe is turned inward (convergent strabismus).

Ocular palsies may be confined to one nerve or to all the three nerves. As to the third nerve several branches may be affected at the same time. **Ophthalmoplegia** is present when all the muscles of the eye are involved or at least the muscles innervated by two nerves, one of which is the third nerve. In **external** ophthalmoplegia the extrinsic muscles of the eye, in **internal** ophthalmoplegia the intrinsic muscles (the sphincters of the pupil and the ciliary muscles) are paralyzed.

Ophthalmoplegia may be **nuclear** and **peripheral.** The first is the most important. When it is **nuclear** and **external,** it is usually **bilateral.** The facies of the patient is then characteristic: the ptosis gives a drowsy,

sleepy expression, the eye globes are immobile, but the pupillary and accommodation reflexes are normal. **Alternating ptosis** is quite frequent. When the ophthalmoplegia is **nuclear** and **internal**, mydriasis is present and the pupils do not react to light, to accommodation and to convergence.

Pupillary reactions. Iridoplegia is present when the light reflex of the pupil is lost. If with the latter the accommodation reflex is preserved, we are in presence of **Argyll-Robertson** pupil. The lesion in this phenomenon is very probably in the ciliary ganglion according to the researches of Marina and others.

Paralysis of accommodation or **Cycloplegia** is met with in syphilis and diphtheria; it is due to a palsy of the third nerve.

Associative Paralysis: (1) **Paralysis of Conjugate Movement.**—It consists of abolition of a movement common to both eyes, for example, paralysis of internal rectus of one eye and of external rectus of the other. This form is not produced by a paralysis of the nerves themselves, but by a lesion of the sixth nucleus which besides his numerous other connections it is associated through the posterior longitudinal bundle with that part of the third nucleus which supplies the internal rectus muscle of the opposite eye. (2) **Paralysis of convergence** of internal recti of both eyes is probably due to a lesion of the nuclei of the third nerve. It is usually associated with paralysis of accommodation.

Ocular palsies are frequent in tabes. Their onset is sudden and characterized frequently by a paralysis of the third nerve and their predilection for the pupil (Argyll-Robertson).

Course, Termination, Prognosis.—They depend upon the cause. The course is variable, it may disappear and recur. In an advanced stage of tabes it may remain permanently. In syphilis ocular palsies are slow in onset, but progressive. They also affect chiefly the third nerve. Like in tabes they are peripheral in origin. They may be promptly ameliorated by salvarsan, mercury and iodides. In **multiple neuritis** ocular palsies disappear when the other symptoms improve. Ocular palsies following infectious diseases usually recover. In tumors, hemorrhages, fractures, the ocular palsies are incurable. **Nuclear palsies** do not recover.

Etiology.—Diseases of the brain may produce a conjugate deviation of the eyes, also a paralysis of the levator palpebræ (blepharoptosis). **Tumors** or other lesions at **the base of the brain** frequently produce ocular palsies in addition to crossed hemiplegia. In **Polioencephalitis** superior (see this chapter) nuclear ophthalmoplegia is a frequent accompaniment. In **tabes** and in paresis the third nerve is frequently involved. The same is observed occasionally in multiple sclerosis and in syringomyelia. In **polyneuritis** (see this chapter) ophthalmoplegia is observed. Various

infectious diseases, intoxications may be the cause of palsy of the ocular muscles. **Syphilis** plays a prominent rôle in the causation of this paralysis. **Traumatism** of the orbital region, compression, fractures at the base of the brain, aneurisms of the arteries at the base, thrombosis of the cavernous sinus, tubercular meningitis or gummata at the base will all involve the motor ocular nerves. Sinusitis, frontal and ethmoidal, may be the cause of ocular palsies either directly or through an inflammatory condition of the orbit. Finally ocular palsies may be congenital.

Treatment.—Removal of the cause is the first indication. In syphilis mercury and iodides are necessary. In toxic or infectious cases bloodletting and sweating may be of benefit. Hypodermic injections of strychnia yield sometimes good results. Electrical stimulation of the palsied muscles may be useful. Diplopia can be corrected either by special glasses or surgical means. The tendency to remissions is great.

Nystagmus.—It is an associated oscillation of the eyes which is accomplished symmetrically and more or less rapidly. Most frequently the nystagmus is **horizontal,** sometimes it is **vertical.** It may be also **rotatory.** In the majority of cases nystagmus is a convulsive phenomenon occurring mostly upon voluntary movement of the eyes. It is due to an irritative lesion of the center which controls associative movements. In exceptional cases it is unilateral.

Nystagmus may be **congenital** or hereditary. It may be present without any eye lesion or else it may accompany cataract, albinism, coloboma, strabismus, errors of refraction.

Myoclonic nystagmus described by Lenoble and Aubineau is a congenital condition consisting of nystagmus accompanied by facial asymmetry and tremor of the muscles of the face and increased reflexes.

Nystagmus is most frequently **acquired.** It may be the result of impaired vision and when the latter improves, the former disappears. Occasionally it is observed in normal individuals during fixation of the eyes on an object. It is met with in the following diseases of the central nervous system: Multiple sclerosis, Friedreich's disease, heredo-cerebellar ataxia, syringomyelia, very frequently in cerebellar diseases, in lesions of the quadrigeminal bodies. In diseases of the medulla when the lesion extends to the pons nystagmus is observed. Here the posterior longitudinal bundle or the centers of association are involved. Thus it is met with in Pseudo-bulbar palsies and in tumors or focal lesions of the pons. In diseases of the brain nystagmus may occur. It may depend upon the distant effect of the cerebral lesion on the posterior cerebral fossa. It may depend also on a lesion of the cortical areas which control the movements of the eyes, viz: frontal lobe, angular gyrus, visual area.

Vestibular Nystagmus.—The involvement of the labyrinth causes nystagmus. The reflex is transmitted to the oculo-motor nerve from the vestibular apparatus through the posterior longitudinal bundle and cerebellar peduncles. The nystagmus is due to a displacement of the endo-lymph fluid in the semicircular canal. Horizontal nystagmus is caused by excitation of the horizontal canal, vertical by excitation of the superior vertical canal, rotatory by excitation of the posterior vertical canal. Bárány's test consists of rotation, caloric and galvanic excitation. In **rotation** (patient is seated on a turning chair, head erect) the nystagmus is in a direction of the rotatory movement, but as soon as the rotation is interrupted, the nystagmus is the opposite direction. Nystagmus of **thermic** origin produced by irrigation of the ear is in a direction opposite to the irritated ear if the water is cold and *vice versa* if warm water is used. In **galvanic** test if the cathode is applied to the mastoid, rotatory nystagmus is in the direction to the same side. If the anode is applied, the nystagmus is to the opposite side.

When the labyrinth is destroyed, the above tests, especially the caloric test, will not be able to produce the least nystagmoid movement of the eye globes. Absence of reflex therefore is an indication of disease of the labyrinth.

Bárány has observed that 60 per cent. of normal individuals present a slight nystagmus on looking to the extreme right or left. In labyrinthine disease on one side the nystagmus is very evident when the eyes are turned to one or the other side, while normally it is present on looking to either side. Besides, pathological nystagmus is generally well developed, while normally it is very imperfect. Continuous nystagmus associated with vertigo is present in purulent disease of the labyrinth. In cerebellar abscess there is also nystagmus associated with vertigo, but here the nystagmus is directed to the side of the lesion, while in labyrinthine diseases to the sound side.

Vestibular nystagmus differs from nystagmus of multiple sclerosis or of other ocular disorders in that the former consists of unequal oscillations, one movement being less rapid than the other and the oscillations become increased when the eyes are turned toward the shorter movement, diminished if the eyes are turned toward the slower movement.

Miners' nystagmus is an occupation disorder occurring in individuals working in coal mines. It is of the **rotatory** form. There is a benign variety which does not interfere with work and a grave variety which is much less frequent. The nystagmus occurs in the benign cases only when the eyes are directed upward. In the severe cases it will occur when the eyes are directed downward. The individual sees objects moving continuously

before him (dancing) which of course prevents him from working. The **cause** if this nystagmus lies in forced elevation and oblique direction of the eyes and poor light in the mines.

Perhaps the displacement of labyrinthine endo-lymph caused by forced elevation of the head is the cause of the nystagmus.

Hippus.—It consists of alternating dilatation and contraction of the pupils. It sometimes accompanies nystagmus. It is probably due to a dynamic disturbance of equilibrium in the movements of the iris.

D. Paralysis of the Fifth Nerve (Trigeminal). Symptoms.—As the fifth nerve consists of sensory and motor portions the symptoms will be **sensory** and **motor.**

Sensory.—The area of the skin supplied by the nerve will be **anæsthetic,** viz. conjunctiva, cornea, cheek, nose, lips, mouth, gums and tongue. The external auditory meatus and the skin over the largest portion of the lower maxilla are not involved. The corneal and lid reflexes are abolished. The secretions of the eyes and nose are impaired. Smell and taste are also impaired. Trophic disturbances are frequent. Herpes is common and herpes zoster ophthalmicus is due to neuritis of the first branch of the fifth nerve.

Neuroparalytic Keratitis is not infrequent. It is characterized by an inflammation and ulceration of the eye. According to the latest view on this affection the keratitis is not due to a disease of supposed trophic fibers of the fifth nerve, but to an **irritation** of the trigeminal fibers.

Motor.—The masticatoy muscles (masseter, temporal and pterygoids) are paralyzed and in unilateral paralysis the tongue instinctively pushes the food toward the normal side in the act of mastication. The unilateral involvement of the pterygoid muscles produces a deviation of the lower jaw to the paralyzed side when the mouth is opened. The affected muscles undergo atrophy and in this case they show reactions of degeneration.

When the paralysis of the fifth nerve is **incomplete,** as it happens in cases of compression, the sensory and motor symptoms are also incomplete. There will be only diminished sensations (hypæsthesia) instead of anæsthesia, but the continuous irritation from the compression will produce pain. If the condition continues, complete anæsthesia will finally develop. In diseases of the pons the spinal root of the fifth nerve may be involved; there will be facial anæsthesia on the same side and paralysis of the arm and leg on the opposite side.

The **prognosis** depends upon the original cause.

Etiology.—The fifth nerve is rarely affected primarily. It usually becomes involved in the course of other diseases. Lesions at the base of the brain (meningitis, syphilis, tumors, hemorrhages, aneurism), otitis

media, caries of the sphenoid, trauma of the orbits or of the maxiliæ—are all causes of disease of the fifth nerve. It is rarely involved in polyneuritis. It may also be affected in tabes, in syringomyelia and its motor nucleus in bulbar palsy. Unilateral trigeminal palsy associated with paralysis of the eighth nerve is frequently met with in tumor of the ponto-cerebellar angle (see this chapter).

Treatment.—Removal of the cause is the first indication. Specific drugs should be administered when syphilis is suspected. Operations for removal of growths, etc., will be resorted to when medications fail. Pain and atrophic disturbances will be treated with appropriate remedies. The eye should be protected against injury or irritation.

E. Paralysis of the Seventh Nerve (Facial) (Bell's Palsy).—Facial palsy may be of **cerebral, nuclear** and **peripheral** origin. The latter two are identical in their manifestations. The description that follows will be that of peripheral facial palsy. In discussion on diagnosis the differential symptoms of cerebral facial palsy will be given.

Etiology.—Cold is a frequent cause (rheumatic paralysis). **Trauma** is next in frequency. It may be produced by fracture of the petrous bone, by a blow or operative procedures over the parotid region, and in obstetrical cases by pressure of the nerve against the pelvis or else by the forceps. Lesions in the vicinity of the nerve, as tumor, abscess, exostosis, caries of the petrous bone, otitis media are apt to affect the nerve. Infectious diseases and intoxications are occasionally accompanied by facial palsy. In the tertiary period of syphilis it may be encountered. An isolated involvement of the seventh nerve in the course of syphilis is rare. In the Archives of Diagnosis, 1908, I published records of six cases in which Bell's palsy was caused by syphilis. In two patients the facial paralysis occurred as early as two months after the initial chancre. It may appear in the course of polyneuritis, alcoholic or lead. It may develop in syphilitic meningitis at the base of the brain, also in the course of tumors of the cerebello-pontile angle. **Neuropathic** predisposition plays a certain rôle.

Pathology.—The morbid changes consist of a parenchymatous neuritis. The usual seat of the lesion is either in the Fallopian canal or in the stylo-mastoid foramen, or else at its exit from the latter.

Symptoms.—The onset of facial palsy is sudden in cases due to cold. It is usually preceded by pain in the neck or back of the ear. It develops slowly when caused by other factors.

In Bell's palsy the striking symptom is **asymmetry** of the face. The latter is deviated to the normal side. The muscles on the entire paralyzed side have lost their normal tonicity and the voluntary motility: they are

relaxed. The naso-labial fold is only slightly marked and the angle of the mouth is lowered. The wrinkles disappear. The asymmetry becomes more evident when the patient attempts to laugh, to show his teeth. Blowing and whistling are impossible. In the latter acts the air raises the cheek. Liquid food runs out of the mouth on the paralyzed side. The forehead on the affected side is also smooth and when the patient is told to wrinkle it, the muscles on the paralyzed side remain immobile (paralysis of the frontal muscle).

The **eye** remains widely open because of the paralysis of the orbicularis palpebrarum. As closure of the eye is impossible, the conjunctiva and cornea being continuously exposed, become inflamed. Bell called attention to the rotation of the eye globe upward and outward at the attempt to close the eye.

FIG. 130.

The **tongue** when protruded appears to be deviated. The **taste** on its anterior two-thirds is sometimes diminished or abolished (involvement of the chorda tympani). The **palate** is relaxed and the uvula deviated. The sense of taste is perceived by the mucous membranes of the tongue and soft palate including the palatine arches. The anterior two-thirds of the tongue are supplied by the chorda tympani, the posterior third by the ninth nerve, the soft palate by a branch from Meckel's ganglion. The facial nerve may be regarded as a mixed nerve. It has a ganglion (geniculate). The sensory fibers are: the great superficial petrosal, the small superficial petrosal and the external petrosal supplying the carotid plexus of the sympathetic. The geniculate ganglion sends fibers running with the seventh nerve in the Fallopian canal, also fibers anastomosing with the fifth and eighth nerves. The sensory fibers of the seventh nerve are represented in the auditory nerve, the tympanic plexus (supplied by the petrosal nerves), the auricle and the external auditory canal.

R. Hunt (*J. Nerv. and Ment. Dis.*, 1907) has brought out a syndrome of the sensory branches of the seventh nerve occurring in facial palsy. The symptoms are: (1) pain in the ear and mastoid region. The pain may be of neuralgic character. Anæsthesia or hyperæsthesia in the region of

concha and external auditory meatus, also hypæsthesia in the chorda tympani distribution. (2) Herpetic otalgia, herpes oticus. The eruption is distributed over the tympanum, external auditory canal and concha. (This is the herpetic area of the geniculate ganglion.) (3) Reflex facial twitchings and spasms.

The **hearing** is sometimes affected. There may be also hyperacuity of hearing.

The **electrical** contractility of the paralyzed muscles is very important. While in some cases (mild) it is normal, in others it is altered quantitatively and qualitatively. Reactions of degeneration are present.

Among the rare symptoms of peripheral facial palsy may be mentioned absence or diminution of sweating of the skin on the affected side. This is observed in the severe forms. Vaso-motor disturbances are sometimes observed.

FIG. 131—RIGHT FACIAL PARALYSIS SHOWING INVOLVEMENT ONLY OF THE UPPER FACIAL BRANCH.

Course, Termination, Prognosis.—Facial palsy due to obstetrical causes has a favorable course, it lasts usually from eight to fifteen days. When the nerve is destroyed by a fractured bone, the paralysis is incurable. In cases of facial palsy due to the middle-ear diseases the outcome is serious: the palsy persists. In cases produced by **cold** (the most frequent variety) the palsy may be mild and complete; recovery follows in from two to three weeks. In other cases in which slight reactions of degeneration are present, the disease may last two to three months. In cases in which the faradic contractility is absent for a period of two to three months, the prognosis is uncertain but not always serious, as I have seen such cases improve greatly after persistent electrical stimulations. In grave cases, in which the reactions of degeneration are marked or electrical irritability is entirely absent, recovery may be only partial or not at all. In cases with incomplete recovery the paralyzed muscles become permanently contracted. The contractured muscles draw the mouth

toward the paralyzed side, the naso-labial fold is deep, the palpebral fissure is small, so that the impression may be formed that the paralysis is on the normal side. It is then sufficient to observe the patient in voluntary acts (laughing, showing teeth, etc.), to determine which half of the face was originally paralyzed. In facial palsy of nuclear origin the prognosis is very serious.

Diagnosis.—The deviation of the face, the lowering of the angle of the mouth, the disappearance of wrinkles, the inability to close the eye and to wrinkle the forehead are sufficient symptoms to recognize a peripheral facial palsy.

Cerebral Facial Palsy.—A cerebral lesion producing a hemiplegia quite frequently involves also the face. This is due to an involvement of the fibers passing from the cortical center of the face through the internal capsule together with those from the arm and leg centers and joining the nucleus of the facial nerve in the pons on the opposite side (see Anatomy). Therefore a facial palsy of cerebral origin almost always accompanies a hemiplegia.

In addition to this the following symptoms are characteristic of cerebral facial paralysis: (*a*) preservation of the musculus frontalis and orbicularis palpebrarum. This is possibly due to the fact that the upper branch of the facial nerve receives its fibers from a different tract (perhaps from the third nerve at the lowest level of the peduncles or sixth nerve) or else it is innervated by both hemispheres; (*b*) normal electrical reactions of the affected muscles; (*c*) absence of atrophy; (*d*) paralysis of the voluntary movements of the mouth and preservation of emotional movements. To sum up, in cerebral facial paralysis only the lower part of the face is affected, while in peripheral or nuclear palsy the upper and the lower halves of the face are paralyzed.

An isolated involvement of the upper branch of the facial nerves has occasionally been reported. The accompanying photograph shows a paralysis of the right musculus frontalis and orbicularis palpebrarum without any other central or peripheral symptom (Fig. 131). In this case there was also a dilatation and a sluggish light reaction of the pupil on the same side. The condition of the pupil points to a physiological connection of the seventh with the third nerve. Indeed Mendel long ago maintained the idea that the upper branch of the facial nerve takes its origin in the lower part of the oculo-motor nucleus (*Neurol. Centralblatt.*, 1898). He found atrophy of the cells in this nucleus after section of the upper facial nerve in rabbits. Besides, in bulbar palsy and amyotrophic lateral sclerosis there is a comparative escape of the muscles of the upper face, while those of the lower are involved. In spite of this evidence, not all

are agreed on a separate origin of the upper branch of the facial nerve. The majority of writers believe that it arises from the dorsal part of the facial nucleus.

In **nuclear** palsy there is frequently a simultaneous involvement of the seventh and the sixth nerve in view of the contiguity of the nuclei, also involvement of the limbs.

Double Peripheral Facial Palsy (facial diplegia) may simulate bulbar and pseudo-bulbar palsies. In the first there are usually present abnormal phenomena of deglutition, phonation and respiration, in the other there is an apoplectic seizure with cerebral phenomena. In facial diplegia the expression is characteristic. The mouth is open, saliva is continuously running, also tears are on the cheeks. The eyelids and the entire musculature of the face are immobile. The voice is nasal; mastication is difficult. Facial diplegia is due most of the time to a bilateral lesion of the petrous bones or basal meningitis, also to bilateral tumors of the cerebello-pontine angle. In the latter case there is also bilateral deafness.

In peripheral facial palsy it is important to determine the **seat of the lesion.** It can be facilitated by the following special symptoms:

(*a*) A lesion between the stylo-mastoid forearm and the periphery will involve only the facial muscles. (*b*) A lesion in the Fallopian canal below the geniculate ganglion will involve also the taste on the anterior two-thirds of the tongue (chorda tympani) and the hearing. (*c*) A lesion at the level of the geniculate ganglion and above it will involve the palate in addition to all the other symptoms except the taste. (*d*) A lesion at the base of the brain will involve simultaneously the eighth nerve, and deafness will appear at the same time as paralysis of the face.

Treatment.—Removal of the cause is the first indication. Tumors, abscesses, otitis media will be treated accordingly. When a syphilitic history is present, iodides and mercury will be given. Cases which are due to **cold** and which come under observation at or shortly after the onset of the palsy should be treated at first with vesicants or bloodletting back of the ear. Warm applications, sedatives or coal-tar products (salicylates, aspirin, phenacetin) will relieve pain. At the end of ten days after the acute symptoms have subsided, galvanic applications to the paralyzed muscles must be instituted. At first they will be daily, but later every other day ten to fifteen minutes' application is sufficient. At the end of a few weeks faradism can be substituted for the galvanism. Massage is a good adjuvant to electricity. A daily rub for five to ten minutes will be useful. Secondary contractures are not easily remedied, although the treatment should be kept up indefinitely. I have obtained good results in very old cases after a prolonged treatment. The surgical treatment

consists of anastomosis of the facial nerve with the spinal or with the hypoglossus. Satisfactory results have been obtained from both procedures.

F. Paralysis of the Eighth Nerve (Auditory).

Etiology.—Middle ear or labyrinth diseases, trauma, caries and other diseases of the bones at the base, meningitis (especially its cerebrospinal form), syphilis—may all be accompanied by a neuritis of the eighth nerve. A basal disease rarely involves only the eighth nerve, the facial nerve also suffers. Degeneration and atrophy of the eighth nerve are occasionally observed in tabes, paresis, multiple sclerosis. A prolonged use of quinine may produce degeneration of the acoustic nerve.

Symptoms.—As the eighth nerve consists of two portions with different functions (see Anatomy), the symptoms will differ according to whether the cochlear or the vestibular nerves are involved.

(*a*) **Cochlear Nerve Symptoms.**—The function of this nerve is hearing. Disturbances of hearing will result from diseases of this nerve.

Tinnitus Aurium is one of the symptoms. It is characterized by subjective sounds, as ringing, buzzing, etc. If the condition continues, deafness may be the result. In making a diagnosis it should be borne in mind that tinnitus may be caused by any disease of the auditory system or by general diseases. Thus anæmia, hyperæmia and aneurism of cerebral vessels; otitis externa or media, cerumen in the external ear—are all apt to produce peculiar sounds.

Deafness, partial or complete, may be caused by a lesion of the cochlear nerve as well as by diseases of the ear itself. The differential diagnosis is based upon the following facts. If deafness is due to a disease of the middle ear, the tuning fork is not heard when kept near the ear, but heard when applied to the temporal bone. In a disease of the cochlear nerve the fork is not heard when applied to the bone. In diseases of the middle ear hearing is increased in a noise but not increased in diseases of the cochlear nerve. **Congenital** or **hereditary deafness** is probably due to a primary atrophy of the eighth nerve.

(*b*) **Vestibular Nerve Symptoms.**—Anatomically the nerve originates in the semilunar canals and labyrinth. Its function is to maintain equilibrium. The reason of it lies mainly in the connections existing between the nuclei of this nerve and the cerebellum, which is the chief organ of equilibrium. An affection of the nerve, or of its nuclei, produces **vertigo.** The nuclei connecting the vestibular nerve with the cerebellum (middle and lateral lobes) are: vestibular, Deiter's, and dorsal auditory (see Anatomy).

It should not be forgotten that vertigo may result from other causes

besides a lesion of the vestibular. Thus diseases of the ear, of the ocular apparatus, of the brain, of the viscera, may be attended by vertigo. It may also be present in hysteria and neurasthenia. A detailed discussion on the differential signs in those various affections will be properly given in the chapter **"Vertigo."** The reader is also referred to text-books on ear diseases for a detailed study of tinnitus aurium and deafness.

Prognosis.—It is unfavorable, as the underlying cause is a degeneration and atrophy of the auditory nerve.

Treatment.—Electricity, and especially the galvanic current, may sometimes relieve the tinnitus aurium (anode to the diseased ear), but not much reliance can be placed on it or on medications. Pilocarpin, quinine may sometimes yield some results. Counter-irritation over the temporal bone (petrous bone) and strychnia may be of some use. In syphilis iodides and mercury give good results.

G. Paralysis of the Ninth Nerve (Glossopharyngeal).

Etiology.—Tumors, gummata, aneurismus, injuries, thrombosis of the jugular veins—are the causes in the diseases of the ninth nerve. An isolated paralysis of this nerve is rare.

Symptoms.—Some of the motor and sensory fibers of the ninth nerve are intimately connected with those of the tenth nerve so that it is difficult to say that the functions of the first depend upon one or the other nerve. However, it is admitted that when the ninth nerve is involved, the following symptoms are observed: **Anæsthesia** of the pharynx, palate and middle ear, **ageusia** (loss of taste) of the posterior third of the tongue and gums, difficulty of **swallowing,** of phonation and respiration (paralysis of some muscles of larynx, pharynx and œsophagus).

H. Paralysis of the Tenth Nerve (Pneumogastric).

Etiology.—In alcoholic and diphtheritic polyneuritis the tenth nerve is frequently involved. Various diseases at the base of the brain or skull (meningitis, tumors, hemorrhages, etc.), suppuration or tumors of the neck and mediastinum, injuries on the neck, pericarditis, ligation of the carotids, are all apt to injure the vagus. Finally a neuritis of rheumatic nature not infrequently involves the recurrent laryngeal nerves (see Anatomy).

Symptoms.—Paralysis of the **larynx, fauces** and **palate** are the most prominent symptoms. Phonation is disturbed. Speech is nasal. Swallowing is only slightly disturbed.

In **unilateral** paralysis there is acceleration of the heart-beat, also irregularity of respiration.

The most important symptoms are those concerning the **larynx.** This organ is supplied by two branches of the tenth nerve, viz. superior

laryngeal and recurrent laryngeal. The first contains sensory fibers for the mucous membrane of the upper portions of the larynx and motor fibers for the epiglottis and cricothyroid muscle. The second supplies all other muscles and the mucous membrane of the larynx below the vocal cords and trachea. The vocal cords are paralyzed and remain immovable during phonation and respiration. In unilateral paralysis the voice is hoarse and a deep inspiration produces stridor, there is no cough. In bilateral paralysis aphonia and dyspnœa are present, stridor only on deep inspiration, and there is no cough.

In **nuclear involvement** (which occurs in bulbar palsies) of the vagus respiratory and laryngeal palsies are manifested by rapid heart action and Cheyne-Stokes' respiration. There is usually an associated paralysis of the muscles of the soft palate and of pharynx. Paralysis of the palate is manifested in disturbed phonation.

Prognosis.—It is always grave. A nuclear palsy of the vagus caused by a hemorrhage or embolism may produce sudden death.

Treatment.—In syphilis mercury and iodides may be of benefit. In palsy of the vagus, caused by alcohol, stimulants are urgent: alcohol should be then freely administered. Iodides are useful in lead laryngeal paralysis. Electricity applied externally to the larynx may be of benefit.

Paralysis of the **recurrent laryngeal** nerve deserves special mention. The paralysis may be **total or partial.** In the first case the vocal cord is in an intermediary position and all the muscles supplied by the nerves are paralyzed. In the second case the vocal cord is immovable in median position, which is due to paralysis of the abductor, viz. posterior crico-arytenoid muscle. The partial paralysis is more frequent than the total, on the left side more than on the right and oftener in men than in women.

The affections in which involvement of this nerve is observed are: (a) **diseases of the thyroid gland,** goiter especially, in which case it is due to compression. Thyroidectomy is not rarely followed by paralysis of the recurrent nerve. In this case it may be due to pulling, pinching with forceps during the operation or else by a cicatrix during the healing process. Most frequently the paralysis is partial. If it is unilateral, the sound cord may compensate the impairment of the affected cord, but in bilateral paralysis there are aphonia and respiratory disturbances. (b) **Aneurism of the Aorta and Aortitis.**—The relation of the left recurrent nerve to the concave portion of the arch of the aorta explains the frequency of the nerve paralysis in aneurism. Not only compression of the aneurism, but also an inflammatory process spreading from the outer wall of the aorta to the nerve is the cause of paralysis of the latter. The right recurrent nerve is very rarely affected. More rarely is observed a bilateral in-

volvement. (c) **Cancer of the œsophagus** causes the disease through compression of the peritracheo-laryngeal glands which are frequently infiltrated in cancer of œsophagus. (d) In cancer of the lungs and (e) in pulmonary tuberculosis, the infiltrated pretracheobronchial glands being in contact with the recurrent laryngeal nerve, produce its paralysis. (f) **Mitral stenosis** has occasionally been the cause of paralysis of the left recurrent nerve. (g) In **Tabes** paralysis of this nerve has been observed and it is probably due to a peripheral neuritis (Dejerine and Petren). (h) Toxic and infectious causes: lead, arsenic, typhoid fever, diphtheria. (i) Finally there may be a primary neuritis the cause of which cannot be ascertained.

I. Paralysis of the Eleventh Nerve (Spinal Accessory).

Etiology.—Colds, trauma of the neck, diseases of the cervical vertebræ, abscesses of the neck, tumors in the same vicinity, cervical myelitis—are all apt to compress, irritate or destroy the eleventh nerve. Finally a **primary neuritis** is also possible.

Symptoms.—The inner branch of the eleventh nerve is in connection with the tenth nerve and controls phonation movements of the larynx. We will therefore be concerned here exclusively with the external branch. The latter innervates the muscles sterno-cleido-mastoid and trapezius. In unilateral paralysis of the first muscle that of the opposite side will bend the head towards it and the face will turn towards the paralyzed muscle. The deviation will be only slight, as the sterno-cleido-mastoid muscle is, besides the eleventh, also supplied by second and third cervical nerves. When the paralysis is bilateral, the head cannot be held straight, but falls backward.

When the trapezius is involved, a marked deformity ensues. The function of this muscle is to turn the head backward and elevate the shoulder. In case of paralysis, the serratus not being counteracted rotates the scapula so as to project its inner angle upward, and when the arm is brought forward, the scapula is no more held against the thorax, but projects. In bilateral paralysis shrugging of the shoulder is impaired.

In advanced cases atrophy, reactions of degeneration, also contractures develop. The deformity is then pronounced. Trapezius and sternomastoid muscles may be paralyzed in the course of progressive muscular atrophy, in syringomyelia, in tabes, when the cells of the anterior cornua of the upper portion of the cord become involved.

Prognosis depends upon the cause. Syphilis has the most favorable outlook.

Treatment consists of electricity and massage. In cases of tumors an operation is indicated. Old cases with contractures will be treated

by tenotomy or orthopedic appliances. In syphilitic cases mercury and iodides should be given.

J. Paralysis of the Twelfth Nerve (Hypoglossus).

Etiology.—Diseases at the base of the skull (tumors, hemorrhages, caries), aneurism of vertebral arteries, dislocation of upper cervical vertebræ, direct injury—are all causes of paralysis.

Symptoms.—In unilateral paralysis the tongue is **deviated** towards the side of the paralysis. This can be explained by the action of the genio-hyo-glossus of the normal side. The affected half of the tongue is **atrophied,** flabby and wrinkled. Fibrillary contractions are marked. Reactions of degeneration are present. **Hemiatrophy** of the tongue does not interfere to a great extent with **mastication** and **deglutition.** These two acts are decidedly impaired in total paralysis of the tongue: the latter lies motionless in the mouth. Articulation of words is indistinct in unilateral paralysis, but marked in bilateral. In **nuclear** disease paralysis of the tongue is associated with paralysis of the lips.

Prognosis.—It depends upon the cause. Recovery has been observed in syphilitic cases.

Treatment.—The original cause must be removed whenever it is possible. Electricity may be of some use. Mercury and iodides should be given, when syphilis is suspected.

II. Paralysis of Spinal Nerves

A. Upper Cervical Nerves

The third and fourth cervical roots give off filaments to form a nerve the function of which is of extreme importance. This is the **phrenic nerve.**

Paralysis of the Phrenic Nerve

Etiology.—Cervical or mediastinal tumors, infectious diseases (diphtheria, etc.), intoxications, toxic polyneuritis, (alcohol, lead, carbon monoxide), Potts' disease, dislocations or fractures of the vertebræ, finally diseases of the upper cervical cord (hemorrhages, tumors, myelitis, poliomyelitis, pachymeningitis, syphilitic meningitis)—are all causes of phrenic nerve palsy. Occasionally the phrenic nerve is involved in tabes. Finally in pleurisy and peritonitis the branches of the nerve distributed in the diaphragm may become affected.

Symptoms.—As the function of this nerve is mainly to innervate the diaphragm, a unilateral paralysis of it will produce **disturbances of**

respiration. Dyspnœa is the chief symptom. It is marked upon the least effort. The acts of coughing, expectoration, defecation and even talking increase the respiratory disorder. In severe cases, when the paralysis is bilateral, the dyspnœa is pronounced even when the patient is at rest. The patient is then threatened with asphyxia. On inspection the **diaphragmatic phenomenon** of Litten is noticeable. It consists of an epigastric depression during the act of inspiration and of a protrusion in the act of expiration. Pressure over the scalene muscles or between the lower insertions of sterno-mastoid muscle produces pain.

Course, Prognosis.—The disease is serious and the danger lies in asphyxia or when bronchitis or pneumonia develop. In the latter case the respiratory trouble is increased. The prognosis is graver when the paralysis depends upon a cord lesion than upon neuritis. It is also very grave in diphtheritic and quite serious in alcoholic neuritis. In making a **diagnosis** it should be borne in mind that hysteria sometimes simulates phrenic paralysis. The special stigmata, the sudden onset of the palsy after an emotion will decide the diagnosis of the hysteria.

Treatment.—Removal of the cause as promptly as possible is the main indication. Counter-irritation applied to the space between the bellies of the sterno-mastoid muscle may be of benefit. Strychnia and electricity are advisable. Asphyxia can be relieved by inhalation of oxygen. Anti-syphilitic treatment should be instituted when specific disease is suspected.

B. Paralysis of the Lower Cervical Nerves

Paralysis of the Brachial Plexus

The last four cervical roots and the first dorsal constitute the brachial plexus. A lesion of the roots cannot be differentiated from that of the plexus itself. In the description that follows we will be concerned with various palsies of the upper limb caused by a lesion extending from the point of emergency of the roots from the cord till the point where the individual nerves leave the plexus.

Etiology.—Injury of the shoulder, dislocation or fracture of the head of the humerus and of the clavicle, operative procedures in the same region, obstetrical manœuvers, forceps, forced reduction of dislocated humerus, forced extension or abduction of the arm, heavy weights on the shoulders (stone or hod carriers)—are all traumatic causes. Tumors or abscesses in the vicinity of the plexus, diseases of the bony tissue (caries, exostosis of the vertebræ or clavicle), localized meningitis, finally a neuritis of rheumatic, toxic and infectious nature are the non-traumatic causes. Cervical rib may be also the cause of the inferior type of brachial palsy (see below).

Symptoms.—The paralysis of the upper extremity is **total** when the lesion affects the entire plexus; **partial,** when only a portion of the plexus is involved.

Total Paralysis.—It is rare. It is usually preceded by a numbness or pain of neuralgic character. The entire upper limb is paralyzed. Abduction, adduction, flexion, extension, movements of the hands are all abolished. The **electrical reactions** are at first only diminished, but later show RD. **Atrophy** develops rapidly and appears first in the upper portions of the limb. The skin is cold and its secretions are diminished. **Sensations** are abolished. There is complete **anæsthesia** to touch, pain and temperature over the entire arm except on the internal aspect of the arm which is also innervated by the second and third dorsal nerves.

As the first dorsal nerve enters into the formation of the brachial plexus, in addition to the above symptoms there will be also **oculo-pupillary** manifestations, viz. myosis, narrowness of the palpebral fissure.

The **course** and **prognosis** depend upon the intensity of the involvement. While recovery is possible, however in the majority of cases partial paralysis and atrophy remain. Retraction of the tendons results in deformities of the arm or hand. Most frequently the paralysis is at first total, then gradually becomes limited to one or two nerves, chiefly musculo-spiral and circumflex.

Partial Paralysis.—It presents two main types: **Superior** and **inferior.**

(*a*) **Superior Type** (Erb's paralysis).—The following muscles of the shoulder and arm are involved: deltoid, biceps, brachialis anticus, supinator longus, supinator brevis, supra- and infraspinatus and the clavicular end of the pectoralis magnus. These muscles are controlled by that part of the brachial plexus which takes its fibers from the fifth and sixth cervical roots.

The disturbed function consists of an **inability to flex, supinate and abduct** the arm. The latter is in a state of extension. The forearm and hand are pronated. Atrophy with partial or complete RD develop rapidly. Sensations as a rule are not disturbed, but there may be anæsthesia in the areas of distribution of the circumflex and musculo-cutaneous nerves.

The symptoms just enumerated may present variations as to their intensity. Finally, the paralysis may be bilateral.

The upper type of brachial palsy is particularly frequent at birth. It is the so-called **Birth or Obstetrical Palsy** (Duchenne). It is almost always due to instrumental delivery (forceps) or difficult labor. The finger or tenaculum introduced to facilitate the delivery, also the forceps

may press directly on the shoulder and the brachial plexus. In breech presentation it is very frequent. The symptoms, course and prognosis are as described above with this difference, however, that the **prognosis is more favorable.** Complete recovery is not infrequent.

(*b*) **Inferior Type** (Klumpke).—Anatomically it is due to a lesion of the seventh, eighth cervical root and the first dorsal. It is characterized by paralysis of the flexors of the hand, thenar, hypothenar and interossei muscles. The hand is claw-like. Atrophy sets in early. Sensory disturbances consist of **anæsthesia** on the inner half of the forearm and hand (ulnar nerve); sometimes it extends to the area of distribution of the median nerve. Pain is not infrequently present.

Fig. 132.—Showing Position of the Arm in Birth Palsy.

As the first dorsal nerve is involved, **oculo-pupillary** symptoms are present. They are: myosis, sluggish contraction of the pupil, retraction of the eyeballs and narrowness of the palpebral fissure.

(*c*) **Complex Type.**—The two types of partial paralysis of the brachial plexus do not always present themselves as accurately localized as described above. Sometimes the lesion extends to neighboring roots. In other cases there may be a combination of both types, but each or only one of them is partially involved. In such cases the symptomatology is naturally complex.

Treatment.—It is that of neuritis in general (see this chapter). Removal of the cause is the main indication. Pain is relieved by appropriate means. Electricity and massage constitute the most important part of the treatment.

Paralysis of Individual Nerves of the Brachial Plexus.

(*a*) **Long Thoracic Nerve** (from fifth and sixth cervical roots).—It supplies the **serratus muscle.**

Etiology.—Injury or prolonged pressure on the neck (in carrying heavy weights on the shoulder), gunshot wounds of the neck, continuous elevation of the arm (as in plastering ceilings), infectious diseases (diphtheria, typhoid fever, grippe), finally exposure to cold—are the causes.

Symptoms.—Pain may or may not precede the onset of paralysis of the serratus muscle. The function of the latter is to rotate the scapula when the arm is put forwards, also to hold the scapula against the thorax.

In case of paralysis of the muscle the arm cannot be raised above a horizontal position and in the attempt to put the arm forwards the inner border of the scapula becomes separated from the thorax (wing-like).

The **Course** and **Prognosis** depend upon the cause. The disease lasts many months. Traumatic cases are the most unpromising. Rest of the arm (sling) is advisable. Massage and electricity should be instituted as early as possible.

(*b*) **Supra-Scapular Nerve** (from fifth, sixth, also fourth roots).—It supplies the **supra- and infraspinatus muscles.**

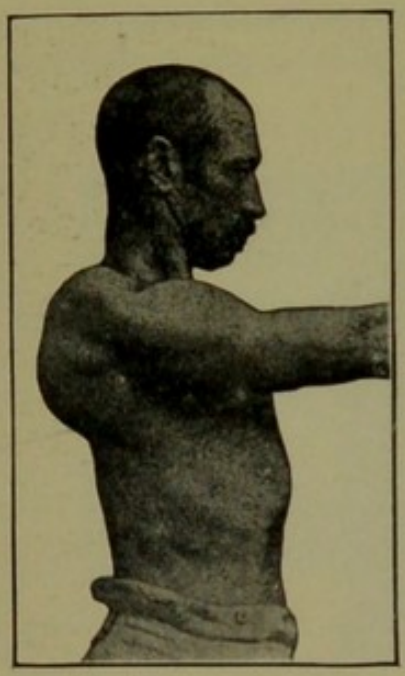


FIG. 133.

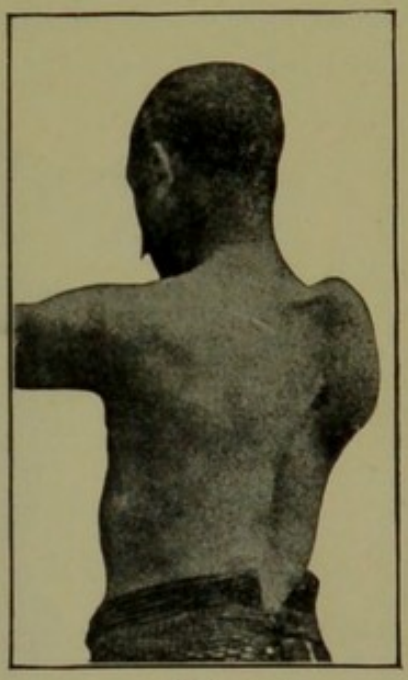


FIG. 134.

FIGS. 133, 134.—PARALYSIS OF SERRATUS MAGNUS CAUSING DISPLACEMENT OF SCAPULA WHEN THE ARM IS HELD FORWARD. (From *Nour. Iconogr. de la Salpêtrière.*)

Etiology—Injury to the shoulder, dislocation of the humerus, pressure upon the shoulder are the causes of palsy of this nerve.

Symptoms.—Pain may be present at the onset. The function of the infraspinatus muscle is to rotate the humerus outward. Its paralysis abolishes this function, and this is seen in the act of writing and sewing. The patient thus affected is unable to move the pen on the paper. Paralysis of the supraspinatus interferes with the act of raising the arm, also permits the head of the humerus to lose its firm position in the glenoid cavity. The muscles eventually atrophy. Pain is frequent and anæsthesia over the scapula is present.

Treatment.—Same as above.

(*c*) **Circumflex Nerve** (from fifth and sixth cervical roots).—It supplies the deltoid and teres minor muscles; also the skin over the deltoid and the articulation.

Etiology.—Injury, dislocation and contusion of the shoulder, compression (crutch), lead poisoning, grippe, diabetes, finally exposure to cold are the usual causes of palsy.

Symptoms.—The function of the deltoid muscle is to elevate the arm. Its paralysis abolishes almost all power of raising the arm. The atrophy of the muscle changes the shape of the shoulder. Anæsthesia is present over the skin covering the muscle. Adhesions may form in the joint and produce ankylosis. Reactions of degeneration appear quite early, except in cases of contusion.

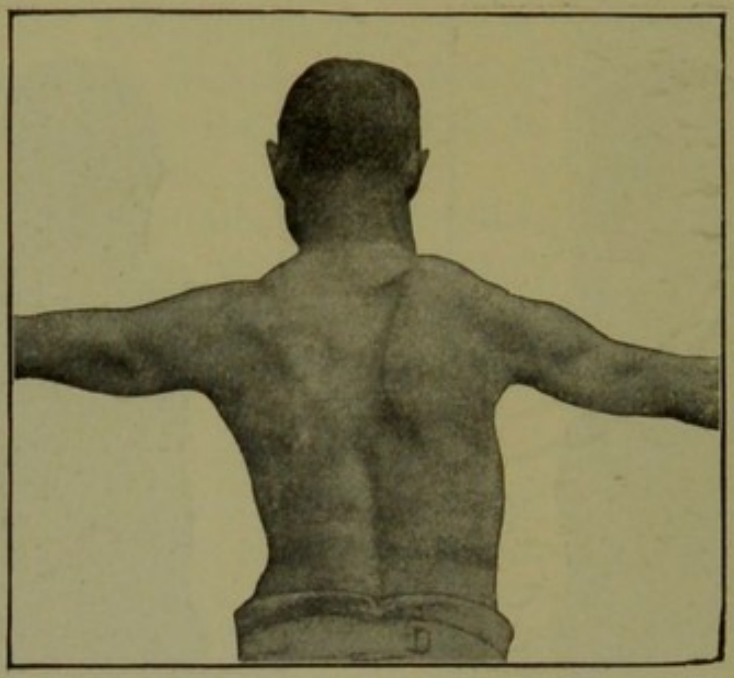


FIG. 135.—PARALYSIS OF SERRATUS MAGNUS. POSITION OF SCAPULA WHEN ARM IS ABDUCTED. FURTHER ELEVATION OF ARM IS IMPOSSIBLE. (*Starr.*)

(*d*) **Musculo-Cutaneous Nerve** (from fifth, sixth, also seventh roots).—It supplies the biceps and brachialis anticus, also the skin over the radial side of the forearm.

Etiology.—Trauma, pressure, dislocation or fracture of the head of the humerus are the usual causes of palsy.

Symptoms.—Paralysis of the flexors of the forearm is the chief symptom. It is particulary marked when the arm is supinated. Atrophy and RD. develop rapidly. There is also anæsthesia on the outer side of the forearm.

(*e*) **Musculo-Spiral Nerve** (from sixth, seventh and eighth roots).—It supplies the triceps, supinators and extensors of the hands and fingers; the skin of the lower half of the arm, of the back of the forearm, also of the dorsum of the thumb and of the first three fingers.

Etiology.—The superficial position of the nerve renders it very susceptible to injuries. **Trauma** is therefore the most frequent of all causes

of musculo-spiral paralysis. **Compression,** acute or chronic, is a common occurrence It develops during a profound sleep, especially in an intoxicated state, when the head rests on the arm a number of hours (Saturday night paralysis). It also occurs during a deep narcosis when the arm is held against a hard support. The superficial position of the nerve in the axilla is the cause of its palsy when the patient walks on crutches (**crutch palsy**). Violent muscular exercise, fracture of the humerus may also produce a musculo-spiral palsy. Infectious diseases, intoxications (alcohol, lead, arsenic) are predisposing causes. Potain mentions hypodermic injections made close to the nerve as an occasional cause of its paralysis.

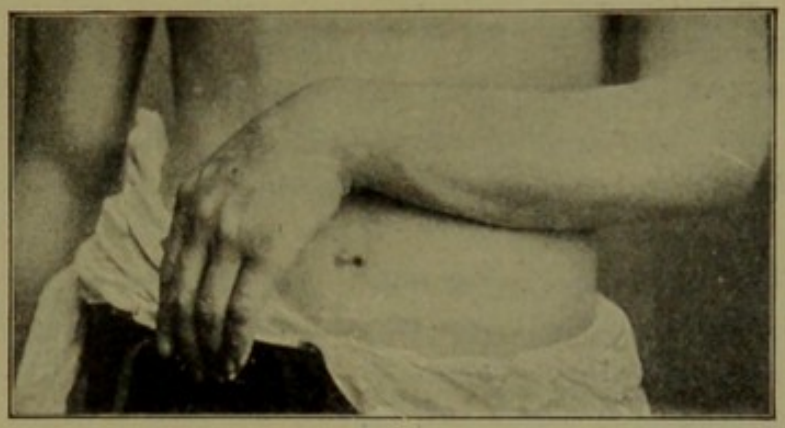


FIG. 136.—POSITION OF THE HAND IN MUSCULO-SPIRAL PALSY. (*Oppenheim.*)

Symptoms.—The onset is usually sudden It may be preceded by some paræsthetic disturbances (tingling, etc.). As the main function of the nerve is extension, its paralysis will place the hand in a special position. The latter is flexed, adducted and semi-pronated; the fingers are equally flexed and the thumb is adducted. There is a gradation of paralysis of extension in the fingers, most pronounced in the fourth finger and least marked in the first (Gowers). Extension of the wrist is impossible (**wrist-drop**). The forearm is also flexed and pronated.

The state of sensations depends upon the seat of the damage done to the nerve. In the majority of cases the injury occurs near the musculo-spiral groove; the sensibility is here only exceptionally disturbed. When the nerve is damaged above that level, **anæsthesia** will be present over the area supplied by the sensory filaments of the nerve (see above).

The paralyzed muscles gradually undergo atrophy and changes in electrical irritability. The latter is at first diminished, but later presents reactions of degeneration.

Course, Termination, Prognosis.—They depend to a large extent upon the cause. Crutch palsy improves rapidly when the crutches are

abandoned. The usual cases of pressure palsy improve and recover. When the nerve is severely injured by fracture, dislocation or contusion, recovery may also follow, but slowly. The prognosis is almost in direct relationship to the electrical condition of the muscles. Diminution of response has a favorable outlook, RD.—is unfavorable. When the nerve is completely severed, restoration of function will be possible only after a reunion of the stumps.

Treatment.—Removal of the cause in cases of compression is the first indication. As alcoholic individuals are particularly apt to suffer, they should be insisted upon abstaining from alcohol. When the nerve is completely severed, suturing of the two ends is necessary. Electricity and massage are the usual procedures for restoring the function of the paralyzed muscles. They should be instituted as early as possible. Mechanical apparatus have been recommended for the relief of flexion of the wrist and fingers; their object is to place the latter in extension.

(*f*) **Ulnar Nerve** (from eighth cervical and first dorsal roots).—It supplies the flexor carpi ulnaris, the inner portion of the flexor digitorum profundus, the interossei of the hand, the muscles of the little finger, the palmaris brevis, the last two lumbricales, hypothenar, the adductor pollicis. It gives off sensory fibers to the integument of the hypothenar to the front of the fifth finger and half of the fourth finger, to the back of the fifth, fourth and half of the third finger.

Etiology.—The superficial position of the nerve at the elbow and wrist predisposes it to injury. Pressure more or less prolonged, laceration of the nerve, fracture and dislocation at the level of the elbow, diseases of the olecranon or internal condyle of the humerus, forced flexion, occupations requiring a prolonged flexion, especially in alcoholic or cachectic individuals are all causes of paralysis of the ulnar nerve. Overextension of the arm during surgical operations is apt to produce palsy of the ulnar nerve. It is probably due to stretching of the nerve in the axilla. In sleep a similar phenomenon may be observed when the individual sleeps with arms elevated, abducted and behind the head. Such a case I placed on record (*Old Dominion Journal of Med. and Surg.*, 1909).

Symptoms.—Paralysis of the above enumerated muscles will be present. The most characteristic disturbance is the **paralysis of the interossei.** The normal function of the latter consists of flexion of the first and extension of the last two phalanges, also adduction and abduction of the fingers. When paralysis occurs, the fingers show an exaggerated extension of the first and flexion of the last two phalanges. This is the **claw**-like hand. The condition, however, is only partial; more pronounced for the fourth and fifth fingers than for the index and middle fingers, because the latter

have their lumbricales (median nerve) preserved. Adduction of the thumb, abduction and adduction of the fingers, lateral movements of the little finger are impossible. The hand is slightly deviated toward the radial side.

Sensory Disturbances are habitually present. Besides pain and hyperæsthesia there may be hypæsthesia or anæsthesia. The latter will be present over the internal surface of the hand (palm and dorsum), little finger

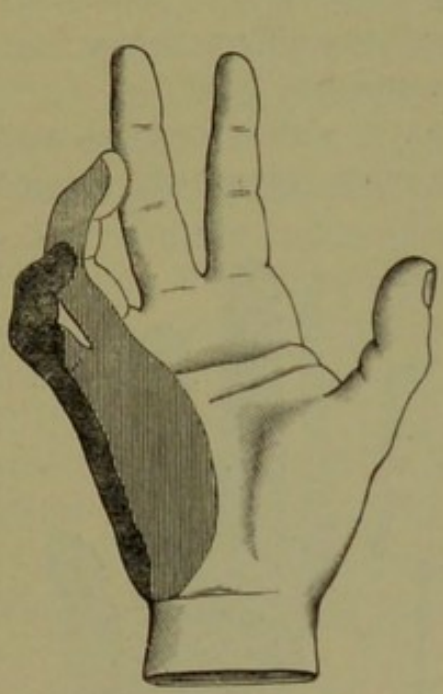


Fig. 137.

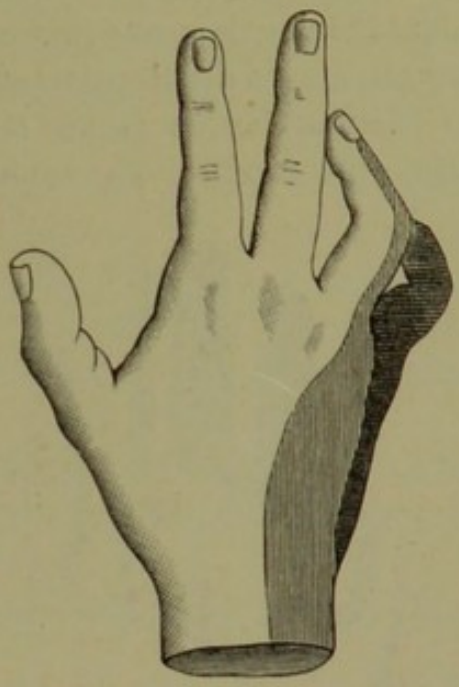


Fig. 138.

Figs. 137, 138.—Showing Sensory Loss and Abnormal Position in Injuries of Ulnar Nerve. (*Bowlby.*)

inner half of the fourth finger, internal and dorsal surface of the first phalanx of the third finger.

Atrophy and reactions of degeneration develop some time after the onset of the palsy.

Course, Prognosis.—In slight traumata the termination is favorable after a few weeks' duration. In lacerations or severance the prognosis is unfavorable as to recovery of function.

Treatment.—Same as in musculo-spiral nerve palsy.

R. Hunt described in 1908 an **occupation neuritis** due to an involvement of the deep palmar branch of the ulnar nerve. This nerve is purely motor and innervates the following intrinsic muscles of the hand: those forming the hypothenar eminence, also some of the thenar region, viz. abductor pollicis and inner head of the flexor brevis pollicis. It innervates as well the palmar and dorsal interossei and the two inner lumbricales.

The **symptoms** are: atrophic paralysis of all the intrinsic muscles of the

hand innervated by the ulnar nerve; reactions of degeneration; total absence of objective sensory disturbances in the ulnar nerve distribution.

The lesion, which is usually due to compression, is below the point where the sensory branch is given off and before the deep branch breaks up into muscular branches. Hunt observed the neuritis in a jeweler, machinist, brass polisher.

H. Gessler in 1896 (*Mediz. Corresp.-blatt., Würtemberg*, Bd. LXVI) described a type of muscular atrophy of the hand in gold polishers, which resembles in many respects that of Hunt with this difference that there was paræsthesia in the distribution of the ulnar nerve.

(g) **Median Nerve** (from sixth, seventh, eighth cervical and first thoracic).—It supplies the superficial anterior (except the flexor carpi

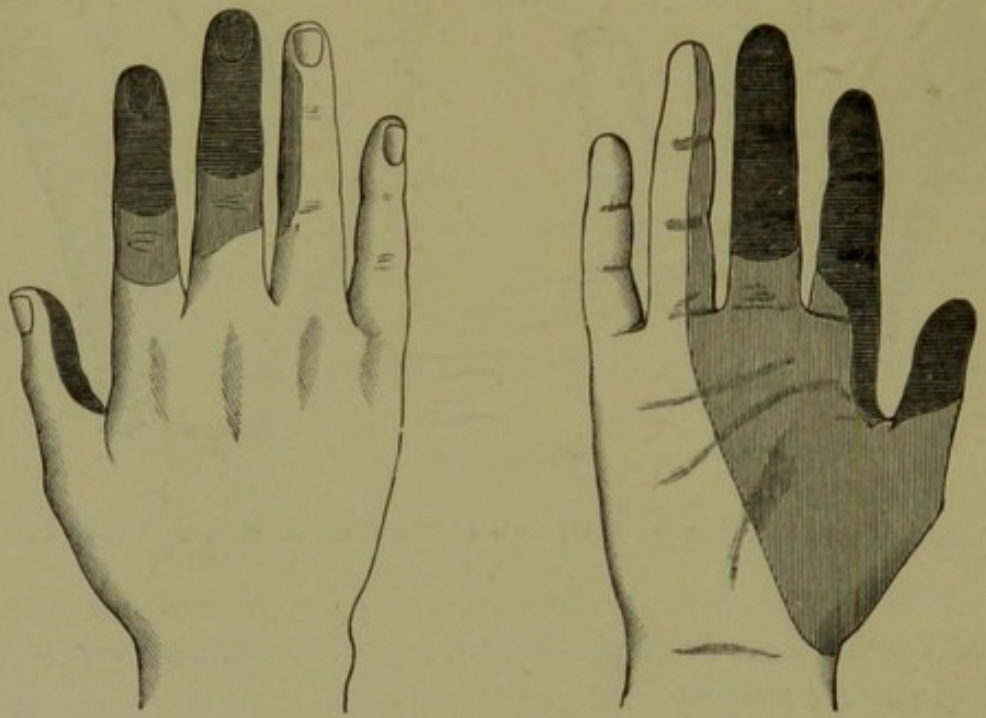


Fig. 139. Fig. 140.

Figs. 139, 140.—Showing Areas of Sensory Losses in Injuries of Median Nerve. (*Bowlby.*)

Horizontal lines, total anæsthesia; vertical lines, partial anæsthesia.

ulnaris) and the deep muscles of the forearm (except the inner half of the flexor digitorum communis). In the hand it supplies the opponens pollicis, the flexors of the thumb, the abductor brevis and the two outer lumbricales. It also gives off sensory branches to the radial part of the palm, inner part of the thenar and the palmar aspect of the first three fingers and the radial side of the fourth finger, also the dorsum of the last two phalanges of the thumb and first three fingers.

Etiology.—Trauma (blows, cuts, fracture) in the lower part of the forearm, compression (tumor, callus, Esmarch's bandage, etc.), violent muscular

efforts are the most frequent causes. Certain occupations (tinners, joiners cigarmakers, milkers) may sometimes involve the muscles innervated by the median nerve. Finally infectious diseases may occasionally cause a neuritis of the median nerve.

Symptoms.—Paralysis of the muscles enumerated above will give the hand a peculiar attitude. The position of the fingers is quite characteristic. The last two phalanges are held in forced extension by the interossei (ulnar nerve) more on the radial than on the ulnar side; the thumb is extended, approached to the index and its opposition is impossible. The wrist is in extension and slightly abducted. Pronation of the hand is impossible.

Sensory Disturbances are usually present. They may consist of pain, hyperæsthesia and especially anæsthesia. The latter extends over the two outer thirds of the palm, the palmar surface of the first three fingers and radial side of the fourth finger, the dorsal aspect of the last two phalanges of the thumbs, second and third fingers and radial side of the fourth finger.

Atrophy with reactions of degenerations appear early. Trophic and vasomotor disturbances of the skin are quite frequent (herpes, glossy skin, loss of nails, hyperhidrosis, cyanosis).

Course, Prognosis and **Treatment** are identical to those of the ulnar nerve.

C. Paralysis of Lumbo-sacral Nerves

Paralysis of the Nerves of the Lower Extremities

(*a*) **Anterior Crural Nerve** (from second, third and fourth lumbar roots).—It supplies the ileo-psoas, extensor cruris quadriceps, sartorius and partly the pectineus and the middle adductor muscles. Its cutaneous branches are distributed over the antero-internal surface of the thigh (lower two-thirds) and internal aspects of the leg and foot.

Etiology.—Palsy of the crural nerve is rare. Diseases of the vertebræ, pelvis and femur, dislocation of the hip-joint, psoas abscess, hernia, aneurism, trauma in the groin or thigh are the main causes. It may become paralyzed in infectious diseases (typhoid fever, diphtheria) also in alcoholism. Finally, in anterior poliomyelitis the muscles supplied by the anterior crural nerve are especially involved.

Symptoms.—The distribution of the nerve indicates that its paralysis affects especially the extensors of the knee and flexors of the thigh. Paralysis of these muscles will cause a loss of the knee-jerk.

Sensory Disturbances consist of hypæsthesia or anæsthesia over the

area of distribution of the cutaneous fibers (see above). Pain is frequent.

Atrophy and reactions of degeneration appear early.

(*b*) **Obturator Nerve** (from second, third and fourth lumbar roots).—It supplies the gracilis, adductor longus et brevis. Its sensory fibers are distributed over the inner side of the thigh and inner and upper third of the leg.

Etiology.—Obturator hernia, compression by tumors or in difficult labor are the usual causes.

Symptoms.—Adduction of the thigh and its outward rotation are affected. The patient is unable to cross and approach the thighs, also to turn the foot inward and outward. Loss of sensation will be present over the area of distribution of the cutaneous filaments.

Atrophy and reactions of degeneration develop early.

(*c*) **Paralysis of the Gluteal Nerves** (from the lumbar and sacral plexus).—They supply the gluteal muscles, the pyriformis and the tensor fasciæ latæ.

Etiology.—Diseases of sacrum and pelvis, fractures, tumors are the usual causes. The disease is rare.

Symptoms.—Paralysis of the above muscles produces loss of rotation, abduction, extension and flexion of the thigh. Station, gait and climbing are difficult.

Atrophy develops rapidly.

(*d*) **External Cutaneous Nerve** (from second and third lumbar nerves).

Symptoms.—As it supplies the outer aspect of the thigh, its paralysis causes a difficulty of walking and standing (see also Meralgia paræsthetica).

(*e*) **Paralysis of the Sciatic Nerve** (from Sacral Plexus).

Etiology.—Pressure by tumors of the pelvis or femur or fractures, by the head of the fœtus in difficult labor, by forceps in instrumental delivery, neuritis of toxic (alcohol, lead, arsenic) or infectious nature, septic processes of the pelvis—are all causes of total paralysis of the sciatic nerve. Complete palsy is rare. More frequently are met partial palsies of the sciatic nerve, viz. paralysis of the **peroneal nerves.** In the latter case infection and intoxications play a prominent etiological rôle. Puerperal neuritis has a special predilection for the external popliteal nerve. Fracture of the fibula, forcible extension, occupations requiring a stooping position are also causes of peroneal nerve palsy.

Symptoms.—In total paralysis of the sciatic nerve (which is exceptionally rare) the foot, the leg, also the flexors of the leg situated on the thigh are all completely powerless. The motility of the entire limb is abolished with the exception of extension of the leg, which is controlled by the crural nerve (see above). The leg is held rigid. The gait is possible to

a certain extent because of the integrity of the extensor muscles (crural nerve). Atrophy and anæsthesia are common. Sometimes trophic disturbances, such as herpes, etc., are present.

In paralysis of the **external popliteal nerve** the following muscles are involved:

(*a*) The peroneal, the function of which is to extend, abduct and rotate (externally) the foot; (*b*) tibialis anticus whose function is to flex, adduct and rotate (internally) the foot; (*c*) extensor communis digitorum which extends the first phalanges of the toes; (*d*) extensor of the great toe which extends its first phalanx.

When these muscles are paralyzed, the action of the posterior muscles predominates; dorsal flexion of the foot is impossible. The latter is in a state of **foot-drop** and of **equino-varus.** The gait is of **steppage** character (see Multiple Neuritis). In an advanced stage contractures place the foot and its constituents in a fixed abnormal position. **Sensations** are usually diminished. There may also be total **anæsthesia.** The latter is distributed over the antero-external surface of the leg and the dorsum of the foot and toes. Trophic and vaso-motor disturbances, atrophy of the muscles, changes in the electrical reactions are the same as in any other nerve palsy.

Special mention must be made of paralysis of the external popliteal nerve occurring during **protracted labor.** This nerve originates from the lumbo-sacral trunk which is formed by the union of the smallest part of the fourth and the entire fifth lumbar nerves. The trunk at its formation is situated on the ala of the sacrum under cover of the psoas. It descends into the pelvis and passes over the brim of the true pelvis. It stands to reason that a protracted labor with head pressing unduly against the brim of the pelvis, or else forceps, are apt to injure the exposed lumbo-sacral cord, and therefore, the peroneal nerve. Besides this grave form of peroneal neuritis there is also a slight form of neuritis occurring in puerperium and which is usually transient. It is probably tonic in nature. In both forms there is pain on pressure, paretic state of the muscles, spontaneous pain, objective sensory disturbances and sometimes œdema with cyanosis of the limb.

The **diagnosis** should be made between neuritis of the peroneal nerve and puerperal phlebitis. Failure of recognizing the true condition may lead to very grave errors of treatment. When for example a neuritis is taken for phlebitis, it means a strict and prolonged immobilization of the limb. This will lead eventually to muscular atrophy, loss of power in the limb, trophic disturbances, otherwise speaking to a protracted infirmity. When on the other hand a phlebitis is taken for a neuritis, it means

mobilization of the limb, electrization and massage of the muscles. In both cases the respective procedures are dangerous. A very careful examination for characteristic symptoms of each of these affections will enable one to avoid errors.

When the **internal popliteal nerve** is involved the following muscles muscles are paralyzed: (*a*) Triceps suræ (which normally produces plantar flexion); (*b*) tibialis posticus, which is an extensor, adductor and rotator (internally) of the foot; (*c*) flexors of the toes, viz. of their first phalanges; (*b*) interossei (which normally flex the first phalanges and extend the others).

Paralysis of these muscles leads to dorsal extension of the first phalanges of the toes and plantar flexion of the last two phalanges (**claw-like**), to loss of plantar flexion. Rising on the tips of the toes is impossible and walking is difficult.

Sensory Disturbances (hypæsthesia or anæsthesia) are present on the plantar surface of the foot and on the postero-internal surface of the leg.

RADICULITIS.

Under this term is understood a primary inflammation of the roots of nerves. Secondarily the nerve roots may be involved in lesions of peripheral nerves or of the spinal cord (poliomyelitis, syringomyelia, etc.) and in traumatic injuries. But radiculitis means a primary infection or toxic inflammation of the roots and their coverings.

Symptoms.—The **onset** may be **rapid** or **slow.** The first is rare; it may occur in the course of cerebro-spinal or spinal meningitis. The second, which is the usual onset, consists of slowly but progressively developing manifestations. As a type the most frequent form of radiculitis will be described here, viz. **cervico-brachial radiculitis** of tertiary syphilis. Gradually and without apparent cause various paræsthesias and pain develop in the cervical region. Pain is continuous but is interrupted by paroxysms of a violent character. The latter are brought on by an effort, cough or sneezing. The exacerbations make the patient scream and Dejerine with others described this phenomenon "sign of sneezing" as a reliable symptom of the disease. The pain radiates along the course of the nerve-trunks. Pressure provokes pain. It can be elicited at the level of the sixth transverse process (Erb's point). Gradually the nervous filaments undergo degeneration and destruction and the hyperæsthesia is replaced by **anæsthesia;** pain then disappears. Not only the skin but also muscles, bones and joints have lost their sensibility. Perception of position and stereognostic sense are abolished. For this reason ataxia

becomes evident. Whether the loss of sensation is complete or incomplete, it is always **radicular** in distribution and the latter is characteristic of the disease.

When the lesion affects also the motor roots as well as the sensory there will be present also motor and trophic symptoms in addition to sensory. Paresis or paralysis, atrophy of the muscles with reactions of degeneration gradually make their appearance. When atrophy affects only a certain group of muscles, the antagonistic muscles through their overaction will produce deformities of the limbs. The tendon reflexes are at first diminished, but later disappear. Trophic disturbances are not infrequent. Herpes zoster may accompany a radiculitis of the sensory roots. A very important diagnostic sign we find in the cerebro-spinal fluid. As the usual etiological factor is syphilis (also tuberculosis), a more or less marked **lymphocytosis** is found.

Similar to neuritis and neuralgia there are also radiculitis and **radiculalgia.** In the latter the lesion consists only of **irritation**, but not of destruction. The pain is very severe, paroxysmal in type and hyperæsthesia of radicular distribution is present. Chipault and Lefur described a neuralgia of the eighth, ninth, and tenth dorsal roots which manifested itself as a tic douloureux of the abdomen. Neuralgia of the roots may be the initial stage of radiculitis.

Forms of Radiculitis

Cervico-brachial.—It was described above as a type. If the eighth cervical and first dorsal are involved, oculo-pupillary symptoms will be also observed, viz. myosis, retraction of the eye globe and narrowness of the palpebral fissure.

Dorsal.—Vaso-motor disturbances, such as herpes zoster, will be observed. Such was the case of Dejerine and Thomas in which the eruption was strictly limited to the area of the right eighth dorsal root. Oppenheim observed paralysis of the abdominal muscles and loss of their reflexes, meteorism, pain and sensory disturbances in lesions of the eighth and ninth dorsal roots.

Lumbar.—It is quite frequent. It presents the picture of neuritis of the anterior crural nerve (see this chapter).

Lumbo-sacral.—Its requisition is very important. It constitutes the so-called **"radicular sciatica."** The differential diagnosis between it and the classical sciatica is as follows: in the latter the objective sensory disturbances (hyperæsthesia or anæsthesia) are distributed irregularly. In the former the sensory disturbance will be present in a longitudinal

form regularly distributed and running parallel with the axis of the limb. Besides, the pain is spontaneous and brought on not exclusively on motion, also it is very persistent and present in regions others than the ischio-sacral notch (see my monograph in *J. Amer. Med. Ass'n*, 1910).

Sacro-coccygeal.—The radiculitis of the cauda-equina is accompanied by disturbances of the function of the sphincter and genitalia.

Etiology.—Any cause that is capable to produce an inflammation or degeneration of the roots in their intra- or extra-dural course can be considered as an etiological factor. Tumors, hemorrhages, cold abscess, aneurism—are all apt to bring on a mechanical irritation of the tissue surrounding the root or else to disturb the circulation and nutrition of the latter. Tuberculosis (in Pott's disease) and cancer (of the vertebræ) are almost always the causes of radiculitis. Hypertension of the cerebro-spinal fluid in tumors of brain and in tabes have also been considered as causes. More important etiological factor is an infectious process. Here an inflammation of the meninges occurs first and of the root subsequently. After syphilis, tuberculosis is the most important factor (pachymeningitis, leptomeningitis). Toxic (lead) and infectious agents have a special predilection for nerve-roots.

Prognosis is quite serious. In syphilitic cases it is most favorable.

Treatment.—It depends on the cause. Operations (laminectomy, resection of posterior roots) are advisable only as a last resort. (For more details consult the thesis of Paris by Camus, 1908.)

NEURALGIA (IN GENERAL)

Definition and Nature.—Under this term is understood a paroxysmal pain over nerve trunks and their branches. Neuralgia is not a morbid entity, but only a symptom, as it may accompany any affection of the nerves. Pain in the nerve may be also present when no lesion of the latter is evident. A sharp distinction between neuralgia and neuritis cannot be established. The fact is that in a number of cases of recent neuralgia degenerative changes have been found in the nerves. From my personal pathological studies (*New York Med. Jour.*, July 21, 1906) it can be seen that the occurrence of degeneration of the peripheral nerves is frequent in neuralgia, that neuralgia is probably a primary neuritis and that changes in the walls of the blood vessels play a certain rôle in the causation of the degenerative condition of the nerves.

Etiology.—From the foregoing remarks it is evident that the causes of neuritis (see this chapter) will be also those of neuralgia.

Besides neuritis, neuralgia may be due to **exhaustion** caused by de-

bilitating and protracted diseases, such as cancer, anæmia, to **infectious** diseases (grippe, typhoid fever, malaria), to **syphilis,** to intoxications (lead, arsenic, etc.). Exposure to **cold** is a frequent factor. **Overwork** and **great excitement,** errors of ocular refraction, often provoke neuralgic pains. In the so-called **reflex-neuralgias** irritation of remote organs (uterus, testicles, etc.) produces a neuralgia of the face or elsewhere.

Among the **predisposing causes** should be mentioned the neuroses (hysteria, neurasthenia), gout, rheumatism, diabetes, tuberculosis.

Symptoms.—The chief symptom is **pain.** Its seat is along the nerve. Generally it is manifested in violent paroxysmal attacks between which the patient is free from pain, but in some cases a slight pain persists in

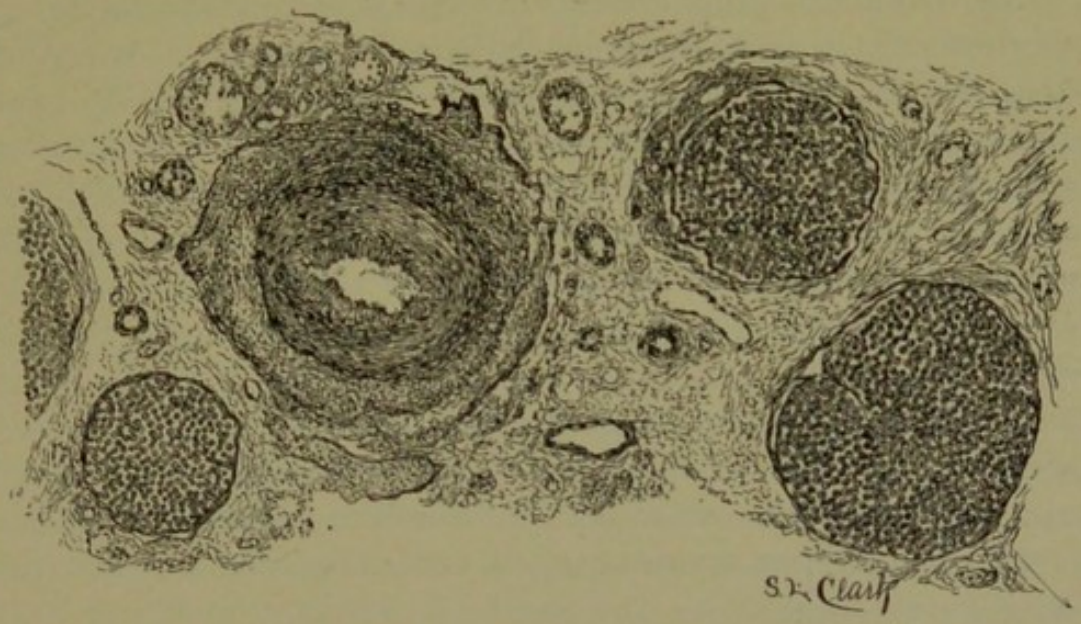


FIG. 141.—NEURALGIA OF THREE MONTHS' DURATION. DEGENERATED NERVE-BUNDLES IN THE IMMEDIATE VICINITY OF THICKENED BLOOD VESSEL. (*Original.*)

the intervals. The paroxysms may last from a few seconds to several minutes; they may recur several times in one hour. The pain may be tolerable or extremely violent, be confined to one or several spots on the skin or else spread from an initial point to several ramifications of the nerve. It appears spontaneously or brought on by the least touch, motion, cold air, mastication, etc.

A characteristic symptom of neuralgia is found in the **tender spots.** They are very small areas situated over the course of the nerve and its branches; they are extremely tender upon slight pressure.

In addition to these two constant symptoms there are others that occur more or less frequently. They are sensory, motor, vaso-motor, trophic and secretory.

Sensory.—They may be hyperæsthesia or anæsthesia over the area of distribution of the affected nerve.

Motor.—Twitching during the paroxysms (in tic douloureux) or else spasmodic contractions (painful cramps) are sometimes observed.

Secretory.—They are ptyalism, hyperhidrosis, watering of the eyes.

Vaso-motor.—Pallor or redness of the skin is sometimes observed during the paroxysms; angioneurotic œdema has been seen in association with neuralgic paroxysms.

Trophic Disturbances accompanying neuralgia are: œdema and herpes.

Neuralgia of long duration may affect the general health. The patient is depressed, irritable, loses his appetite, cannot sleep and finally begins to lose in weight.

Course, Duration, Prognosis.—Neuralgia is usually of long duration. The paroxysms may occur at regular or irregular intervals. It may after a certain time disappear completely or else become chronic. In cases of **malarial** origin the neuralgia presents a special feature: it may substitute a malarial attack or accompany it, viz. occur at certain intervals. Malarial neuralgia affects mainly the fifth and the sciatic nerves. In grippe the supra-orbital nerve is most frequently affected.

Neuralgia of diabetic origin has a special predilection for the sciatic nerve.

The course and prognosis depend upon the cause. Neuralgia caused by neuritis is serious, because it recurs easily and readily becomes chronic. Malarial and syphilitic neuralgias are usually amenable to treatment. The outlook is bad when there is a constitutional unfavorable basis (tuberculosis, diabetes, etc.).

Diagnosis.—Pain along the course of the nerve, its tendency to radiation, its remittence or intermittence, finally the tender points are all sufficiently characteristic symptoms for making a diagnosis of neuralgia.

Sharp pain may occur also in **tabes** (tabetic crises), but in the latter it does not follow the course of a nerve trunk and the tender points are absent. It is present in tumors located in the vicinity of the spinal roots.

In **neuritis** pain is usually present, but it is continuous, while in neuralgia it is paroxysmal. Neuritis will be also recognized by motor, sensory and trophic disturbances, which are absent in neuralgia.

Treatment.—(1) Relief from pain and (2) removal of the cause of neuralgia are the chief indications. The pain can be relieved by sedatives. Morphine will give almost immediate relief, but its repeated administration is to be feared in view of the habit which is easily acquired. Coal-tar products (aspirin, sodium salicylate, phenacetin, etc.) without or with small doses of codein has given me satisfactory results. Local counter-

irritation (cantharides) has given me excellent results in a number of cases where a host of internal medications have failed. Malarial and grippal cases need an energetic treatment with quinine. Syphilic cases require mercury and iodides or salvarsan. In very obstinate cases, even without a syphilitic history, the specific treatment should be tried.

Good results have been obtained by me from **Strychnia** in gradually increasing doses. In one case of obstinate cervical neuralgia in a middle-aged woman, when all possible medications had been tried during a period of two years and operative procedures had been almost decided upon, strychnia was given as a last trial and curiously enough the pain disappeared completely.

Thyroid extract has also given me very satisfactory results in four cases when everything else failed.

Electricity has given some relief to a very limited number of my patients. Galvanism and sometimes faradism as well as static electricity had been used by me in those cases. Care should be taken to apply weak currents (of 12–15 milliamperes) to the affected nerves, especially in facial neuralgia.

The general nutrition, a constitutional diathesis, a neuropathic state, must be taken special care of. Appropriate hygiene and diet, proper and regular elimination (bowels, kidney), hydrotherapy, massage, regular mode of living, avoidance of stimulants (alcoholic beverages), reduction of obesity if it exists, administration of arsenic and iron in cases of anæmia, a special diet in diabetes, exercises for those who lead a sedentary life and on the contrary much rest for those whose occupation is exhausting—all these points should invariably be taken into consideration in treating a case of neuralgia.

Injections of alcohol into the nerve trunks in facial neuralgia at the points of their exit in the bony foramina has proven to be a very useful procedure. Five minims of 80 per cent. of alcohol should be injected without a preliminary local anæsthetic. The injection can be repeated every few days. In the majority of cases the pain disappears immediately after the injection. Burning and swelling of the surrounding tissue appear rapidly and persist for several days, but disappear without leaving any trace. For supraorbital neuralgia injections are made in the supraorbital notch, which is easily felt. For the second branch, alcohol is injected into the infraorbital foramen. For the third branch of the fifth nerve injections are to be made into the mental foramen of the lower jaw. I have obtained in this manner considerable relief in a number of instances. I have also observed that when several injections into the infraorbital nerve were not successful, subsequent injections into the supraorbital

nerve gave relief for weeks. Should the alcoholic injections made at the exit of the sensory nerves on the face fail, deep injections are to be carried out. It must be mentioned immediately that the **first branch** of the trigeminus should not be treated with deep injections, as the second, third, fourth and sixth cranial nerves, also large blood vessels, are in immediate vicinity of the ophthalmic branch and the risk of their injury is very great. Deep injections into the **second branch** are made with a needle inserted at the lower border of the zygoma; 0.5 cm. behind a perpendicular running from the posterior edge of the orbital process of the malar bone it is diverted upward; at a depth of 5 cm. the needle reaches the nerve as it leaves the foramen rotundum to enter the sphenomaxillary fossa (Lévy, Baudouin, Patrick). For the **third branch** of the trigeminus the needle is inserted also at the lower border of the zygoma but 2.5 cm. in front of the anterior root of the zygoma; it is directed backward and upward. At a depth of 4 cm. it reaches the nerve. The directions just given are not absolute, as the anatomical interrelations between foramina and bony structures through which the nerves pass vary considerably in some individuals. Considerable experience is necessary on cadavers before an attempt can be made to perform a deep injection on patients. Superficial and deep injections do not always cure the sufferer, but they may give enormous relief for a very long time. In case alcoholic injections after several attempts fail, a radical operation for removal of the Gasserian ganglion or for cutting off only the sensory roots may be resorted to. The radical operation, unfortunately, does not remove pain in every case, besides being dangerous. For more details on the technic of deep injections of alcohol consult Lévy and Baudouin (*Presse Médicale*, 1906), also Patrick (*J. Am. Med. Ass'n.*, 1912).

While alcoholic injections are an excellent procedure for relieving nerve pain, nevertheless it must be borne in mind that grave damage may be done to the nerve trunk when the latter is placed in intimate contact with alcohol. I have recently shown it in an experimental study (Transaction of Amer. Neurol. Ass'n., 1913).

When all the means enumerated above fail to give permanent results, surgical intervention must be resorted to. The latter is indicated not only in cases of compression of a nerve by a tumor and abscess or of any other pathological condition in the vicinity of the nerve, but also in all cases of neuralgia which ordinary therapeutics, including the injections of alcohol, fail to relieve. The operative procedures consist of **nerve-stretching, neurectomy** and **neurorhexis.** In the first the results are frequently only transitory. The second consists of a resection of a certain portion of the

affected nerve. The first two methods fail to give permanent results. The third method is based upon the pathological fact that a violent tearing out of a nerve produces permanent cellular changes in its nucleus (Van Gehuchten). This procedure has given by far better results than any other.

NEURALGIA OF INDIVIDUAL NERVES

A. Neuralgia of the Fifth Nerve (Facial Neuralgia). Etiology.—All the causes mentioned in the chapter on neuralgia in general are also factors in facial neuralgia. Special stress must be laid upon **cold** and **malaria,** which are frequent causes.

Lesions in the area of distribution of the fifth nerve, viz. diseases of the alveoli, of the ear, of the cavities of the face (nose, frontal sinus), of the eye; diseases of the bones of the face through which the nerve passes, traumatism, inflammation, are all local causes of facial neuralgia.

The disease is very frequent and is observed usually in the middle age. It is more frequent in females than in males.

Pathology.—The nerve had been frequently found in an inflammatory state or in a state of degeneration. Atrophy of the cells of the Gasserian ganglion have been also observed in a few cases. Endarteritis has also been found.

Symptoms.—**Pain** is the chief and sometimes the only symptom of the disease. It is agonizing in character. Its onset may be preceded by some paræsthetic disturbances. Among the three branches of the nerve the first (ophthalmic) is most frequently affected.

The **tender points** characteristic of neuralgia in general (see above) are: for the ophthalmic branch—**supraorbital** (at the level of supraorbital foramen); **nasal** (upper part of the nose); for the supramaxillary branch—**infraorbital** (below the lower eyelid); **malar;** for the inferior maxillary branch—**temporal** (in front of the ear); **labial** (on the lower lip); **mental** (place of exit of the mental nerve on the chin).

The nature of the pain (tearing, boring), its paroxysmal character, the tendency to radiation over the branch affected and even beyond its boundaries, repetition of the attacks at regular or irregular intervals and their duration (a few seconds), the development of the pain from the least influence (cough, touch, mastication, change of temperature of the food, etc.), are described in the chapter on neuralgia in general.

The **less constant symptoms** of facial neuralgia are: secretory, vasomotor, trophic, sensory and motor.

Secretory.—During an attack there may be an increased nasal secretion, also watering of the eye and salivation.

Vasomotor.—The conjunctiva on the affected side is congested, the eyelids may be œdematous and the face flushed.

Trophic Disturbances consist chiefly of **herpes**, which is not infrequent in neuralgia of the first branch (**ophthalmic herpes zoster**). Falling of the hair and hemiatrophy of the face are occasionally observed.

Sensory disorder in the form of hyperæsthesia is frequent at the beginning, but **anæsthesia** is observed in older cases. Photophobia is frequent. Hearing and taste are sometimes affected.

Motor phenomena are not rare. They consist of convulsive movements of the muscles of the affected side of the face accompanying the paroxysms of pain. This is the so-called **"Tic douloureux."** It is the most obstinate form of facial neuralgia.

Course, Termination, Prognosis.—In the majority of cases neuralgia increases in intensity at first, then gradually decreases. It runs an indefinite course. It may disappear completely, but usually persists. Recurrences are frequent. The prognosis is serious.

Diagnosis.—See this chapter on neuralgia in general.

Treatment.—It is the same as of neuralgia in general (see above). The surgical treatment consists of stretching the nerve, section or excision of a portion of it or tearing out the nerve (neurorhexis) as near as possible the bony foramen from which it emerges. The latter procedure gave better results than the former. The reason of it lies in this observation (Van Gehuchten and others) that the cells of Gasserian ganglion corresponding to the torn-off nerve degenerate more readily and permanently. Finally removal of the Gasserian ganglion may be resorted to in protracted cases in which the above means have failed.

In treating a case of facial neuralgia it should be borne in mind that a diseased condition of the nose, ear, eye, teeth, of the sinuses may be the immediate cause of the pain. An examination of these organs is indispensable and a proper treatment is to be applied if indicated. In elderly individuals I have obtained very satisfactory results from nitroglycerine given either by the mouth or hypodermically.

Favorable results in treatment of tic douloureux have been obtained from **injections of alcohol** into the nerve trunks as they appear on the face, or into their roots near the foramina from which they emerge. Schlösser, Lévy, Baudouin, Ostwald, Brissaud, Patrick, Hecht and myself have published encouraging reports. For details see page 433.

B. Cervico-occipital Neuralgia.—Under this name is understood a neuralgia of the upper four cervical nerves. In the majority of cases it is the **major occipital nerve** that is involved, but the minor occipital, great auricular, superficial cervical and superclavicular nerves may also be affected.

Etiology.—Cold, trauma, aneurism of the vertebral artery, infectious diseases (grippe, typhoid fever and others), syphilis, lead poisoning, auto-intoxication, gout, diabetes, anæmia are the causes of occipital neuralgia. It is also observed in cervical Potts' disease, cervical adenitis, hypertrophic cervical pachymeningitis, localized tumors of the neck or of the cervical vertebræ.

Symptoms.—The classical symptoms of neuralgia in general are present. The pain is unusually severe. It is continuous with paroxysms of exacerbation. The slightest movement, cough, sneezing, cause an attack. The patient complains of a sensation of burning, tearing, stabbing in the neck.

The **tender points** (of Valleix) are present. The most important and constant is situated midway between the mastoid process and the first cervical vertebræ, at the level of emergency of the major occipital nerve. Other points: posterior border of the sterno-mastoid muscle; mastoid process. In severe cases there is also hyperæsthesia of the skin of the neck, tenderness between the scapulæ and in the supraclavicular fossæ, loss of hair in the same region, myosis on the affected side.

Prognosis.—It is usually good, but the disease is apt to recur. In one case under my observation it recurred six times in four years. The duration is uncertain. It may last several weeks or months. In the above case the last attack lasted three and a half months.

Diagnosis.—The characteristic tender spots, the radiation of the pain along the nerve are typical enough to make a diagnosis.

Treatment.—Cauterization, blistering, application of iodine, blood-letting all over the painful area, will be of benefit. In the case mentioned above the local treatment was of no avail. Relief and subsequent recovery followed administration of strychnia in ascending doses. For details see Treatment of Neuralgia in general.

C. Brachial Neuralgia.—This is a neuralgia of the brachial plexus.

Etiology.—Traumatism of the brachial plexus, fractures and dislocations of the humerus, injury of a peripheral nerve, as a burn, compression, bite of the finger, etc. (**reflex neuralgia**), infectious diseases (grippe, malaria, typhoid), diabetes, gout, anæmia, neuræsthenia, are all causes of this affection.

Symptoms.—Usually the pain affects one or two nerves of the entire plexus. The **ulnar** nerve is most frequently involved. The pain follows the course of the nerve and is characterized by paroxysmal attacks between which the patient is free from pain. The position of the limb is characteristic. The patient holds it with the unaffected hand as if the arm were fractured.

The **tender spots** are found on the nerves of the plexus: for the musculo-spiral nerve the groove of this name on the arm; for the circumplex nerve the deltoid area; for the ulnar nerve the elbow; for the median nerve the wrist.

Paræsthesia, hyperæsthesia or anæsthesia of the skin, hyperhidrosis, are frequently present.

Prognosis is the same as in neuralgia in general but it depends greatly on the cause.

Diagnosis.—The radiation of the pain along the nerve-trunks and the tender points are characteristic enough to recognize the disease. In compression of the cervical spinal cord pain may be present in the arms, but there are always other symptoms, as paralysis, atrophy, anæsthesia. In arthritis, in rheumatism, the pain does not follow the nerve-trunks.

Treatment.—It is the same as in other forms of neuralgia. When local treatment and internal medications are of no avail, an operation must be resorted to.

D. Intercostal Neuralgia.

Etiology.—The causes of this affection are: cold, contusion of the nerves, fracture or diseases of the ribs, compression from within or without, diseases of the lungs or pleuræ, an inflammation of which is transmitted to the nerves. As predisposing factors may be mentioned cachexia, anæmia, protracted diseases, syphilis, gout, chronic rheumatism. It is more frequently met with in women than in men.

Symptoms.—The pain is usually unilateral and involves several intercostal nerves. It may be spontaneous and then is usually **deep seated.** Similar to neuralgia in general, it presents paroxysms which are extremely severe. As the movements of the thorax provoke pain, the patient holds his chest immobile, avoids deep respiration and speaks with a low voice. The pain frequently radiates to the back and to the arm.

The **tender spots** characteristic of neuralgia are here three in number: anterior, lateral and posterior. The first is at **sternocostal** articulation, the second on the **axillary** line, the third near the **spinal processes.** The skin on the affected side is hyperæsthetic. Herpes quite frequently accompanies or follows intercostal neuralgia.

Prognosis.—It depends upon the cause. The disease is usually persistent.

Diagnosis.—The above-mentioned character of pain, its radiation along the nerves, the tender points, are all typical enough for the diagnosis. **Pleurodynia** is recognized by a diffuse pain. In **mastodynia** the pain is in the breast. **Tumors of the spine** or spinal cord may present neuralgic

pain as the only symptom for a long time before pressure symptoms make their appearance.

Treatment.—The first indication is removal of the cause. General dyscrasic condition, compression, fractures, etc., must be taken care of. For relief of pain counter-irritation, cauterization and local applications of revulsive measures are of great benefit. Obstinate cases should be treated surgically (stretching, resection, tearing out of the nerves). For further details see Treatment of Neuralgia in general. For Franke's operation see page 254.

E. Sciatic Neuralgia. Sciatica.

Etiology.—The causes are **local** and **general.**

Local.—Cold; trauma (contusion, injury of the nerve by a bony fragment); prolonged pressure against a hard object while sitting; compression in the pelvis by tumors and abscesses or by the head of the fœtus during a protracted labor; inflammation of the meninges extending to the roots of the sacral plexus and then to the sciatic nerve.

General.—Gout, chronic rheumatism, malaria, diabetes, intoxications (lead, carbonic acid gas, alcohol), infectious diseases (grippe, typhoid fever, puerperal infection), syphilis.

Certain occupations predispose to sciatica. It has been observed quite frequently in tailors and dressmakers.

Sciatica is a common affection. It is met more frequently in men than in women and particularly between thirty and fifty years of age.

Symptoms.—They are **sensory, motor** and **trophic,** as the nerve is a mixed nerve.

The first and chief symptom is **pain.** It is localized on the posterior aspect of the limb. It is continuous and paroxysmal. The pain may present various forms and degrees. In some cases it is tearing, burning; in others it is pulling. The paroxysmal exacerbations may be of an unusual severity. The pain may affect the entire nerve or only segments of it. It may radiate also during a paroxysm to the lumbar region, perineum and genitalia.

The pain is usually worse in walking or standing. It is increased when the leg is extended. Exposure to cold aggravates the condition. Atmospheric changes also increase the pain.

The **tender points** characteristic of neuralgia are: (1) In the groove between the trochanter and the tuber ischii, (2) in the middle of the popliteal space, (3) below the head of the fibula, (4) behind the external malleolus, (5) dorsum of the foot, (6) Gara (*Deut. mediz. Wochenschr.*, 1911) has recently called attention to an **abdominal tender spot** in sciatica which he found constantly present in 118 out of 124 cases. It is elicited by press-

ing the finger into the abdomen over the spinous process of the last lumbar vertebra on the affected side. He locates the point at a finger-breadth below the umbilicus and two fingerbreadths to the side of the median line. The pain from compression radiates down the sciatic nerve.

The skin over the posterior aspect of the limb is usually intact, but sometimes a diminution of sensations is present. In grave cases of sciatica complete anæsthesia has been observed. Paræsthesiæ, as numbness, coldness, tingling, etc., are frequently present.

Motor Disturbances consist of some loss of power, fibrillary contractions, cramps in the musculature of the leg. Gradually, if there is no improvement, the weakness increases. The patient assumes certain fixed **attitudes.** In order to avoid extension of the nerve the patient places the leg in flexion, the trunk naturally bends toward the sound side; a scoliosis with the convexity to the affected side is therefore formed. Eventually this **sciatic scoliosis** becomes permanent. Sometimes the curvature of the spine is in the opposite direction, viz. the concavity is toward the diseased side.

The patellar tendon reflexes are usually increased on the diseased side, although decreased and normal reflexes have been observed. The Achilles-tendon reflex is usually lost.

The **trophic disturbances** show wasting of the muscles. There may be a simple flabbiness of the muscles or a genuine atrophy with qualitative electrical alterations (RD.). In such cases the changes of the nerve are profound. The skin of the limb is bluish, its secretions are diminished, eruptions (herpes, acne, erythema) may develop.

Course, Termination, Prognosis.—In the **mild** forms the pain is only paroxysmal. Recovery follows in a few weeks. In the **severe** forms the pain is continuous, persistent and lasts months or years. In such cases deformities develop and the patient never recovers. Between these two extremes there are many intermediary forms. The prognosis is particularly unfavorable, when atrophy with reactions of degeneration is present.

The chronic form is less hopeful than the acute.

Double sciatica has an unfavorable prognosis, as it is usually the consequence of diseases of the spine or of the spinal meninges. It may also occur in diabetes.

Diagnosis.—The exact seat of the pain on the posterior aspect of the leg and the characteristic tender points are usually sufficient for making a diagnosis. There is another important diagnostic test which is decidedly pathognomonic. It consists of overextending the leg of the patient when he is in a sitting or lying position; this manipulation brings on pain. It is the so-called **"sciatic phenomenon"** of Lasègue.

In some affections of the lower limbs pain may simulate sciatica, but a careful examination will reveal the true condition.

In **muscular rheumatism** the pain is diffuse and the tender points are absent.

In **arthritis** of the sacro-iliac or hip-joints pressure from outside causes pain only at the level of the articulation.

In **sacro-coxalgia** Laségue's sign is also present. The following differential phenomena, according to Gueit (*Gas. des hôp.*, 1910) may be of use: when the patient lying flat on his back flexes his legs and an attempt is made to continue flexion of the thighs, a sharp pain in the upper and inner portion of the buttock will be felt in sacrocoxalgia, but not in sciatica. In the latter the preëxisting pain is not augmented.

In **Meningo-myelitis** or **tumors of Cauda equina** there is in addition to the pain in the limb also involvement of the sphincters. Besides, the symptoms are generally bilateral.

An examination of the pelvic organs should be made in cases of pressure-sciatica, as a local unsuspected lesion may be the direct cause of the affection (tumor, adhesions, etc.). For **radicular sciatica** see page 429. In all such cases an X-ray examination must be made.

Treatment.—In cases of compression by a tumor or other elements removal of the cause is the first indication.

The sciatica itself can be relieved first of all by rest. It should be a rule to put every patient suffering from this disease to bed in order to secure absolute rest for the limb. When the patient is restless, the limb should be immobilized by means of a board and a bandage. Otherwise speaking, it should be treated as if fractured. Every four or five days the bandage will be taken off for an hour or so and a gentle massage given to the leg. In the majority of cases absolute immobilization gives relief. Amelioration of pain can be also obtained from cauterization, counter-irritation or any revulsion over the course of the sciatic nerve, from blood-letting (leeches or scarification), from local spraying of chloride of methyl, and especially from hot baths. The latter can be given once or twice a day. The patient sits in the tub fifteen or thirty minutes. In very obstinate cases I obtained considerable relief from prolonged baths—even two hours at a time. Galvanism, the faradic brush and static electricity may sometimes be useful, but as a rule these means cannot be relied upon for relieving pain.

Internally the following drugs can be given: salicylates, any coal-tar product, iodides and bromides. Aspirin and salophen are superior to salicylates and in combination with small doses of codein have proven to be very useful in my hands. Very satisfactory results I have also obtained

in some cases from strychnia in gradually increasing doses. In cases with a history of syphilis or malaria, mercury and quinine respectively will be useful.

As sciatica frequently occurs in gouty and rheumatic individuals, autointoxication probably plays an important rôle. In such cases avoidance of nitrogenous food (meats) is advisable. In some of my cases a return to a meat diet has decidedly aggravated the sciatica. Alcoholic beverages are absolutely forbidden, because alcohol has a special predilection for peripheral nerves. Purgation at regular intervals is advisable. In favorable cases when the pain has subsided, the patient is allowed to exercise moderately his limb. In order to combat the wasting caused by disuse, massage should be then instituted: first gentle manipulation every day for ten to fifteen minutes, later deeper rubbing for a longer period of time. Faradism may be added to the massage in order to accelerate the return of power in the muscles.

Recently injections of alcohol into the sheath of the nerve or in the immediate vicinity have been recommended, but some observations show that this procedure may be followed by disastrous results; permanent paralysis and marked trophic disturbances have been reported. For the limitation of alcoholic injections see page 434. The same remarks can be applied to injections of osmic acid advised by some writers. Relief has also been obtained from injections of saline solutions (chloride of sodium, sulphate of magnesium) or of plain distilled water or else of sterile air.

A **subcutaneous injection** of salts does not differ from an ordinary saline infusion. The dose is from 50 to 100 c.c. It has a mechanical and analgesic effect on the nerve. It may be made more effective by adding cocain or morphine according to Schleich's formula (feeble solution: cocain, 0.01; chloride of sodium, 0.20; morphia, 0.02; aqua, 100 grm.).

Cordier recommended injections of sterile air in the close vicinity of the nerve, believing that the distention of the tissues stretches the nerve filaments. The injection is made in the lumbar region, at the external surface of the thigh and above the head of the fibula. The quantity of air injected at each of these points is 50 c.c. The injected area becomes pale. Vigorous massage must follow.

Deep injections of chloride of sodium or sulphate of magnesia in 4 per cent. solutions have given better results. The point at which the injection is to be made lies at the junction of the inner third with the two outer thirds of a line drawn between the sacrococcygeal articulation and the postero-external border of the great trochanter. A long needle is inserted at a depth of 4 to 9 cm. and 40 to 60 c.c. of the solution are injected.

These injections may be repeated several times. In one case I made six such injections at intervals of five or six days.

Recently **"spinal anæsthesia"** has been proposed for the relief of sciatic pain. This procedure (see chapter on Lumbar puncture) which is employed by surgeons in operations upon the lower part of the body, gives excellent results also in sciatica. Its efficiency is due to the effect of the injected drug upon the roots. The medications used in these cases are **cocain** or **stovain** (see Schleich's formula above). **Epidural** injections have given in some cases satisfactory results. Through the sacrococcygeal space are injected two centigrams of cocain solution (1 per cent.) or 5–20 c.c. of saline solution to which one or two centigrams of cocain are added. The needle is inserted at a depth of 3 or 4 cm. This method while less efficacious than the preceding one as far as duration of relief of pain is concerned, is nevertheless useful. It can be applied not only in sciatica, but also in gastric and vesical crises of Tabes (Läven, *Centralbatt f. Chir.*, 1910), in lead colic (Achard, *Gaz. hébd. de méd. et chir.*, 1901), in cancer of the vertebræ (Chipault, *Congrés de Chir.*, 1901). Langbein (*Deut. Mediz. Wchn.*, 1913) uses 1 grm. of novocain mixed with one quarter its weight of sodium bicarbonate and one-half its weight of sodium chlorid. The powder is dissolved in 100 c.c. of distilled water, cold, and the mixture is injected. After the injection the patient reclines with the shoulders raised and the legs low. If there is a tendency to weakness, the patient is laid horizontal for a few minutes. In fifteen to twenty minutes all symptoms of sciatica disappear, but he keeps the patient in bed for two days. Out of twelve patients seven were permanently cured. For details of epidural injections consult Sicard and Cathelin (*Compt. rend. Soc. de Biol.*, 1910).

Rosi (*Policlinico*, 1912) has applied in eleven cases Baccelli's method of intravenous injection of a very weak solution of carbolic acid in treatment of chronic sciatica (1 per cent. or 1 per thousand solution). He obtained complete recoveries; from ten to sixty injections were required for the purpose.

In grave and protracted cases, when the above treatment has failed, surgical procedures must be resorted to. Nerve-stretching, excision of portions of the nerve sometimes give relief. For more details see Treatment of Neuralgia in general.

Recently Pers (*Ugeskrift für Laeger*, Copenhagen, October, 1911) devised a method called **"neurolysis,"** which he applied to seventy cases. After a considerable interval forty-seven out of fifty-eight were found permanently cured as also two others after a second neurolysis. The method consists of freeing the sciatic nerve from all adhesions that may

have developed as the result of perineuritis and which according to the author are responsible for persistent sciatica. The incision along the back of the femur, from the lower margin of the gluteus maximus down to and below the middle of the thigh. The nerve is sought between the vastus externus and biceps and with the finger detached from its adhesions, working always from below upward to the sciatic foramen. Baracz modified this method: he works his finger through the gluteus maximus fibers down to the nerve where it enters the notch and breaks the adhesions.

The methods of Pers and Baracz may be successful in uncomplicated typical sciatica without two much atrophy. If the sciatica is associated with pain in adjoining regions, especially along the crural nerve and upward in the lumbar region, there is evidently an involvement of the plexus and no relief can be expected from neurolysis. To detect an old fracture or old arthritis of the lowest lumbar vertebræ or of sacro-iliac articulation, which all may simulate sciatica, an examination with Roentgen rays is necessary before neurolysis is decided upon.

F. Lumbar Neuralgia.—This form concerns the branches of the **lumbar plexus.**

Etiology.—Cold, rheumatism, grippe, malaria, diabetes, anæmia are the general causes. Locally the nerves may be compressed by pelvic, renal and mesenteric tumors.

Symptoms.—According to the branches of the plexus involved the neuralgis will be present in different areas.

(*a*) In **Lumbo-abdominal** neuralgia the pain is in the lumbar region, lower abdominal area, scrotum and spermatic cord. To this category belongs also **neuralgia of the testicle.** During the paroxysmal attacks the abdominal muscles may become contracted. The skin may be hyperæsthetic, but in old cases it is usually hypæsthetic. Trophic and vasomotor disturbances (herpes, etc.) may be observed. Spontaneous ejauculation may occur.

(*b*) **Obturator neuralgia** is associated with an obturator hernia. The pain radiates to the internal surface of the thigh.

(*c*) In **Crural neuralgia** the pain extends along the course of the crural nerve to the inner surface of the leg and foot. The tender spots are found in the inguinal region, on the inner surface of the knee, internal malleolus and inner border of the foot.

(*d*) Neuralgia of the **femoro-cutaneous** nerve is known under the name of **Meralgia paræsthetica.** It was first described by Roth and Bernhardt in 1895. As the name implies, there is not only a neuralgic pain, but also paræsthesia. The latter consists of a burning sensation and a numbness over the antero-external surface of the thigh. The burning sensation is

slightly decreased by flexing the thigh on the pelvis and the leg on the thigh.

The paræsthetic disturbances are continuous irrespective of the position of the body, but the walking or any displacement of the limb increases their severity and sometimes causes intolerable pain. Generally rest gives relief, but in some cases immobility increases the suffering.

The tactile sense is diminished and the patient feels at each contact that something is interposed between the thigh and the hand applied to it. In some cases the least touch provokes severe pain. The diminished tactile sense (hypæsthesia) is frequently associated with a diminished pain sense (hypalgesia) and thermic sense.

The tender spots characteristic of neuralgia in general are found here at the point of emergency of the external cutaneous nerve from the crest of the ilium.

The **causes** of meralgia paræsthetica are: trauma, cold, infectious diseases, intoxications, constitutional diseases. The most frequent cause is injury. The anatomical relations of the external cutaneous nerve make it vulnerable: the nerve is placed in a muscle indispensable to standing and walking (psoas) and in a muscle (fascia lata) the contraction of which in walking presses upon it and stretches it.

The anatomical examination made in a few cases has shown that the affection is due to a neuritis.

Treatment.—A prolonged rest may in some cases give relief. Sulphur baths, massage, galvanism, internal administration of iodides have been advised. In obstinate cases resection of the nerve is the only means to be used.

Meralgia paræsthetica is not infrequently associated with **"intermittent claudication."** This phenomenon, to which Charcot first called attention in 1856, is characterized by intense pain in the calves of the legs and difficulty of walking appearing a few minutes after the patient begins to walk. As soon as he sits down, the pain begins to subside and finally disappears. According to Charcot, the paroxysms are due to contraction and obliteration of the arteries in the affected limbs.

Men are much more frequently affected than women. Russians and especially Hebrews are particularly predisposed to this affection. Erb made a statistical study of the etiological factors (*Münch. Med. Wchn.*, 1910) and he reaches the following conclusions: Syphilis which is so frequently the cause of arteritis, plays apparently a slight rôle in intermittent claudication. He found only 8 per cent. cases of syphilis. Gout, diabetes, alcohol play a very negligeable part. **Tobacco,** however, is apparently a prominent etiological factor. Over a half of Erb's cases were heavy smokers.

Besides these general causes, local causes have an important place. **Cold** is particularly frequent.

As to the clinical forms of intermittent claudications the following are observed:

(1) **Mild form,** in which the entire condition consists of a sensation of weakness and fatigue, slight pain, some paraesthesia. They all disappear when the patient walks slowly.

(2) **Severe form** in which all the symptoms are very pronounced from the beginning.

(3) Association with Meralgia paræsthetica (see above).

(4) **Vaso-motor form,** in which the vaso-motor disturbances predominate over the other symptoms. Here the individual complains of "dead feet." The latter appear either pale or red. Goldflam (*Neur. Centr.*, 1910) described the following phenomenon. Normally when the leg is extended several times in succession, and then left to drop, the foot becomes very pale; the pallor gradually disappears to be replaced by a hyperæmia. In cases of intermittent claudication this phenomenon is produced with great facility and is very pronounced.

The **diagnosis** depends considerably upon the state of the pulse in the pedis dorsalis artery and posterior tibial artery. The pulse may be very small or totally absent. For differential diagnosis with spinal intermittent claudication see page 334.

The **prognosis** must be made guardedly, as the condition may precede an eventual gangrene through the endarteritis. However great amelioration may be expected from proper management.

Treatment.—An effort should be made to remove the cause. The general and local circulation must be improved. Rest in a horizontal position, local heat and perhaps galvanism—may all be of benefit.

In meralgia paræsthetica the intermittent lameness is clinically the exact reproduction of Charcot's type, but the etiological factor is different.

G. Rare Forms of Neuralgia.

(*a*) **Coccygodynia** or **Coccygeal Neuralgia.**—It occurs mostly in women. Trauma is a frequent cause. Cold, protracted labor may also produce it. Hysteria and neurasthenia are the predisposing causes. The disease is characterized by a pain in the region of the coccyx. The pain is increased in walking, defecation, micturition. The normal sitting position is extremely painful. Rectal examination shows extreme tenderness of the coccyx.

The treatment consists of faradization of the coccyx and rectal suppositories of opium or cocain. When hysteria or neurasthenia are the underlying causes, an appropriate treatment of these affections is the first

indication. The disease is generally very obstinate. Extirpation of the coccyx is sometimes the only remedy.

(*b*) **Spermatic neuralgia** is characterized by pain along the spermatic cord extending to the scrotum and epididymis.

(*c*) **Perineal neuralgia** is characterized by pain radiating from the perineum to the penis, producing a desire for micturition which then becomes painful.

(*d*) **Vesical, rectal, urethral** neuralgias may also occur.

(*e*) **Metatarsalgia** (Morton's disease), which is characterized by pain in the fourth metatarsophalangeal articulation, sometimes also in the second, is considered by some as the result of articular or bony changes and by others as due to a neuralgia of the external plantar nerve. Tightly fitting shoes or prolonged standing are probably the causes of this affection. The treatment consists of avoiding pressure on the heads of the metatarsal bones. Wide shoes may help. In protracted cases removal of the head of the affected metatarsal is the only means.

HERPES ZOSTER (ZONA)

This affection should be considered here, as it is very frequently associated with neuralgia (see preceding chapter).

It is characterized by an acute vesicular eruption developed upon an erythematous base and following the course of a nerve or several nerves; it is accompanied by neuralgic pain.

Pathology.—The lesion of herpes zoster is not definitely settled. Some believe that it is due to a **peripheral neuritis.** Others bring forward ample proofs of an involvement of the **spinal ganglia** (acute inflammation or hemorrhage) or of the **Gasserian ganglion** in cases of zona following the course of the fifth nerve. When the spinal ganglia are at fault, a secondary degeneration develops in the posterior roots and hence in the peripheral nerves. Some observers believe that the primary lesion lies in the **spinal cord.** The researches of Hedinger (*Deut. Ztschr. f. Nerv.*, 1903), of Dejeine and Thomas (*Revue Neur.*, 1907), of Head and Campbell (Brain, 1909) show that the ganglia and both portions (peripheral and central) of the roots are chiefly involved. Laminière (*Rev. Neur.*, 1907) has shown also a degeneration of communicating branches of the sympathetic which corresponded to changes in the lateral columns of cells in the gray matter. A radiculo-ganglionic lesion accompanied sometimes by a lesion in the cord is the anatomical basis of herpes zoster.

Head's explanation of the pain and eruption is very interesting. A lesion of a spinal ganglion will produce a continuous sensory impression

which is directed towards the centers, hence the pain. On the other hand, a peripheral sensory fiber containing vasomoter fibrils of a centrifugal nature will transmit to the cutaneous blood-vessels the irritation caused by the lesion in the ganglion; hence, the erythema upon which the herpetic eruption appears.

As to the **pathogenesis** of zona, within the last few years micro-örganisms have been observed by some authors in cultures obtained from the cerebro-spinal fluid of patients suffering from herpes zoster. Very recently (Deut. Mediz. Wchn. N° 18, 1913) Sunde reported a case of ophthalmic zona which came to autopsy, and a coccus was found in large quantities in the Gasserian ganglion. The micro-organism was especially in abundance in the hemorrhagic foci and small blood-vessels. The ganglion was in a state of acute inflammation; small hemorrhages and round-cell infiltration were marked.

Symptoms.—**Pain** and **eruption** are the characteristic signs of the affection. Pain is shooting in character. According to the localization of the zona special symptoms will be present.

(*a*) **Intercostal Zona.**—This is the most frequent form. The onset of the disease is usually preceded by fever and general malaise. The development of the vesicles may be sudden or gradual but commonly it appears on the third or fourth day. Pain may precede the eruption or follow it. Besides pain there may be a burning, itching or tingling sensation.

The eruption may follow the course of an intercostal nerve, anteriorly and posteriorly, but sometimes it has a different direction and crosses the nerves. At first the skin is only erythematous, but soon elevations are noticeable in areas. The center of the latter becomes a vesicle; the contents of the vesicle is at first serous, but later becomes purulent and hemorrhagic. The lymphatic glands in the vicinity become enlarged in the majority of cases. The bleb soon dries; on the fifth or tenth day a crust is formed and when it falls off, a small scar is left. Usually when the rash begins to heal, the pain disappears, but in elderly individuals it persists months after the eruption has ceased to exist.

The affected area presents objective sensory disturbances: there may be hyperæsthesia or anæsthesia. The latter is present especially over deep and extensive scars.

The affection lasts from several days to several weeks.

(*b*) **Ophthalmic Zona.**—There is usually no prodromal period. The symptoms may consist only of an erythema with œdema of the eyelid. In the majority of cases vesicles appear with very violent neuralgic pain on the forehead near the middle line, on the upper eyelid, and the base of the nose. The center of the eruption is the supraorbital foramen. Sometimes

the rash extends to the side of the nose down to the alæ. Hutchinson pointed out the seriousness of the latter occurrence: he observed in such cases almost invariably ulceration of the cornea.

Grave symptoms frequently accompany ophthalmic zona, viz. conjunctivitis, keratitis (neuro-paralytic), iritis, suppuration of the cornea and of the entire globe. Optic neuritis, paralysis of the third and sixth nerves have been observed in some cases.

(*c*) **Zona of the face** may affect all the sensory branches of the fifth nerve. It may involve the lips, nose and cheeks. The tongue is also occasionally involved. R. Hunt has shown that a herpetic eruption over the auricle and in the external meatus is due to an inflammation of the geniculate ganglion (geniculate poliomyelitis). It is frequently associated with palsy of the seventh nerve (see chapter of peripheral facial palsy).

(*d*) **Zona of the extremities** may follow the exact course of individual nerves or, similarly to intercostal herpes, present an independent or irregular distribution. In a case of **cervico-brachial** herpes zoster observed by me (*Amer. Medicine*, 1909) on an aged woman excruciating pain was followed three days later by a vesicular eruption extending from above the left scapula down over the external aspect of the left upper limb to the wrist.

(*e*) Zona may occur on the **abdomen, dorsum, neck** and **genitalia.**

Course, Prognosis.—Apart from the complications of the ophthalmic form, the prognosis is usually favorable. Recurrences take place, although rare. In one of my patients, a man of twenty-nine, three attacks occurred within two years and invariably in the same area (intercostal). Bilateral herpes is also rare.

Etiology.—**Trauma** and **cold,** intoxications with carbonic acid gas, lead and arsenic are not infrequently traced as causes.

It has also been observed in the course of infectious diseases, (grippe, pneumonia, etc.) of diabetes. Finally zona may develop independently as a **primary affection** with a febrile onset and glandular swelling; otherwise speaking, as an **infectious disease.** The analogy with the onset of acute anterior poliomyelitis is striking. Epidemics of zona have been observed by a number of writers. Herpes may occur at any age, but predominately under the age of twenty-five. Infants are also subject to it. Aged individuals are peculiarly susceptible to it.

Treatment.—The **primary** form should be treated as any other infectious disease by diuretics, purgatives and rest in bed. The **secondary** or symptomatic form will be managed according to the original malady. Locally the vesicles should never be pierced. Applications of sedative ointments or liniments with opium and cocain and later during

the drying stage of zinc powder are usually sufficient. I obtained good results with application of ichthyol. For relief of pain bromides and coal-tar products can be given. In protracted cases arsenic and galvanism are advisable. Resection of the posterior spinal roots at the level corresponding to the area involved has been recommended and good results reported (*Leriche. Lyon Chir.*, 1912).

CHAPTER XXV

FUNCTIONAL NERVOUS DISEASES

NEURASTHENIA (NERVOUS EXHAUSTION)

UNDER this name is known a symptom-group the chief characteristic of which is a persistent **neuro-muscular fatigue with general irritability.**

Beard (in 1880) was the first who grouped together the individual symptoms of the disease which prior to him were attributed simply to nervousness.

Symptoms.—(*a*) **Diminution of Muscular Energy** is the most prominent symptom. It is manifested in a **lassitude** which interferes with the patient's daily work. The usual physical exercises cannot be carried out. The least exertion brings on an undue **fatigue.** In some cases the tired feeling is felt only in the legs, in others in the entire body. In extreme cases the patient refuses to stand, walk or move from one place to another. The characteristic feature of this fatigue is that it is particularly **marked in the morning.** In such cases the sleep is not refreshing to the patient; in getting up he feels as if he had not spent the night in bed.

In other cases the fatigue is not continuous, but paroxysmal. The patient gets some relief for a few hours and then suddenly upon a slight exertion or even without it begins to feel the fatigue.

(*b*) Neurasthenics very frequently complain of **backache.** In some cases it is not a genuine pain, but a pressure, a burning or only an indescribable discomfort along the spine. These sensations usually increase upon exertion or more or less prolonged standing. The skin over the spine is over-sensitive, so that the least touch or even the contact of the clothes makes the patient uncomfortable. The most frequent seat of the backache is the lower (sacral) portion of the spinal column.

(*c*) **Headache** is a common occurrence. It consists frequently of a sense of fullness, heaviness, pressure or of a band-like constriction around the head (neurasthenic lead-cap). It is sometimes confined to the forehead, the temples, but most frequently to the occipital region. In some cases instead of fullness there is a sensation of emptiness, of a vacuum. In other cases the headache is accompanied by vertigo, noises in the ears, dimness of vision.

The headache may be continuous or paroxysmal. It is particularly evident in the morning.

(*d*) **Gastro-intestinal Disorder** is a very frequent symptom. In the mild form there is usually **preservation of appetite.** The patient enjoys his food, but an hour after the meals a discomfort appears. Fullness and pressure in the epigastrium, eructations, are tormenting the patient. At the same time he feels oppressed, flushes of heat go to his head, the heart palpitates. He feels somnolent, heavy. This condition lasts during the entire process of digestion.

Constipation is a usual accompaniment of neurasthenic dyspepsia.

As to the chemical processes of digestion, the latest observations show that they are about normal in mild cases. The dyspeptic symptoms are due to a deficient motor innervation of the gastro-intestinal tract (**atony**). It is also interesting to notice that in spite of the digestive disorder the patients do not lose in weight.

When, however, the digestion is considerably disturbed, the general nutrition suffers. In such cases there is a marked diminution of free hydrochloric acid and the constipation is obstinate. The patient loses in weight, he is emaciated and pale.

(*e*) **Insomnia,** while not constant, is quite frequently met with. Either the patient has great difficulty to fall asleep or awakes several times during the night in a state of anxiety and excitement. As a rule the neurasthenic sleep is **incomplete.** Terrifying dreams are very frequently observed.

(*f*) **Mental Fatigue** almost always accompanies muscular fatigue. The majority of the neurasthenics suffer from inability to concentrate their thoughts upon one subject for any reasonable length of time. They are absent-minded, incapable to solve problems of a more or less complex nature. Figuring becomes a difficult task. A long conversation on a serious subject is impossible. They are hesitating in their actions and decisions. The least attempt to resume their usual mental occupation brings on headache.

In some cases, however, the patient retains his mental energy, but the muscular fatigue is persistent. In others the mental fatigue appears only after a certain number of hours' work. There are great variations in the degree and intensity of the mental exhaustion.

The **disposition** of the patient usually undergoes a change. He is **depressed, discouraged, highly irritable,** cannot stand contradiction. He avoids his best friends, wants to be let alone. He is pessimistic and does not find pleasure in anything.

(*g*) **Circulatory** disturbances are very frequent. Cardiac palpitation occurs upon the least emotion. Sensations of cold or heat along the spine or in the extremities, lowering of vascular tension, are all due to deficient

tone of the vasomotor apparatus. The patient's skin and especially the hands and feet are moist, clammy.

The **six symptoms** just described are **characteristic** of neurasthenia. There are some minor manifestations which are not observed in every case, but frequent enough to deserve mentioning.

Sensory disturbances may be: hyperæsthesia generalized or, more frequently, localized; various paræsthesias, as tingling, numbness, burning, itching or pain. Pruritus ani, prurigo in general, may occur in neurasthenics. Neurasthenia predisposes the individual to neuralgia. The **special senses** may also be disturbed. The **eyes** get easily fatigued. As soon as the patient begins to read, the letters become blurred and the eyeglobes painful. The **hearing** is somewhat affected; the least noise gives a painful sensation in the ears; various noises are heard by the patient The **taste** and **smell** also suffer sometimes.

Motor disturbances are sometimes manifested in cramps in the legs and tremor. The latter is seen in the tongue and fingers.

Vertigo is occasionally observed. It may be continuous or paroxysmal. In the latter case it occurs mostly in the morning.

Secretory disturbances are various. Either there is a diminution or increase of secretion. In the first case the skin and the mucous membranes are dry; the quantity of urine is diminished. In the second case the perspiration and the amount of urine are abundant. Polyuria and phosphaturia are sometimes observed.

Sexual Disturbances are as a rule present In the average case it consists of some degree of impotence. In some cases, however, the disorder in the function of the genitalia is so predominant that the disease deserves the special name: **"sexual neurasthenia."** Sexual excitement, priapism, nocturnal emissions, premature ejaculation, spermatorrhea, burning sensation in the urethra, extreme tenderness of the scrotum, testicles, penis, are all symptoms of this form of neurasthenia. Added to the other symptoms of the disease, they put the patient in a state of extreme anxiety. Gradually his sexual desire decreases and even disappears. The latter together with impotence and frequent emissions has an unusual depressing effect on the sufferer.

Course, Prognosis.—When the cause is removed and the proper treatment promptly instituted, recovery may follow in a few weeks. The duration of the malady depends upon these two factors. It should, however, be borne in mind that recurrences are not infrequent. Generally speaking, the prognosis is favorable. Except the cases in which the neurasthenic symptoms are secondary to some grave disease (tuberculosis, syphilis, etc.), neurasthenia is a curable disease.

Pathogenesis.—There are no absolutely certain facts concerning the nature of neurasthenia. Various theories have been advanced. Whether in the course of neurasthenia some toxic material circulates in the blood and produces the fatigue in the nervous and muscular systems, or else the nervous system is affected first from overuse and the other organs are disturbed in their nutrition secondarily, it is impossible to tell in the state of our present knowledge. The cells of various cerebral centers primarily and systems of neurones secondarily are probably disturbed in their function.

Diagnosis.—Continuous **fatigue** in the physical, intellectual and moral spheres, especially manifested upon the least physical or mental exertion, irritability, prolonged backache and headache, insomnia, gastro-intestinal atony, are all symptoms sufficiently characteristic of neurasthenia. Each of these symptoms individually taken may be observed in other diseases. It is therefore essential that the entire symptom-group be present.

There are some organic nervous or mental diseases which may be preceded by a prolonged period of symptoms resembling neurasthenic manifestations. The prodromal stage of paresis, of melancholia and mania are characterized by such symptoms. They resemble, however, neurasthenia only superficially, they never present the true and complete picture of the latter. They are merely **neurasthenoid** (neurasthenia-like). The differentiation is therefore necessary, as the prognosis will be radically different.

Neurasthenic symptoms may be observed in the course of **cerebral tumors,** of **tabes,** of **cerebral syphilis,** of **exophthalmic goiter**. A careful examination is necessary in every case with a neurasthenic symptom-group, as an organic disease of the nervous system may be overlooked. This occurrence is particularly noticeable during the earliest period of paresis (see page 436).

Neurasthenia is frequently associated with hysteria. As the symptoms of the former are **mostly subjective** and of the latter objective, there will be no great difficulty in differentiating them.

Etiology.—Neurasthenia is a very common affection. Excesses of any kind, sexual, alcoholic and others, masturbation, prolonged and uninterrupted intellectual effort, depressive emotions, fright, anxiety, worry, traumatism, are the most frequent causes of nervous exhaustion. Neurasthenia is also observed in syphilis; it follows an attack of grippe, typhoid fever or any other protracted infectious disease. The symptom-group accompanies also tuberculosis, anæmia, chlorosis, lead intoxication. This is the so-called **"Neurasthenia symptomatica."**

A neurasthenic state develops more easily in individuals with a pathologic heredity (nervous or mental diseases), as their predisposed nervous system is more apt to succumb under the influences mentioned above than in persons with a normal make-up. It should not be forgotten that nervous exhaustion may occur in any individual subject to the effect of the described causative factors. The latter may make a perfect recovery, while the former is easily apt to suffer recurrences.

Treatment.—The first indication in neurasthenia is to combat its most distressing symptom, viz. **fatigue.** Physical and mental **rest** is the most important element of the treatment. When the tired feeling is marked, the patient must be put to bed. While in bed all mental exertion should be avoided. He should be kept away from all possible causes of worriment or excitement. The latter can be accomplished when the patient is isolated and placed under the immediate care of an intelligent nurse. Full and nutritious feeding is necessary. The state of gastro-intestinal digestion should be taken into consideration in each individual case. Atony of the bowels can be combated by massage of the abdomen, by internal administration of laxatives, among which cascara sagrada is the most desirable, by daily ingestion of sufficient amount of fruit or green vegetables. Atony of the stomach can also be remedied by local massage over the gastric region and by administration of bitters, as nux vomica with gentian, nitro-muriatic acid, etc., immediately before meals. The feeding should be done regularly three or four times a day. Stimulants of all kinds are forbidden. Coffee or tea may be given in very small amount and forbidden, if the sleep is disturbed. In selecting a diet, special attention must be given to the character of food. Some patients, for example, refuse to drink large quantities of milk. It is advisable in such cases to have the patient take the milk at first in very small quantities and then very gradually increase the amount of it. By doing so I have succeeded in a number of instances in making the most stubborn patients drink large quantities of milk. Milk is an ideal food for patients confined to bed. If in exceptional cases it causes gastric disturbances in spite of all possible precautions, it should be discontinued. Vegetables, fruit, small amount of meat, eggs, custards, are very nutritious articles for neurasthenics. Sweets and very starchy food should be avoided. Drugs are not absolutely necessary in every case. Glycero-phosphates, lecithin are excellent in asthenic conditions of the nervous system. Iron and arsenic are indicated if the blood examination shows a state of anæmia. In case of insomnia or only disturbed sleep sedatives should be avoided as long as possible. It can be combated sometimes by simple measures, as a cool sponge bath or a tepid general bath of ten minutes'

duration; sometimes also by the application of a cold wet towel to the neck. If these means fail, bromides, veronal, sulfonal, trional, medinal, may be tried. Ten grains of any of these drugs is generally an average dose. Of course the latter will be increased according to the indications.

A prolonged rest in bed is not without some inconveniences, as it may lead to loss of appetite, to retardation of all the functions, to muscular wasting. To obviate these disturbances, S. Weir Mitchell recommended to associate with the rest in bed also massage, passive movements and electricity. At first these measures may be applied daily, but later only every other day. By these means the general nutrition is benefited considerably.

Hydrotherapy is a very useful adjuvant. It can be administered as a brief douche, a sponging or ablution. The temperature of the water will vary according to the case. Some patients cannot stand cold, some prefer tepid water, others feel better after hot water. A gentle general rubbing after the application of water is not to be neglected.

In **mild cases** of neurasthenia the rest may not necessarily be absolute. The majority of the cases are walking neurasthenics, who are compelled to work. In such cases a brief relaxation from work at the beginning of the treatment should be insisted upon. If it is impossible to obtain it, the patient is instructed to take rest as much and as often as he can. An hour's relaxation once or twice a day will be very refreshing to him. Cool sponge-baths, mornings and evenings, followed by a hard rub, will help the patient considerably. He must retire early, avoid all excitement, including sexual life; otherwise speaking, he must secure as perfect rest as possible. Smoking must be done very moderately and avoided at night, as it is an excitant to some individuals. As to diet, etc., see above.

NEURASTHENIC PSYCHOSES (PSYCHONEUROSES) (PSYCHASTHENIA)

Closely associated with neurasthenia are certain mental disturbances which develop in individuals especially predisposed, viz. **neuropaths.** The latter present a special make-up of their nervous system which becomes easily affected from the least cause. They are individuals whose nervous or mental equilibrium has not a solid basis and is constantly threatened with a break. They are usually burdened with a heavy hereditary predisposition. They are peculiar, very emotional, impressionable, self-analyzing, extremely sensitive and scrupulous. They are eccentric, dreamers, with romantic tendencies. They are subject to attacks of great anxiety, to morbid fears, obsessions. The mental symptoms may appear at various periods of the patient's life, but they ordinarily develop

when the organism is in a state of exhaustion. In such individuals the neurasthenic symptom-group develops with the greatest facility and is characterized by its special tenacity and persistence.

The nerasthenic manifestations may disappear, while the psychic disturbances will persist. Usually, however, they develop and improve simultaneously.

The chief characteristics of the mental symptoms is complete lucidity of the patient's mind, his complete consciousness of their presence. The patient realizes the absurdity of the phenomena, but is unable to overcome the irresistible sensations. A brief description of the psychic disturbances is necessary.

A. **Phobia** or **Fear.**—The most common form of fears is **agoraphobia.** The patient fears an open space. He will avoid crossing a large avenue, a boulevard or a field.

Monophobia is the fear to be alone.

Claustrophobia is the fear of a closed space. The number of phobias can be increased indefinitely.

To this category belongs also a special phenomenon, viz. fear of touching objects; it is called: **Délire du toucher.**

A fear naturally develops a **state of anxiety.** If, for example, the patient suffers from agoraphobia and he is compelled to cross a field, he will become agitated, he will tremble, he will make several attempts to do the act, his heart will palpitate violently, he will perspire abundantly. The state of anxiety will be so great that he will have to abandon his plan, or else to procure himself someone who will accompany him.

B. **Doubts** or **Indecision. Folie de Doute.**—A patient affected with morbid doubts may reveal the state of his mind in every act of his life. In writing a letter, for example, he will not be certain whether the proper sentences were used or his signature is placed. He will therefore tear up the envelope, read the letter over again or write another. He may repeat the same act several times. Another patient after turning out the gas in his room at night may be tormented by the possibility of a mistake. He will get up, light the gas again, turn it out and a few minutes later perform the same act over again. Not being sure of the correctness of their actions, such patients are continuously in a state of doubt. This condition may interfere with their daily occupations, with their sleep and with their whole life. They feel unhappy. The French call it madness of doubt (Folie de doute.)

C. **Obsessions.**—Under this term is understood a state of mind when a certain thought, idea or image invade it and the patient is unable to free himself from them. He realizes the groundlessness, the absurdity

of such tenacious ideas or images, but he finds himself powerless in the presence of the irresistible force. **Kleptomania,** for example, is a variety of obsessions. The patient cannot resist the irresistible impulse to steal insignificant objects. One of my female patients cannot resist a knife. She would get paroxysmal desires to kill, although she would make efforts to avoid the sight of sharp instruments. Another patient was obsessed by the idea that her child will die at a certain hour. In some cases the obsessions may be so intense that irresistible impulses follow and crimes are committed.

What characterizes the obsessions is the complete lucidity of mind. The patient is perfectly conscious of the criminality or absurdity of a certain act. He struggles against the thought and impulse, he suffers morally. He may sometimes overcome the fight, but sometimes he succumbs and commits a criminal act.

D. **Abulia** or **Deficient Will.**—The patient thus affected has not the power to do a certain act. One of my patients while walking on the street could not step over a loose brick or a leaf of a tree. Another patient in going to his business office could not walk over the shortest distance, but had to select the longest route. Should these patients, recognizing the ridiculous side of their condition, attempt to overcome it, they would be thrown into a state of anxiety, would tremble and even cry. Their suffering is indeed great.

E. HYPOCHONDRIA

This affection is quite frequent. It is related in some of its manifestations to neurasthenia, but it is different from the latter in its essential features. It occurs mostly in men before middle life. It develops usually upon a neuropathic basis (see preceding chapter).

Symptoms.—The disease is characterized by a vivid **sense** of having one or more organs in a diseased state. Functional changes are usually absent, but if they are present, they are extremely slight. The patient complains of a multitude of vague symptoms referable to one or several organs. Believing that a special organ is affected, he will observe its function from day to day or from hour to hour and the most insignificant symptom will be interpreted by him as pathognomonic of some disease in a state of development. If he concentrates his thoughts upon the digestive apparatus, he will adopt a special diet, exclude important articles of his food, change the régime every few days until a new symptom from another source will make its appearance. At once he will give up the idea of the gastro-intestinal tract and attribute his illness to another

organ. Should he notice an increase or diminution in the daily quantity of urine or a change in its color, his thoughts will be transferred to the kidneys. One of my patients, a male of thirty-eight, believed two years ago that his testicles were in a diseased state. He began to wear a suspensory padded with cotton in the hottest days of July, would apply extreme heat mornings and evenings, so that the scrotum became intensely erythematous. Six months later, subsequently to exposure, he developed a mild lumbago. At once he began to believe that the origin of his troubles lies in the kidneys. He removed the suspensory with the cotton, also all application from the testicles and abandoned himself to the analysis of the renal function. His urine had to be examined chemically and microscopically every week. Gradually he took up each of the abdominal and thoracic viscera and now he is under the impression that his brain is affected. In spite of his continuous preoccupation over his body, he never missed a day in attending to his affairs (he is a successful broker).

In another series of cases the patients complain of symptoms referable to the entire organism. One of my patients, a male of thirty-eight, has disagreeable sensations in the region of the liver and of the stomach, in the epigastrium; he has palpitation of the heart, numbness and aching in the limbs, shortness of breath, pressure and pulling sensations about his head. A thorough examination failed to reveal physical signs of diseases of any organ. What is striking about these patients is good general development of the body. The above mentioned patient has been suffering for several years and still he has a healthy appearance. In some cases, however, they lose in weight, especially when they restrict their diet to a very limited amount of food and exclude all nutritious articles. The improper food selected by the patient himself may lead to gastro-intestinal disturbances.

Believing that he may be affected with some pulmonary disease, the patient will take special precautions against cold. He will fear to go out and when he does go out, he will put on chest protectors, scarfs, abdominal binders, etc. Overheated he then exposes himself to cold more readily and frequently takes cold. All these disturbances are usually slight, but the patient will always exaggerate them to an unusual degree.

A very frequent form of hypochondria is the one pertaining to the sexual function. Impotence is a common complaint. In reality there is only a fear and anxiety about the sexual power. In the majority of cases the patients are capable to perform the act. It is therefore a **"psychic impotence."** Being preoccupied by their ideas, they look for causes

of their impotence, and should masturbation be present in their history, they attribute the former to the latter and believe themselves incurable. An occasional emission will reinforce such a belief.

Another characteristic feature of the hypochondriacs is the tendency to speak to everybody about their ills. Not infrequently they keep accurate records of the slightest changes in their condition. One of my patients used to send me every fourth day a most detailed account of his hourly state of health. Going from physician to physician, from hospital to hospital, they carry with them their records and insist upon reading them. During the examinations they watch the expression of the physician's face all ready to interpret in their own way the least change. The educated hypochondriac frequently consults medical books, looks for his symptoms in various diseases and usually in the most incurable affections. His fear and anxiety are naturally increased and the more he examines himself the more he becomes convinced of the hopelessness of his condition. Desperate he leaves the regular physician, procures patent medicines, consults quacks, takes up osteopathy, faith cure, Christian science, etc. Sometimes he keeps on taking medicines and follows at the same time some of the above special cures. This state of affairs continues months and even years.

Diagnosis.—The disease is frequently confounded with neurasthenia. In the latter there is the typical chronic fatigue manifested upon the least mental or physical exertion, the striking irritability upon the least provocation. In hypochondria these symptoms are absent. It is true that in some cases of neurasthenia there may be present some hypochondriacal ideas. In the majority of cases each of these affections is separately encountered and the typical pictures characteristic of each are observed.

A hypochondriacal condition is observed as an early stage of some forms of insanity. The qualitative mental changes of the latter are not found in hypochondria.

Etiology.—A neuropathic basis is commonly observed. Idleness, monotony, absence of interest in any special pursuit in life, sedentary occupations, alcoholic or other excesses, lowering of the general nutrition, are all important causes of hypochondria. It is frequently observed in advanced medical students and young practitioners. It is also observed in those individuals who, after having accumulated large means, retire at a too early age and begin to lead a life free from activity.

Course. Duration. Prognosis of Psychoneuroses.—The phenomena described above do not necessarily accompany the psychoneurotic individual through his entire life. They are but episodic manifestations. They may appear at any time upon the least accidental psychic shock

or after some intercurrent disease. They disappear after an appropriate treatment, but may reappear with the greatest facility. These individuals possess a special make-up of their nervous system, whose reaction to external and internal stimuli is different from that of normal individuals and demonstrates an extraordinary emotionality, impressionability, extremely prompt response to the slightest stimulation. Instability of character, of disposition and of mentality—are the chief characteristics of the psychoneurotic or psychasthenic individuals.

Recovery is possible, but usually the condition lasts many years. It may disappear with advanced age.

Treatment of Psychoneuroses. Psychotherapy. Psychoanalysis.—Before psychotherapy and psychoanalysis are discussed, a few words will be said of the **general management** of the cases. Psychoneurotic individuals are persons with a specially unstable nervous system. Appropriate hygienic measures should be always applied. Careful discrimination must be made in the choice of occupation. A career free from great mental strain and from multiple obligations should be chosen. A simple life, regular habits, avoidance of alcohol and of sexual excesses, hydrotherapy, outdoor moderate exercises, are essential.

The nature of the manifestations observed in psychoneurosis is a sufficient indication of the fact that mere medications or physical means will fail to relieve the tormenting thoughts of psychasthenic individuals.

The chief element which is affected is the **psyche.** The treatment in such cases must therefore be before everything else psychic. The main object consists in enabling the sufferer to overcome the distressing thoughts by means of mastering his will power and thus dislodge the obsessive thoughts which are so persistent and so tenacious. The chief aim in psychotherapeutics is to reach the point where the patient begins to gain conviction of the curability of his case. As soon as this is attained, recovery follows rapidly. But to reach that conviction requires considerable effort on the part of the physician. It can be accomplished by gradual and repeated suggestive influence. **Suggestion** can be exercised in any form for practical purposes. Thus advantage was taken of this idea by various sects and lay persons. The history of Lourdes in France, of Christian Science, of hypnotism, of Emmanuel movement, as well as of mesmerism with its theory of magnetism—all proves that suggestion has given even with the unscientific methods some good results in some selected cases. The scientific conception of suggestive methods begins with Bernheim of Nancy. Suggestion consists of forcing into the patient's mind the idea or the conviction of his eventual recovery. This conviction can be gained in two ways: either **blindly** or by the way of

reasoning. In the first case the patient falls entirely under the healer's influence, such as is seen in hypnotism or in the methods employed by various cults or religious movements. In the second case, viz. with the method of reasoning the conviction is gained by the patient after one or several séances of logical and reasonable demonstration of the unfounded cause of anxiety or worry over ills that are not in existence. This method is **persuasion.** It is rational and scientific. We have here an exposition of facts in the interpretation of which the patient himself participates intelligently. It is most enduring as the patient's intelligence is thereby trained in the method of observation. A hypochondriac, for example, complains of his heart, kidneys, liver, which undergo degeneration in his judgment. The first requisite in such a case is the necessity of the patient's confidence, and this can be accomplished by giving the patient our close attention. He must feel that we are ready and anxious to hear his account from beginning to end, that we, as a friend, are interested in his welfare. We must exhibit a certain amount of sympathy. Sincerity and actual charitable spirit must be present in the physician. The next step is to teach the patient that his heart is not diseased by teaching him to count his own pulse, by pointing out the absence of various disorders in the lungs, kidneys. If the patient's fixed idea has a relation to the kidneys, examine his urine in his presence, demonstrate the absence of abnormal elements indicative of a renal lesion. Should the patient actually suffer from some slight disturbances in the domain of any of his organs, an appropriate treatment must be instituted. Special stress must be laid on the slightest improvement, on the disappearance of symptoms, so that the patient will see in it each time a new reason for encouragement. If the patient's expression, tone of voice, reveals still some skepticism, it must be at once attacked with gentle vigor, always basing oneself on facts which should be actually demonstrated to the patient. In such a manner the patient will succumb to evidences brought before him and accept our statements as incontrovertible. Considerable tact and skill must be displayed by the physician. By tactful and encouraging words or demeanor he will be able to exercise a powerful influence in his attempts to dislodge the morbid fixed ideas. **Psychotherapy** understood in its proper sense is useful in management of all ills, but finds its greater field of usefulness in psychoneuroses.

Psychoanalysis.—In spite of the excellent results obtained in a large number of cases by the methods of persuasion and suggestion, there are nevertheless instances in which psychotherapeutics are insufficient and sometimes meet with total failure. A new psychological method has been

developed by Breuer and especially by Freud, which enables one to get a deeper insight into the nature and origin of the morbid phenomena of the psychoneuroses. It is based on the principle that the mental processes of an individual are composed of two separate groups. One of them controls the conscious acts of daily life. The other group constitutes ideas and wishes which occurred to us some time ago, but which the conscious ego endeavored to reject, but as they cannot be annihilated totally, they remain in a latent state and therefore subconscious. The conscious personality continuously strives to absorb the latter by assimilation, but when it is incapable to do so, a conflict is established between the two groups of thoughts, and then various phenomena characteristic of psychoneuroses make their appearance. Normally the conscious ego is victorious in the conflict, the subconscious ideas are assimilated and destroyed, but if the latter are only pushed to the background, they will invariably emerge and under the influence of any exciting cause will manifest themselves in some morbid act or morbid tendency. The latter may not take the form of the original idea or wish, but some other form and become thus merely a substitute.

If there is a possibility of bringing to the surface the subconscious mental presentations so that the conscious ego will thus be enabled to meet them, get full control over them and assimilate them, the morbid mental phenomena will be removed. One of the procedures utilized for this purpose is the **"free association"** method. The patient is told to concentrate his thoughts on his special psychoneurotic disturbance and bring to his memory all possible ideas that occur to him at that time. He must be reminded not to select only those ideas that in his opinion are proper to relate but repeat indiscriminately every insignificant fact, no matter how ridiculous or improper it may appear. In the mass of expressions and sentences uttered by the patient who is unburdening himself from long forgotten thoughts the so-called **pathogenetic link** will be found. It will be seen that the patient's suffering is but the outer expression of an effect produced by ideas or wishes concealed in the subconscious world of the individual's ego. When they are brought to light, the patient sees them one by one passing before him, he gains control over them and finally frees his personality from them.

The **psychoanalysis** just described is unquestionably a very useful procedure. Unfortunately in its practical applications it presents some difficulties. The most objectionable among them is that it consumes considerable time and many séances with the patient before we succeed in having him unburden himself freely from all his thoughts, especially those of a disagreeable nature or of a sexual character. Freud claims that

the chief causal factor in all psychoneuroses including hysteria is always a **sexual moment,** a sexual trauma occurring some time in the patient's life even in childhood; that the psychoneurotic phenomena are the result of the conflict between libido and sexual repression and the symptoms are a sort of as compromise between these two psychic streams.

This conception of a sexual factor as being the only etiological factor in psychoneurosis is not as yet accepted by all.

F. ANXIETY NEUROSIS

A state of "anxiety" is found in every form of psychoneurosis, but there the anxiety is a secondary phenomenon or the consequence of other disorders of the psyche. Freud has isolated a clinical picture in which all the components of the symptom-complex are grouped around the main symptom of anxiety. He termed it "anxiety neurosis." He places it among the psychoneuroses. The symptoms are as follows.

(1) **General Irritability.**—Auditory hyperæsthesia is frequent. The latter causes insomnia.

(2) **Anxious Expectation.**—It is associated with a pessimistic view of things. The patient interprets every occurrence as an unfavorable omen to himself. This is the most important symptom of the neurosis. The anxiety may develop even without an associated idea, such as fear of going insane or of sudden death, or else it may be connected with disturbances of the somatic functions, viz. cardiac, respiratory, vasomotor, gastro-intestinal, muscular, vesical, etc. Freud considers this syndrome as having nothing to do with neurasthenia, contrary to the custom of many who consider it only as manifestations of neurasthenia. There can be no doubt that among the multiple intermediate and unclassifiable forms of psycho-neurosis and nervousness there is a variety in which the chief symptom is "anxiety." The latter predominates in the clinical picture and every other symptom is subordinate to and dependent on the state of anxiety.

Similarly to other forms of psychoneurosis Freud considers here sexual influences as the direct etiological factor. The treatment therefore should be psychoanalysis.

HYSTERIA

Hysteria had been known to the most ancient writers. For a long time it was considered as the result of disturbed function of the genitalia and especially of the uterus. Only in the seventeenth century (Lépois and Sydenham) a more modern view was proposed and the cause of hysteria was placed in the brain. With the advent of the school of

Salpêtrière and Charcot at its head, the disease was given the first proper interpretation and placed upon a solid scientific basis. Since then hysteria has been considered as a well-defined morbid entity which presents its **special physical** and **psychic** stigmata. Charcot said that it was essentially a pyschic malady and this was practically accepted by all.

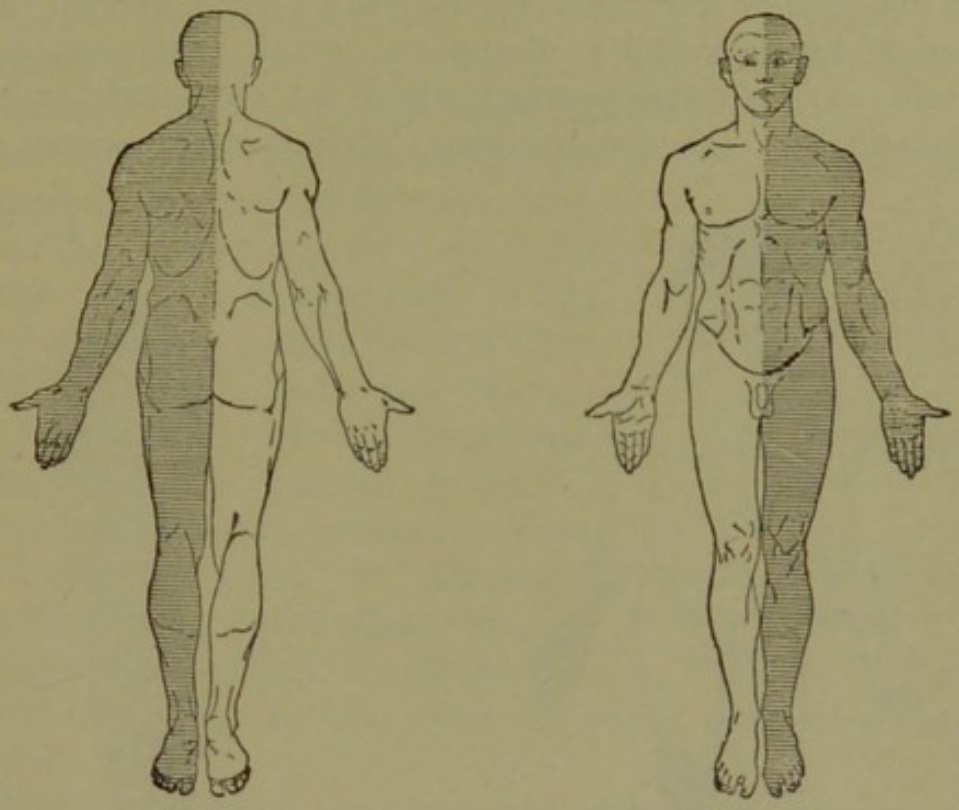


FIG. 142 FIG. 143

FIGS. 142, 143.—HYSTERIC HEMIANÆSTHESIA. (*Original.*)
Anæsthetic parts are shaded.

Symptoms.—The clinical manifestations of hysteria are: **Sensory, Motor, Psychic** and **Visceral.**

I. **Sensory Disturbances.**—They affect the general sensations as well as the special senses. **Anæsthesia** is the most frequent occurrence. It is characterized by its mobility; it disappears and reappears; it changes in its intensity under the influence of the most insignificant cause. The seat of hysterical anæsthesia presents a great variability; it may be confined to very small areas on the limbs or, what is more frequent, to geometrical segments of the latter, and we speak then of glove-like or stocking-like anæsthesia. Finally an entire half of the body may be affected and we say **hemianæsthesia.** Among other regions in which the loss of sensations is frequently observed is the **pharynx.** The anæsthesia may not be absolute; we then deal with a diminished sensibility or **hypæsthesia.** Loss or decreased sensation may be observed in regard to all forms of sensations—touch, pain and temperature.

Hysterical anæsthesia, generally speaking, does not inconvenience the patient and has to be looked for. Thus the patient writes, sews, takes a good hold of fine objects.

The anæsthesia may be associated with or substituted by **hyperæsthesia** (excessive sensibility). The latter is rarely generalized; it is usually confined to a limb, to a segment of a limb, to a very small area. It is also characterized by its mobility, disappearance and reappearance. There are certain zones (**hysterogenetic**) which are quite constantly found hyperæsthetic, viz. the spinal column, inframammary regions, sternum

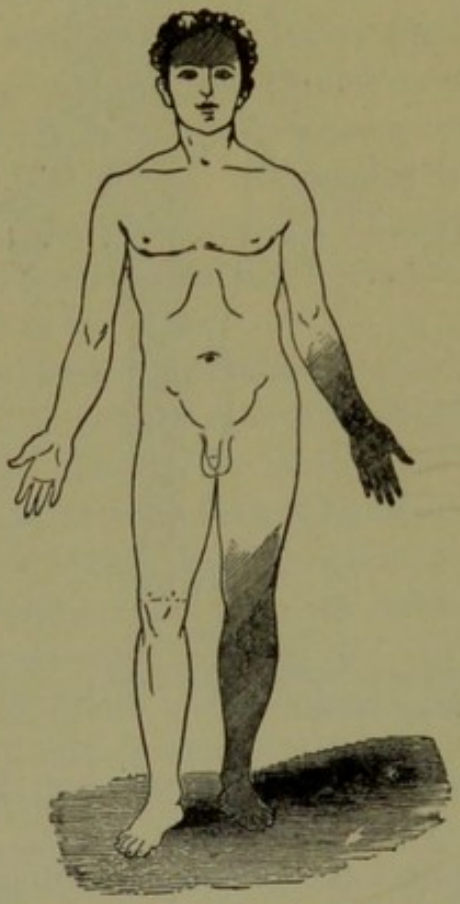


FIG. 144.—HYSTERIC ANÆSTHESIA. Anæsthetic parts are shaded.

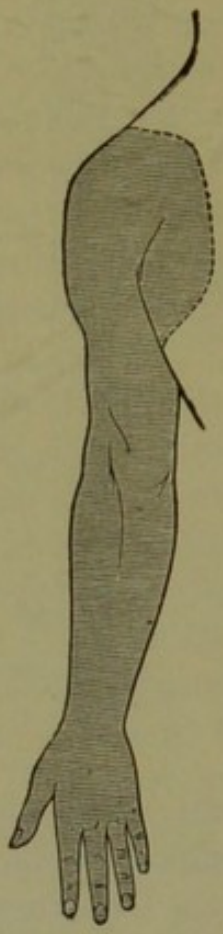


FIG. 145.—HYSTERIC ANÆSTHESIA.

and the groins. Hysterical **headache** is not infrequent and may assume the form of migraine or syphilitic headache by its exacerbations at night. Very frequently the pain is only a hyperæsthesia of the scalp. **Clavus** is a characteristic hysterical pain; it is confined to a very limited area of the vertex of the head. Occipital, nuchal, intercostal, coccygeal pain may occur in hysteria.

Hysterical **pseudomeningitis** is a well-known syndrome; general malaise, anorexia, insomnia, headache and delirium are present. What will differentiate it from true meningitis is the persistent absence of fever, also the pathological state of the cerebro-spinal fluid (see the latter).

Paræsthesias in the form of coldness, numbness, burning, etc., are quite common in hysteria. The so-called **"globus hystericus"** is a well-known paræsthetic phenomenon. It consists of a sensation of pressure in the throat or of a ball passing from the epigastrium to the throat.

The **special senses** also suffer sometimes in hysteria. Taste, smell, audition and vision are not infrequently involved. Perverted hysterical taste and smell are well known. Hysterical deafness is usually unilateral. Special mention must be made of **visual disturbances.**

Total **amaurosis** is rare, but contraction of the visual fields, disturbed perception of color and disturbed accommodation are quite frequent. As to the **contracted field,** it is concentric in the majority of cases; it is more often bilateral than unilateral. The latter is in the majority of cases on the side where sensory losses are most marked, as for example in case of hemianæsthesia. Visual acuity is usually intact. To this symptom is sometimes added disturbance of color perception; it may be dyschromatopsia or achromatopsia: the colors are either confused or not at all recognized.

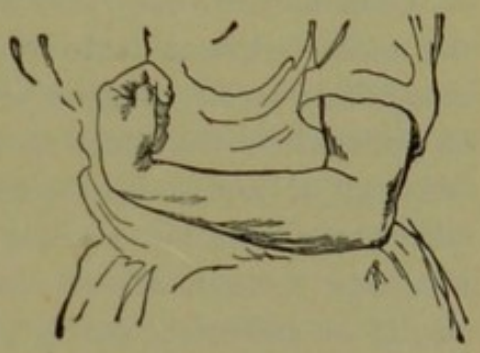


FIG. 146.—HYSTERICAL CONTRACTURE OF THE ARM. (*Gilles de la Tourette.*)

II. **Motor Symptoms.**—**Paralysis** and **contractures** are very frequent. Palsies of a segment of a limb, of an entire extremity or extremities—monoplegia, hemiplegia and paraplegia—may occur. Hysterical palsies and contractures vary considerably from the standpoint of their onset, duration and mode of disappearance. They may last but a few minutes or persist for years. Their essential feature lies in complete absence of objective signs of organic nature. In hysterical hemiplegia, for example, there is no spasticity of the muscles, no increased or abnormal reflexes.

As to contractures, they may affect only the fingers or the toes, a limb, the muscles of the face or the neck. **Blepharospasm** or contracture of the orbicularis palpebrarum, **facial hemispasm** are not very rare. A spasm of the eyelid will produce a ptosis or rather a **pseudoptosis.** When the head is thrown back, the ptosis disappears. The ocular muscles may similarly be affected. An insufficiency of the internal rectus is not infrequent. Diplopia is sometimes observed. A. Westphal observed pupillary rigidity with myosis when the patient concentrated his mind upon it, but the pupils reacted normally when his mind was diverted. The essential feature of hysterical facial spasm is its disappearance during sleep. **Facial palsy** is sometimes observed, but it affects only the

lower half of the face. It is recognized by this sign that when the affected muscles are held with the fingers and moved, no muscular relaxation is noticed, but on the contrary a certain amount of resistance is felt. Besides, the palsy may become ameliorated, disappear and recur almost every day.

Hysterical **torticollis** may be due to a hysterical palsy or contracture of the muscles of the neck. In the first case the head can easily be placed in a straight position, but it returns as soon as it is abandoned. The latter form may disappear suddenly and reappear.

Contractures may also affect the joints. The hip and the knee are the most frequent seats of this condition. Hysterical **coxalgia** can be recognized by the absence of swelling and of increased local temperature, of pain when the great trochanter is percussed and by the absence of crepitation and subluxation. Hysterical individuals may be affected with **choreiform** movements or present special **tremors.** The former may simulate Sydenham's chorea with its irregular and arhythmical movements or more frequently presents rhythmical movements affecting one limb, the face or the neck and occurring at regular intervals. Sometimes the movement resembles dancing (**saltatory** chorea), or swimming (**natatory** chorea). One of my patients, a girl of twelve, with distinct hysterical stigmata, would suddenly jump off the chair and reproduce for one minute dancing movements. This came on in attacks, five or six a day, and she suffered from it eighteen months.

Hysterical tremors are polymorphous in character. They may be slow, rapid, slight or pronounced. They may be oscillatory or of a large amplitude. They are present when the patient is at rest and become increased by excitement.

An interesting motor phenomenon occurring in hysteria is **Astasia-abasia.** It consists of a functional impotence in gait and station; the patient cannot stand and walk, but when seated or bed-ridden he is able to perform all movements with his limbs. Various degrees of this motor disturbance may be present, from absolute inability to preservation of some movements.

III. **Psychic Symptoms.** (*a*) **Suggestibility.**—The most essential and characteristic feature of hysterical individuals is suggestibility. They are easily influenced to change their thoughts, to do certain acts, to acquire certain sensations in the general sensorium or in the sphere of the special senses, to execute or to adopt certain motor phenomena. Experimental palsies, anæsthesias, contractions, modifications of the personality are the best illustration. The hysterical persons are not conscious of the suggested act, they do not understand it, they do not connect it with

their own personality; the suggested ideas develop in them automatically without the controlling power of the will. For the same reason **self-suggestion** is also observed; they have a remarkable tendency to reproduce what they have once seen or heard because of extreme sensibility and impressionableness of their psychic centers, so that real hallucinations are formed. In a case reported by me in the *American Journal of the Medical Sciences*, April, 1906, numerous psychic phenomena of autosuggestion developed under the influence of reading or seeing.

(*b*) **Amnesia.**—Disturbance of memory is a frequent occurrence in hystericals. Variability of answers, untruthfulness, contradiction, capriciousness, inconsistencies in their mode of living and conduct are the result of temporary amnesia. The loss of memory may be: **localized** when events of a certain period are forgotten; **systematized,** when several events concerning several persons or several periods are forgotten; **general,** when the hysterical forgets his entire past life (see Sidis and Goodhart on Multiple Personality). A person thus affected may lose all remembrance of his previous life; he possesses a **new personality.** In some cases the patient passes through two or more different short periods of life as **two or more personalities** (double consciousness) and one is not aware of the other, so that the lives the two personalities lead may be diametrically opposite to each other. (See case of Mary Reynolds—double consciousness by Weir-Mitchell and my case reported in the *American Journal of Medical Sciences*, 1906.)

(*c*) **Disturbance of Speech.**—**Mutism** occurs in hysteria. The patient is not only unable to utter words in a loud or low voice, but is also unable to emit a sound, although the movements of the lips and tongue are well preserved. **Stuttering** may also occur in hysteria, but it is different from the usual stuttering acquired in childhood.

In the latter case the patient is able to make himself understood. He pronounces whole sentences, but stutters especially in pronouncing certain letters, such as m, t, b. After the stoppage is overcome, he goes on speaking clearly until again the special letters interrupt him. Moreover when the patient speaks very slowly and deliberately, the stuttering may disappear. The stuttering is particularle noticeably in rapid speech.

Hysterical stuttering is present not only for special letters, but it is continuous. The patient stutters at every word or syllable and during his entire conversation. The words are pronounced by him slowly as he has difficulty in emitting all verbal sounds. His entire speech is slow and the syllables are pronounced with prolonged effort. Finally hysterical stuttering occurs after a violent emotion, such as railway accident, etc.

Aphasia is not frequent in hysteria. Motor and sensory aphasia

have been observed. They presented all the typical features of genuine aphasia, except the onset, course and termination. Hysterical aphasia may occur after an emotion, imitation or suggestion. It is variable in its course, disappears and reappears and always ends in recovery. Agraphia in association with aphasia has also been recorded. It has the same characteristics as aphasia.

(*d*) **Somnambulism. Catalepsy. Lethargy.**—The first consists of a sleeping state, during which the patient leaves the bed and walks around. In such a manner he is likely to walk through the window and fall down on the roof or on the street. **Catalepsy** is characterized by a rigidity of one or several extremities which, placed in a certain position, may remain so for hours. The patient hears what is going on around him, but unable to control himself; he is probably under the influence of a hallucination. External stimulations (cold water, electricity) frequently arouse the patient. **Lethargy** is characterized by a sleep which comes on suddenly and during which the patient reacts to external stimulation; there is also no relaxation of the muscles, but some rigidity; the masticator muscles are contracted, the eyelids are animated with a tremor. When the patient awakes, he has no recollection of the attack of sleep.

(*e*) **Hysterical Fainting.**—With or without premonitory symptoms the patient suddenly feels fainting and falls. The eyes are half-closed. The hands are animated with slight twitchings. The face is pale, but the **heart beats are normal.**

(*f*) **Hysterical Paroxysms** (Major Hysteria of Charcot).—They remind of epileptiform seizures, but they differ essentially from the latter. An attack is usually preceded by prodromal symptoms which are of a psychic order. The patient becomes sad, depressed or else very excitable; sometimes he has visual or auditory hallucinations. An **aura** is present, but it is slow in coming on; it is usually the well known **"globus hystericus"** (see above). Then follows the attack. The patient becomes unconscious or rather semiconscious, as in falling he rarely injures himself, and in some cases he places himself comfortably before he is seized with the convulsions. The latter are at first **tonic.** The entire body is tetanized: the respiration stops, the arms and legs are stretched out, the face is cyanotic. Rapidly the **clonic** contractions appear. Unlike those of epilepsy they are of wide range and last longer. The body assumes all sorts of attitudes. **Contortions** characterize this phase of the attack. Flexion or extension of the trunk and of the limbs, throwing the legs in all directions, rolling the body from one side to another, position of opisthotonos, grimaces of the face—these are the various positions rapidly succeeding each other. At the same time the patient screams,

cries or else laughs, tears his clothing, etc. Under the influence of hallucinations accompanying this period the patient soon develops **passionate attitudes.** They are expressed in the gestures and the display of the muscles of the face. The images he sees in his dream express alternately sadness, anger, terror, joy or ecstasy. In my case referred to above the patient stood in the middle of the room in the attitude of an orator, reciting a pathetic story which she read, gesticulating, raising and lowering her voice, crying and laughing.

Gradually the violent movements cease, relaxation takes place and the patient enters into the last period of the paroxysm which

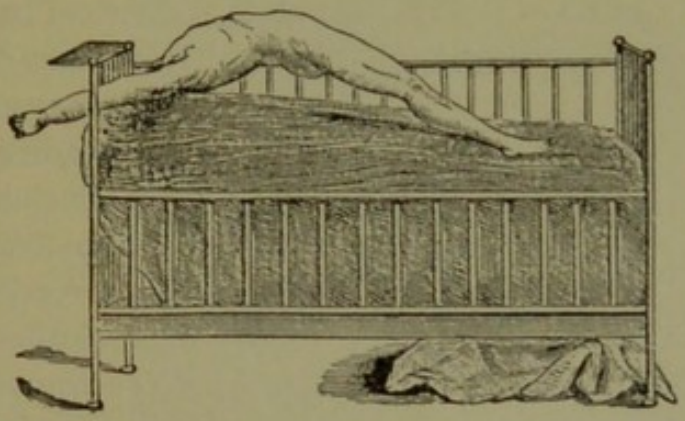


FIG. 147.—HYSTERICAL PAROXYSM. OPISTHOTONOS. (*Charcot.*)

FIG. 148.—PASSIONATE ATTITUDE IN A HYSTERICAL ATTACK. (*Gilles de la Tourette.*)

is characterized by a state of **delirium.** He talks in a low tone of voice, speaks of a certain recent event, which has made a special impression on him. In some cases instead of a delirium the attack may terminate by an out-burst of laughing, singing or crying.

Little by little and sometimes suddenly the patient becomes silent, awakens and the attack is over. The patient may feel somewhat fatigued, but he does not present the profound exhaustion following an epileptic seizure. An attack usually lasts from ten to fifteen minutes, but the periods of grand movements and of delirium may last hours.

The paroxysms do not always present the typical picture just described. They may vary considerably. In some cases the convulsive movements begin suddenly without an aura. In others the delirium—as a final phase—is absent. In still others there are only tonic movements. The movements themselves may be prolonged or unusually brief. They may be limited to a certain limb or portion of the body. In one of my recent

cases the convulsions simulated Jacksonian epilepsy, as they were confined to the right arm and leg.

The hysterical paroxysms are rarely isolated. They repeat themselves sometimes at regular intervals, upon the least emotion. Sometimes one attack follows another and this condition may last hours and days. The patient is then in **status hystericus.** Hysterical paroxysms may be associated with epileptic seizures. Such a combination is called **hystero-epilepsy.** The latter should, however, not be considered as a special entity, as it is only an association of two independent neuroses, each with its characteristic symptoms.

FIG. 149.—HYSTERICAL PAROXYSM. ARE DE CERCLE. (*Gilles de la Tourette.*)

The So-called Hysterical Insanities.—The last two stages of a typical hysterical paroxysm can be placed under this heading. The delirium and the hallucinations under which the patient is laboring at that time render him totally irresponsible. Hysteria may **simulate** various mental affections. Attacks of depression or else of excitement, persecutory or other delusions associated with hallucinations may give the impression of classical psychoses. Hysterical melancholia, hysterical mania, hysterical paranoia have been described, but they are not genuine forms of mental alienation. A careful analysis of the symptoms will show that attacks of delirium, of depression, of excitement and even periods of apparent mental derangement are only episodes in the life of hysterical individuals; they are equivalents of ordinary hysterical paroxysms, just as psychic phenomena take sometimes the place of ordinary epileptic seizures. The course, character, duration and the consequences of the psychic manifestations are not at all those of the typical psychoses; they are all a sort of transformation of ordinary hysterical paroxysms and they all may be the result of autosuggestion. The conception of hysterical insanities as morbid entities is erroneous.

IV. **Visceral Disturbances.**—The **larynx** may be the seat of various symptoms. **Aphonia** is not infrequent, it comes on suddenly; patient is unable to raise his voice; he only whispers. The laryngoscope reveals total absence of palsy of laryngeal muscles. Various laryngeal noises are also observed. **Hiccough** is a familiar phenomenon. It develops suddenly and may disappear suddenly. It occurs in attacks lasting a

certain number of days or weeks. It is usually very loud and may resemble the barking of a dog. **Hysterical cough** is paroxysmal. It usually appears upon emotion and disappears when the patient's mind is diverted toward a special subject. It disappears when the patient is asleep. **Hysterical dysphagia** consists of an intermittent spasm of the pharynx and œsophagus. If it is continued, it will lead to inanition. The same can be said of **hysterical anorexia,** which may become associated with attacks of **vomiting.** These gastric disturbances may be only temporary or persistent. In the latter case they are quite serious.

Tympanitis is sometimes observed; it may be partial or generalized. It may simulate **pregnancy.** Hysterical **pseudo-peritonitis** may occur.

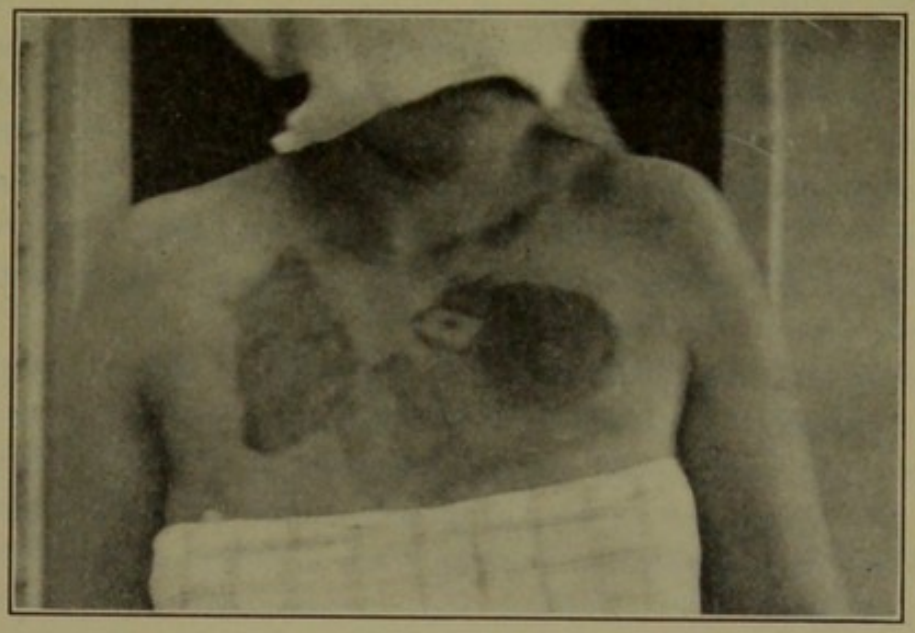


FIG. 150.—HYSTERIA. SYMMETRICAL ERYTHEMATOUS PATCHES ON THE CHEST WHICH DEVELOPED RAPIDLY AFTER A VIOLENT EMOTION.

It may be accompanied by hyperæsthesia of the abdomen, vomiting, constipation, but **fever is absent.** It may also simulate **appendicitis.** In one of my cases the patient was operated without my knowledge and the appendix was found absolutely normal. Shortly after the operation the hyperæsthesia of the same region returned.

Hysterical anuria and dysuria, also retention of urine, are occasionally observed. One of my female patients, after a series of shocks—death and diseases in the family—developed anuria which alternated with retention of urine. **Polyuria** is not an infrequent phenomenon. **Sexual function** may be altered. Either there is an increase or diminution of sexual appetite; sometimes there are perversions of sexual desire.

Vasomotor disturbances are seen as sudden flushing or pallor of the skin, dermography. The latter consists of persistency of a red line

on the skin when the latter is slightly irritated. Erythematous patches following a violent emotion may occur. Various spontaneous hemorrhages (independently of cases of malingering to which hysterical patients are subject) may be observed: ecchymosis, epistaxis, hematemesis, hematuria, hemoptysis (Fig. 150). They are, however, rare. The menstruation may be disturbed. **Hysterical fever** cannot be denied. It develops spontaneously and comes down without any treatment. The oscillation is of very wide range. It may be present without the usual accompanying symptoms, viz. headache, rapid pulse, thirst or burning of the skin. Hysterical fever is rare. Malingering should always be excluded.

Trophic disturbances occasionally occurring in hysteria are: œdema, ulcerations, sudden falling out or graying of the hair. I have observed a remarkable case of gangrenous patches on the chest and arms which leave no doubt as to their hysterical nature. They developed rapidly after a fright, began with a simple erythema, persisted for a long time without spreading, but commenced to improve and finally recovered when electricity was applied a few times. Another shock occurred, again a few patches appeared and again they disappeared when electricity was resorted to.

Course, Duration, Prognosis.—The above described sensory, motor psychic and visceral symptoms are not always present in every case. There are mild and severe cases. The disease usually runs a chronic course. The symptoms develop gradually under ordinary circumstances. They may, however, develop rapidly when a shock, an emotion, are the etiological factors. The essential characteristic feature of hysteria is the great variation in the manifestations. Some symptoms disappear, but may reappear upon the least emotion; others persist. The prognosis as to life is good, except in cases of marked visceral disturbances, as spasms of the larynx or dysphagia or persistent anorexia.

Hysteria as a rule is a curable affection, but in some cases it may last indefinitely. The duration and the prospects of recovery depend upon the circumstances, surroundings, mode of living and the treatment.

The hysterical paroxysms are difficult to combat, they are exceedingly stubborn. The contractures are also difficult to cure. In **children** the disease is less persistent than in adults and the usual stigmata are frequently absent. The symptoms are rather single; either mutism, or a paralysis, or a contracture, etc.

Diagnosis.—In making a diagnosis of hysteria, first of all organic diseases should be eliminated. Hysterical palsies, for example, may simulate hemiplegia or monoplegia. The following are the differential signs of the two forms of paralysis.

Organic Hemiplegia	Hysterical Hemiplegia
1. Spasticity of the affected limbs.	1. Flaccidity of the affected limbs. If some rigidity is present, a voluntary resistance during passive movements is felt by the hand of the examiner, showing therefore the patient's ability to oppose the movements. In organic hemiplegia the patient is unable to resist.
2. Tendon reflexes abolished immediately after the onset, but later exaggerated.	2. Tendon reflexes intact.
3. Babinski sign present.	3. Babinski sign absent.
4. Oppenheim's and paradoxical reflexes present.	4. Oppenheim's and paradoxical reflexes absent.
5. Ankle-clonus present.	5. Ankle-clonus absent.
6. Voluntary movements on the paralyzed side are abolished.	6. Voluntary movements on the paralyzed side are not totally abolished.
7. Abdominal and cremasteric reflexes are usually, esepcially at the beginning, diminished or abolished.	7. Abdominal and cremasteric reflexes are normal.
8. The course is regular: spasticity follows flaccidity; improvement is gradual.	8. The course is irregular: flaccidity or some rigidity remains; the disturbances may change from time to time, become aggravated or ameliorated or else last a very short time.
9. Hoover's "opposition movement" phenomenon is present. (When the patient makes an effort to raise the paralyzed leg, there is a very energetic "opposition movement" in the other leg; a hand placed between the heel of the sound foot and the bed will appreciate the degree of force displayed during the opposition movement).	9. Hoover's "opposition movement" phenomenon absent. A hand placed between the heel of the sound foot and bed will fail to find the counter-pressure of the latter.

Sicard has recently called attention to the following phenomenon. If an elastic bandage is applied tightly to the paralyzed and non-paralyzed symmetrical limb at equal distances from the periphery (usually in the middle of the limb), it is observed that in functional cases the vaso-motor disturbance will be equal on both sides, but unequal in organic cases.

For other additional reflexes observed in organic and absent in hysterical hemiplegia see pages 92, 93, 94.

Hysterical paraplegia will be recognized by the integrity of the reflexes, absence of Babinski's, Oppenheim's and paradoxical signs or ankle-clonus.

Hysterical contractures are usually sudden in onset, disappear under suggestion and under general anæsthesia.

Hysterical paroxysms (see above) are differentiated from epileptic seizures by the following signs.

Epilepsy	Hysteria
1. Onset abrupt. Patient drops unconscious.	1. Onset after an emotion. Patient feels the oncoming attack and takes measures not to fall. Semi-consciousness.
2. Patient bites his tongue.	2. Patient does not bite the tongue.
3. Involuntary micturition or defecation.	3. No involuntary micturition or defecation.
4. Convulsive movements of small range.	4. Convulsive movements of wide range.
5. Pallor of the face after a seizure.	5. No pallor of the face.
6. Exhaustion and profound sleep after a seizure; also some mental hebetude for hours following.	6. Either immediate return to usual occupation or a superficial sleep after an attack. Mentality clear.

Sensory disturbances are observed in organic diseases of the nervous system as well as in hysteria. The characteristic features of hysterical anæsthesia was sufficiently described in the symptomatology (anæsthesia of segmentary or hemiplegic form). **Hysterical neuralgia** will be recognized by the absence of tenderness of the nerve-trunks characteristic of true neuralgia. **Hysterical headache** may sometimes simulate headache caused by a cerebral tumor, but the mental condition accompanying the latter dullness, apathy, etc.) in addition to other objective symptoms will reveal the true nature of the headache. Besides, the failure of pain to affect the general condition of the patient is strongly in favor of hysteria. Pain caused by an organic disease, such as deep-seated tumors and other affections, produces usually a reaction on the general health.

Etiology.—The disease is very frequent. It occurs not only in adults, but also in children and aged individuals. Both sexes are subject to it.

The causes are **predisposing** and **exciting.**

Predisposing.—Heredity plays an important rôle. Functional nervous diseases, insanities, alcoholism, tuberculosis, syphilis in the parents, are predisposing factors in the offspring. Excesses of all sorts, unhygienic mode of living, sedentary life, certain occupations accompanied by a great mental strain and anxiety are all predisposing causes for hysteria.

Among the **exciting** causes the most common is a **shock,** an emotion. It frequently follows **accidents.** Traumatic hysteria is a very well-known condition. In such a case hysteria is due exclusively to the fright and to the psychic shock. In the chapter on traumatic neuroses the subject will be considered in detail. In the chapter on "Psychic Symptoms" it was mentioned that the chief characteristic of hysteria is "suggestibility." Suggestion therefore is also one of the causes of hysteria. It may be induced sometimes by the examining physician, if he is not careful in his procedures. In examining, for example, for sensory disturbances, care must be taken not to speak to the patient of the desired element of investigation. An excellent precaution is to have the patient blindfolded during the examination and not to ask any questions of her while you test with a pin various areas of the skin. There is no doubt that she will make some defense movement if the prick causes pain. Or else blindfolded she may be asked to say "yes" the moment she feels pain or the touch of an object. Moreover she should never be spoken to of possibilities of loss of power in one or more limbs or segment of a limb, or loss of memory, of a grave outlook, etc., etc. The patient may be suggested various disturbances of function in the motor, sensory, vaso-motor, vegetative and psychic spheres with great facility.

Protracted diseases of any nature may be a cause of hysteria. **Intoxications** (alcohol, lead, mercury, morphin, CO_2) are also among the exciting causes of hysteria. **Hypnotism,** especially when repeated, develops hysteria. The provocative factors of hysteria are therefore multiple. However it should be borne in mind that this malady can develop only in individuals who are **predisposed** to it. A special morbid basis is essential: a physical or mental shock will have no special effect on an individual free from hereditary taint, but will disturb considerably the nervous system of a person whose make-up is morbid through heredity or otherwise.

Nature of Hysteria.—At the present time two views are held concerning the nature of hysterical phenomena, viz. the so-called **psychological** and **physiological.**

According to the first, hysteria is a psychic disease. Charcot, Moebius,

Strumpell, Bernheim and Janet see in hysterical manifestations either disturbance of the faculty of mental representation or autosuggestion (see above), or else double or multiple personality; finally a narrowing of the field of conscience. Anæsthesia, the most important according to Charcot of all stigmata, may be explained by Janet's "restrictions of the field of consciousness." The hysterical patient perceives only a small number of sensations and gradually they may entirely disappear and he thus becomes anæsthetic. For the same reason memories of movements may be lost, hence hysterical paralysis. In astasia-abasia, for example, the field of consciousness is so limited that the patient loses the association of mental operation only for standing or walking, but for no other act. Hysterical paralysis is sometimes due to a **suggestion.** The patient adopts, for example, the idea of not being able to walk. This idea may remain in the subconscious state without the patient's knowing it. **Fixed idea** is a very important element in hysteria. It plays a predominant rôle in the life of the hysterical individual: other ideas and experiences have no restraining influence on the fixed idea. Consequently suggestion is a very potent factor when ideas are forced on a hysterical person by another person. Such is the case in **hypnotism.** There is introduced into the patient's mind the idea of the hypnotizer's personality, which finally becomes predominating. It is therefore evident that what characterizes especially the hysterical individual is the want of mental **synthesis,** the inability of grouping individual ideas. He will attach himself to one **fixed** idea and the others will remain in a subconscious state. Hysterical paroxysms cannot be totally explained by the conception of "restriction of the field of consciousness." It is true that a paroxysm is the result of the workings of some predominating idea which was brought on by other secondary ideas of past experiences. Nevertheless during a hysterical attack consciousness has disappeared and consequently restriction of the field of consciousness is not altogether applicable here.

According to the **physiological** theory advanced by Sollier, the hysterical stigmata or paroxysms are of purely **physical nature.** Here the primary disorder is a functional disturbance of the entire brain or only of some of its centers. The sensory, motor, psychic, trophic, visceral and circulatory phenomena will depend upon the centers involved. An inhibition of the latter produces the corresponding symptoms.

In the chapter on Psychoanalysis (page 462) the ideas of Breuer and Freud on the origin of Psychoneuroses were discussed in detail. These two authors consider also hysteria as a form of psychoneurosis. They argue that the various physical manifestations of hysteria are only substitutions for the mental disturbances, stress and unpleasantness (to all

of which they give the name **"affect"**) created by past experiences. The latter are never annulled but remain in the subconscious mind. The distress produced by these latent thoughts which are continuously at work, must be relieved and this is frequently done in hysteria through some physical occurrence as a substitute. Breuer and Freud state that this power of **"conversion"** of mental disturbances into physical manifestations is characteristic of hysteria. When the conversion does not take place, the result will be evidenced in various phenomena characteristic of psychoneuroses such as obsessions, phobias, etc. (see Psychasthenia.) By reason of the efforts of the patient to relegate the painful ideas into subconsciousness, Breuer and Freud call all psychoneuroses as **"defense-neurosis."** In all psychoneuroses they find that the repressed disagreeable thoughts are always of sexual nature. At some time during the patient's life even in infancy a sexual shock of some kind occurred. The contention as to hysterical phenomena being the result of sexual experiences, hidden in the subconscious world is not accepted by all. The psycho-analytic method of treatment of hysteria is based on bringing to light all concealed and repressed ideas and thus have them asimilated by the conscious ego (see chapter on Psychoanalysis).

Recently Babinski (*Semaine Médicale*, 1909) proposed a new revision of Hysteria, from the symptoms of which he removed some manifestations which had been and are still considered by many as belonging to the great neurosis. Dismembered in this manner Hysteria is called by Babinski **"pithiatisme."** In the first place he believes that in a great many cases the phenomena so-called hysterical are the result of malingering. In the next place a number of symptoms which are not brought on by suggestion are fictitious, for example: fever and anuria. In the third place symptoms such as erythema, ecchymosis, ulcerations, gangrene, œdema—are intentionally brought on; they are fraudulent, they are the result of local applications used by the patient for the purpose of arising sympathy or otherwise.

Babinski believes that genuine pithiatic symptoms are those which can be produced and removed by suggestion. Suggestion thus understood implies the idea of the will being the master of the entire situation. Babinski does not believe that **emotion** is the cause of hysteria. It is a factor totally different from suggestion.

Finally he removes from hysteria also exaggeration of tendon reflexes and vaso-motor disturbances of the skin.

To sum up: Babinski considers the phenomena of hysteria or pithiatisme depending essentially on the psychic surroundings or on the predisposition of the patient. They are the result of suggestion or auto-

suggestion, they are subordinate to the will of the patient. Their mutability is characteristic.

Babinski's views as to limitation of phenomena belonging to hysteria are too radical. While it is true that simulation (conscious or unconscious) is met with in a number of cases of circulatory, secretory or trophic disturbances, nevertheless observations reported by competent men prove conclusively that hysteria *per se* is capable to produce these disturbances.

Treatment.—It was mentioned above that hysteria develops usually in individuals with a morbid hereditary history. A tendency to hysteria or to other nervous affections is therefore great. Consequently **prophylaxy** should play a considerable rôle in treatment of hysteria. Nothing should be neglected in building up the body. General hygiene, hydrotherapy (cold or warm water—according to the tolerance), massage, moderate outdoor exercise, good nutritious food are the general measures.

As these individuals are very impressionable, special care must be taken in selecting occupations. The latter must be free from emotional incidents as far as possible. The entire life of hystericals must be so regulated as to avoid shocks, emotions of any nature. As they show a tendency to suggestion and autosuggestion, they must avoid emotional and pathetic scenes, and read only books free from fantastic and terrifying descriptions.

The meals, sleep and healthful recreation must be regulated and strictly adhered to. Stimulants, including tea and coffee, are forbidden. Smoking should be avoided as much as possible. Sexual intercourse must be extremely moderate. The summer months should be spent in a quiet place, free from undue excitement, although some pleasurable entertainment is permissible. It is also advisable to take short vacations during the working months of the year. By all these preventive measures, applied at an early age of life, the patient will grow up physically and mentally strong and his resistant power to shocks or violent emotions will be thus greatly increased.

The treatment of hysterical manifestations is largely **psychic.** In presence of localized palsies, contractures, anæsthesias, globus hystericus, aphonia, paroxysms, etc., all symptoms of psychic nature, the physician must endeavor to convince the patient that the trouble is not serious, is curable and that by strict adherence to the rules of the treatment he will make a complete recovery. This **suggestion** and **persuasion** (**psychotherapy**) must be repeated frequently and its manner must be modified according to the variations of the patient's condition. It must be done carefully and tactfully. By doing so the patient will gain confidence and a new orientation of the mental processes will thus be brought about with

the greatest facility. All that was said of the psychotherapeutic procedures in the chapter on Psychasthenia is applicable to Hysteria. As to Psychoanalysis, it was mentioned that Freud considers Hysteria as a psychoneurosis and therefore the psychoanalytical method finds its proper place in treatment of Hysteria.

It is perfectly legitimate to employ some physical means in order to reinforce the ordinary suggestion. For example, a mild electrical current applied to the neck in case of aphonia or globus hystericus, to the muscles of a contractured segment of a limb may give excellent results. In such cases the electricity does not possess a special curvative power, but acts as a suggestive agent.

Simple manipulations of the affected region when administered regularly, so as to impress the patient as if it were a necessary procedure, may yield some results. Hysterical backache, headache, neuralgia of long standing have been cured by application of liniments, water-bags, etc.

Hypnosis, which at a certain period reigned in treatment of functional nervous diseases, should **never** be employed. Hypnotic sleep exaggerates the suggestibility of the individual and practically induces hysteria and consequently aggravates the preëxisting hysterical state. It should be totally abandoned. When the hysterical phenomena are marked, **isolation** should be practised in conjunction with suggestive treatment.

Removal of the patients from their usual surroundings has given me excellent results. The patients gradually get accustomed to their new life, become obedient and fall entirely under the physician's control. This method gives the patient a simple, quiet and regular life; all sources of irritation are thus removed. In cases of marked emotionality, restlessness, tremor and spasms, **rest in bed** is very beneficial. Massage, hydrotherapy will then be excellent adjuvants in preventing wasting and sluggish digestive function (Weir-Mitchell's plan).

Medications will be administered only in cases of special indications. Restlessness, undue emotionality (crying, laughing, etc.) can be combated by bromides and tepid baths. For insomnia small doses of veronal or bromides can be given. When the patient gets accustomed to the drug and expects his capsule at night, veronal should be substituted by some placebo, as bicarbonate of soda, or plain flour. Gastro-intestinal disturbances should be treated accordingly. A hysterical paroxysm may be prevented by sprinkling cold water on the face or by inhalation of a few drops of ether. In one of my female patients I succeeded in checking a paroxysm by an application of a faradic current to the left hand. In another of my male cases the patient regained consciousness after a slight pressure was exercised upon the left hypochondrium.

Speaking generally, very little medication is necessary in treatment of hysteria.

To sum up, rest, tonifying measures, isolation, suggestion in a waking state (not hypnosis), persistent efforts to remove fixed ideas, which are so characteristic of the disease by persuasion or psychoanalysis—this is the main treatment of hysteria.

EPILEPSY

Epilepsy is characterized by a sudden loss of consciousness with (or without) convulsions.

Forms.— I. Focal or Jacksonian Epilepsy.
II. Major Epilepsy (Grand Mal).
III. Minor Epilepsy (Petit Mal).
IV. Equivalents of Epileptic Attacks.

I. Focal or Jacksonian Epilepsy

See description in Diseases of the Brain.

II. Major Epilepsy (Grand Mal)

Essential or Idiopathic Epilepsy.

Symptoms.—The disease is essentially **paroxysmal.**

A seizure is usually preceded by **prodromal** symptoms or **auræ** (warnings). The latter may be of **long** or **short** duration, may be of motor, sensory, psychic, vaso-motor or visceral nature.

A **motor aura** consists of tremor, of twitching affecting one or several muscular groups, vertigo, of automatic movements, as running, striking, etc.

A **sensory aura** may present itself as an epigastric pain, headache, sensation of cold or heat, of numbness, of tingling (usually in the limb in which the convulsion will commence), sensation of a vapor. Olfactory and gustatory, visual and auditory disturbances may also constitute an aura. The **"dreamy state"** described by H. Jackson as uncinate fits is accompanied by a smacking of the lips which suggests a gustatory phenomenon. "Sudden darkness" is a frequent visual aura, special colors, images may also appear. Sounds of a whistle, bell or of a voice—are the auditory auræ.

A **psychic aura** is particularly interesting, as it shows the rôle of the brain in the causation of epileptic seizures. It may consist of a delirious state, of a depression or else of gaiety, of terror, of sudden appearance of old images, etc.

Vasomotor aura presents itself as a sudden pallor or else redness of the face, perspiration.

Visceral auræ are: cardiac pain, palpitation, dyspnœa, laryngeal spasm, nausea, vomiting, cramps. Epigastric aura is one of the most common. It may be a pain associated with nausea, which rapidly ascends to the throat or to the head and is followed by loss of consciousness.

Cephalic aura consists of a sensation of "fulness" or "rush of blood" in the head. It is accompanied by giddiness. Auræ do not always precede an attack. The latter may come on suddenly without the least premonitory symptoms. In **exceptional** cases an attack may be prevented or checked at the very onset by certain manipulations as, for example, by tightly constricting the wrist. In such cases the patient carries a small girdle on the wrist with a string attached to it. As soon as an aura appears, the patient immediately pulls at it; constriction is then produced. One of my patients could inhibit an attack by putting some food in his mouth as soon as his aura (pain in the epigastrium) appeared. It may be arrested also by strong sensory impressions, such as smelling strong odors, or a splash of cold water on the face. Sometimes inhalation of amyl-nitrite is sufficient.

The seizure itself is characterized by the following symptoms.

The patient becomes **pale, screams** and falls **unconscious.** Immediately **tonic convulsions** appear. The entire body becomes **rigid.** The head is turned backward or to one side. The arms and legs are extended, the hands are closed and the thumbs adducted. The trunk is immobile. The eyeballs are bulging and turned, the pupils dilated. The face, which was pale at the beginning, becomes bluish and bloated. The tongue if protruded between the tight jaws bleeds. Involuntary evacuation of urine frequently occurs, probably because of a convulsive spasm of the walls of the bladder. The disturbance of circulation (venous stasis) produces sometimes ecchymoses and epistaxis. The duration of the tetanic phase is about half a minute.

The following phase, which lasts from a half to several minutes, is characterized by **clonic convulsions.** Here the rigidity is replaced by convulsive seizures. The latter rapidly follow each other, are abrupt and irregular. The muscles of the entire body twitch. The head turns from side to side, the grimaces of the face are frightful. The cyanosis disappears as the breathing becomes less difficult. Saliva froths at the mouth. The alternative closure and opening of the jaws cause the tongue to be bitten. The eyeballs turn rapidly in all directions. The body is covered with perspiration. The muscular contractions of the limbs may be so violent that dislocations occur. Gradually the twitchings subside and the patient,

exhausted, enters the third stage of the paroxysms. In the majority of cases he falls asleep. The sleep is profound and may last several hours. Awakened he has no recollection of his attack. Sometimes the **amnesia** covers a period of several hours preceding the attack (retrograde amnesia). Sleep does not always terminate an attack. The patient then opens his eyes, is confused and remains so for some time. In exceptional cases the patient in sleep turns on his face and as the sleep is very deep, asphyxia may occur.

Certain **objective phenomena** usually **accompany** or **follow** an epileptic seizure.

The temperature rises during an attack, but not above 0.5°. The pupils are dilated during the tetanic phase, but myotic in the subsequent phase. The pupillary reflex is abolished. The tendon and cutaneous reflexes are exaggerated. I have observed frequently increased knee-jerks, also sometimes ankle-clonus and Babinski sign, but paradoxical reflex is met more frequently than the latter. This state of tendon reaction is continued for some time after the attack. Relaxation of anal sphincter is frequent during a seizure.

Paralytic phenomena sometimes **follow** an attack. They are the result of cerebral exhaustion. Hemiplegia, monoplegia, asphasia may occur, but they are usually of a transient nature. Concentric contraction of the visual field and ocular palsies are occasionally observed. The general nutrition usually suffers, especially when the attacks are frequent. Phosphaturia and albuminuria are not infrequent. The toxicity of urine is diminished before and during the attack, but increased after an attack. The toxicity of the blood serum is increased during an attack and the toxicity of the cerebro-spinal fluid is increased after an attack.

The mental depression following an epileptic seizure presents variations from a slight hebetude to stupor. It increases in intensity when the attacks are frequent. In the latter case a feebleness of the mental faculties develops gradually and if the attacks are not kept under control, a true and **permanent dementia** will ensue. In children frequently repeated epileptic seizures will gravely interfere with the intellectual development and **mental arrest** will be the result.

Status Epilepticus.—Under this term is understood a series of attacks which rapidly follow each other without the patient regaining consciousness. This form of epilepsy is very grave. Death may occur during the convulsive period or during the stage of stupor. However the condition is not invariably fatal. When the intervals between individual series of attacks increase, the patient may regain consciousness and improve.

Atypical Epileptic Seizures.—An epileptic attack as described above does not always present itself in its typical form. It varies in different individuals and even in the same individual. The phase of tonic convulsions, for example, may be extremely brief and this phase may represent the entire attack. In other cases the **clonic phase** may be unusually prolonged even as long as 15 minutes. The clonic contractions may be of wider range than in typical epilepsy and they may give the impression of a hysterical paroxysm; they are therefore called **"hysteroid."** They usually alternate with typical epileptic convulsions, but when they do not, the diagnosis between hysteria and epilepsy is very difficult and remains necessarily in suspense until a classical attack occurs.

There are cases in which clonic convulsions may not affect the entire body. When the **tonic phase alone** exists, the convulsive movements pass into muscular relaxation without the clonic spasms. It is a serious condition: symptoms of asphyxia are marked toward the end of the attack. The rigidity of various muscles is quite pronounced: tetanic contraction of the jaws and opisthotonos may be observed. In some rare cases the attack may consist only of a sudden loss of consciousness with falling but without convulsions. This is the so-called **apoplectic form** of epilepsy. Usually such attacks alternate with convulsive attacks.

In spite of the fact that loss of consciousness and inability to recall the attack are characteristic of epilepsy, nevertheless there are caess in which the individual is conscious of what is going on during an attack but forgets after the attack is over, also are there cases in which consciousness is preserved as well as memory after the attack. I have seen cases in which patients witnessed the attacks. Such cases may be taken for hysteria if they do not alternate with typical seizures. They are called **hysteroid.** There are other atypical occurrences among varieties of epilepsy. The initial cry and the biting of the tongue may be absent. In some cases the seizures occur only at night and therefore are not recognized until an occasional bleeding of the tongue or an involuntary micturition attracts attention.

The deviations from the regular type of epilepsy may be multiple, but what characterizes all the typical varieties of epilepsy is the total inability of the patient to realize what occurred during an attack when consciousness is regained.

III. Minor Epilepsy (Petit Mal)

It is characterized by a sudden loss of consciousness lasting but a few seconds. In the midst of a conversation, of reading, of playing an in-

strument, of eating, etc., the patient suddenly becomes pale, interrupts the act he is doing and loses consciousness. In a few seconds the attack is over and he resumes his work. Like in major epilepsy there is total amnesia of the attack after consciousness is regained. The attack is usually so brief that the patient does not fall. In a great many cases some sensation precedes the attack, such as sudden light or appearance of colors.

Petit mal may present itself in various forms. In some patients an attack is noticed from sudden staring of the eyes. Others are taken with a sudden vertigo of a very short duration. One of my patients would be taken several times a day with a sudden inability to realize where he was.

IV. Epileptic Equivalents

They consist of motor, sensory, visceral and psychic manifestations which are sudden in onset and accompanied by total loss of consciousness.

To this group belongs the so-called **ambulatory** form of epilepsy. The patient thus affected usually without and exceptionally with a few premonitory symptoms, as headache, depression, etc., suddenly leaves his home and automatically wanders away; sometimes he gets on the train and travels hours or days. Suddenly he realizes his situation and is astonished to find himself removed from his home. Similarly to genuine epilepsy there is total **amnesia** of the accomplished act. In another form of the disease there is an irresistible desire for **sleep.** It comes on suddenly and in attacks. It may terminate with a mild delirium.

Equivalents of epilepsy are also seen in a sudden irresistible desire to do unusual acts. One of my patients had attacks in which he would suddenly undress himself. Another, a clerk, would suddenly unbutton his trousers in his office and urinate.

Various subjective sensations or hallucinations, visceral disturbances, as pain in the abdomen, sudden desire for defecation, attacks of angina pectoris, of migraine, of syncope, of asthma, may be considered as equivalents of epilepsy when their onset is sudden, their duration is short and when there are no organic visceral changes that could account for the disturbance.

The so-called **psychic epilepsy** belongs to the same group. The patient thus affected is suddenly taken with a desire to do the most absurd and impossible acts. Sometimes he attacks, injures or kills. Not infrequently he is prompted in his actions by various auditory or visual hallucinations. The seizure terminates as suddenly as it began. Sometimes it is followed by a state of exhaustion or depression. In some cases the onset of the attack is preceded by headache, undue irritability, depres-

sion. The characteristic feature of the seizure is the complete inability of the patient to recall a single act committed by him during the attack.

Pavor Nocturnus or Night Terrors.—In **adults** the condition occurs in the beginning of sleep. Suddenly the body is seized with a very pronounced jerk and the patient at once sits up. He wakes up, is frightened and the heart beats rapidly. Considering the fact that individuals suffering from this manifestation are usually epileptics, the relation of it to epilepsy becomes evident.

In **children** the same sudden onset with the state of anxiety is present but in addition to it there are also frightful hallucinations with amnesia. The latter manifestations are assuredly analogous to some equivalents of epilepsy mentioned above. There are cases on record pointing to the fact that night terrors are in reality attacks of petit mal, also that they eventually are substituted by genuine attacks of epilepsy.

Epileptic Character.—Epileptic individuals very frequently present, certain peculiarities of character which deserve to be mentioned. **Irritability,** quite pronounced, is characteristic. Anger is brought out upon the least provocation. With it **maliciousness** is frequently exhibited even toward his own people. **Obstinacy** is another feature; it is quite pronounced. It frequently accompanies a **morbid egotism.** This leads to excessive **impulsiveness** in actions and words; it is exhibited even in the most insignificant occurrences. The obstinacy naturally shows **deficient judgment** and illogicality. **Moral sentiments** are also at fault. The morbid egotism mentioned above develops an unforgiving spirit toward those who happened to injure them even slightly. A certain **revengefulness** and even **cruelty** are not infrequently noticed in the epileptics in their relation to others.

Course, Termination, Prognosis.—There are great variations in the course and duration of epilepsy. Some patients may have several seizures daily, others only one attack in weeks or months; some only during the night, others during the day. Petit mal attacks occur only during waking hours. Some epileptics have only attacks of grand mal, others only of petit mal and still others have both forms. Intervening diseases sometimes arrest the seizures. Gastro-intestinal disturbances have a considerable influence upon the frequency of the attacks. Constipation is frequently followed by seizures. Use of stimulants and excesses of any kind increase the intensity of individual attacks as well as their frequency.

Menstruation may have some relation to attacks of epilepsy. In a certain group of cases the seizures occur only around the time of menstruating periods. Perhaps the disturbance in the internal secretion of the

ovaries plays a certain direct or indirect rôle on the cerebral irritation thus producing convulsions.

Oncoming puberty, which ordinarily is associated with physiological disturbances in the organism, usually increases the severity or frequency of epileptic seizures. Sometimes epilepsy makes its first appearance at puberty.

Not infrequently the disease begins with attacks of petit mal which gradually may pass into major seizures. Sometimes both forms coexist.

When the disease commences early in infancy, it has a slight tendency to disappear at the age of ten. The same tendency is also observed in middle life.

Life is not directly threatened by epilepsy, except when injuries occur in falling. Status epilepticus is very dangerous to life. In the majority of cases epilepsy is not curable. Recoveries follow very occasionally. The disease may last many years. Petit mal is more obstinate and less easily influenced by treatment than grand mal. Mental failure frequently accompanies the advanced cases of epilepsy and in such cases dementia is the ultimate outcome. Epilepsy beginning in early life interferes with the mental development and imbecility is frequent.

Alcoholic and syphilitic epilepsy are sometimes amenable to treatment and present a favorable prognosis.

Diagnosis.—In the majority of cases the individual phenomena of an epileptic seizure are so typical that there is no difficulty in making a diagnosis. In the atypical or irregular forms (see above) the symptoms may simulate other affections.

A **hysterical** paroxysm is recognized by the following symptoms. It never occurs at night during sleep. Its onset is not sudden and not accompanied by an initial cry. There is no biting of the tongue and no involuntary loss of urine or feces. The muscular twitchings are of wide range, and they possess a "purposive" character. The pupillary reflexes are intact. The attack does not terminate by stupor or coma, but by an outbreak of laughing or crying. If sleep occurs, it is superficial and not as profound as in epilepsy. Mental failure or dementia, which is observed in epilepsy of long standing, is not present in hysteria. Hysterical attacks are amenable to treatment sometimes by suggestion.

Uremic convulsion occurs without an initial cry or without an aura. There is no biting of the tongue. The urinary examination will show quantitative and qualitative changes characteristic of diseases of the kidneys.

Epileptiform convulsions may occur in cases with increased intracranial pressure, as tumors, meningitis, etc. They may also occur in

the course of paresis. It is therefore important to eliminate organic diseases in every case presenting a history of convulsions.

Attacks of petit mal present no diagnostic difficulty from their sudden onset, sudden termination, extremely brief and absolute loss of consciousness.

The **psychic form of epilepsy** presents at times great difficulties, as irresistible impulses are observed in the course of psychoses accompanied by delusions and hallucinations. The diagnosis will be made by exclusion.

The **equivalents of epilepsy** (see above), as attacks of vertigo, syncope, angina pectoris, etc., will be easily recognized by eliminating visceral diseases (labyrinth, heart, etc.). In aural vertigo there is no loss of consciousness. In syncope which usually follows an emotion or occurs in an overheated room loss of consciousness takes place, but when the latter is regained, it is perfect from the beginning. The same is not observed in epilepsy. Gowers believes that repeated attacks of syncope which are due to cardiovascular changes with alteration in the cerebral circulation produce a lessened resistance of the nervous mechanism of the cardiovascular apparatus, the reflex becomes easier and loss of consciousness more sudden. He believes that a change in nerve elements of the brain occurs in syncope and by virtue of frequent repetition of the attacks, the state of nerve elements acquires a tendency to spontaneous development which is characteristic of petit mal. Thus an ordinary faint or syncope may eventually develop into minor epilepsy.

Pathogenesis of Epilepsy.—Post-mortem examinations have given nothing definite as to a possible organic cause of essential epilepsy. In spite of this fact experimental investigations (Fritch, Hitzig, Charcot, François-Frank and others) have shown that convulsions are essentially a **cortical** phenomenon. An irritation of any area of the cerebral cortex, but particularly of the motor area, produces convulsive seizures. François-Frank has also shown that cortical irritation is followed by vascular spasm, by changes in cardiac rhythm, by dilatation of pupils, by incontinence of urine and feces; otherwise speaking, by the symptom-group observed during an epileptic seizure.

It is now generally conceded that the point of departure of a convulsive attack or of petit mal attack lies in the cortex, which at that moment undergoes an undue irritation. Various factors are supposed to cause this irritation. Anæmia, hyperæmia of the brain, toxic substances originating in the organism (autointoxication) or introduced from without (alcohol, etc.) are considered the immediate exciting factors capable of producing a cortical discharge in the form of **grand mal, petit mal** or **equivalent of epilepsy.**

In favor of the cortical origin of epilepsy speaks also the fact that mental feebleness and dementia develop in the course of the disease. The toxic nature of essential epilepsy is corroborated by the influence of gastro-intestinal disturbances upon the intensity and frequency of its manifestations, by the degree of toxicity of the urine and of the cerebro-spinal fluid before and after the attacks. Not infrequently at autopsy of epileptics old meningeal or cortico-meningeal lesions are found. Such individuals may have had attacks in their younger years which gradually dis-disappeared or else occurred since at very rare intervals. Recurrences in such cases take place à propos of an autointoxication (gastro-intestinal, renal, glandular). Experiments on animals show that an intoxication which is without an effect on a normal animal is a cause of epileptic seizures in an animal presenting old cortico-meningeal lesions (Claude and Lejonne, *Comptes-rendus de Soc. de Biologie*, 1910). Guidi (*Rivista sperim. di Friniatria*, 1908), has also shown experimentally that there are profound metabolic changes in epileptics: transformation of proteids is not like in normal individuals; the most manifest deviation is seen in the production of urea, hence an acid intoxication takes place; excess of ammoniacal compounds is found in the urine of epileptics. Removal of these chemical compounds by an appropriate diet reduces the number and intensity of seizures.

Etiology.—The causes of epilepsy are **predisposing** and **exciting.** A neuropathic heredity (epilepsy, other neuroses, insanity in the family), consanguinity, hereditary alcoholism, syphilis, lead poisoning, are all predisposing factors. The influence of heredity is considerable. The exciting causes are: **infections, intoxications, traumata.** Among infectious diseases scarlet fever, typhoid fever, measles and smallpox play a considerable röle. Gastro-intestinal disturbances are a common cause for epileptic convulsions. Alcohol taken in large doses and for a prolonged period of time is a potent factor in epilepsy. It is possible, however, that arterial degeneration with the rise of blood pressure are apt to produce cortical irritation and therefore epileptiform attacks in aged individuals (**Senile Epilepsy**). Among other toxic elements capable of causing epilepsy can be mentioned: lead, mercury, cocain, morphin, chloroform, ether.

Syphilis may produce epilepsy without definite anatomical lesions, especially during the secondary period.

Gout, diabetes, anæmia, cardiac diseases and circulatory disturbances in general are also considered as causes of epilepsy, but their relation to the disease is not entirely elucidated. Alcohol may be an exciting cause for attacks in an epileptic individual or *per se* be a factor in development of

epilepsy in a neuropathic individual. In chronic alcoholic persons who used alcohol to excess for a very prolonged period, withdrawal of alcohol is likely to bring on epileptic seizures. This is observed particularly in asylums. **Traumata** may cause epilepsy either through direct injury of cerebral tissue or only in cases of concussion without apparent lesions. In considering trauma as a cause it should be borne in mind that the symptoms of epilepsy may appear shortly after the accident or a long time later, as late as ten, fifteen or twenty years. Peripheral injuries, viz. of an eye, ear, nasal fossæ, of a nerve, may be the direct cause of convulsive seizures.

The relation of epilepsy to visceral diseases, as uterus, intestinal worms, polyps in the nose or ear, errors of refraction, etc., is maintained by some observers, but not definitely proven. These cases are the so-called **"Reflex Epilepsy."** A large number of cases date from infancy. Infantile convulsions or petit mal occur frequently during the period of dentition. If they persist, they become distinctly epileptiform at puberty or even earlier. Infantile convulsions may cause permanent hemiplegia.

Epilepsy may occur at any age, but in the majority of cases between the age of ten and twenty. There are cases in which epilepsy occurs only during or immediately before the menstrual periods. There are also cases in which epilepsy begins only at puberty. The period of menopause has sometimes a favorable influence on epilepsy. As a rule the disease is rare in old age, but senile epilepsy is well known.

Treatment.—In the chapter on the Pathogenesis of Epilepsy cortical irritation was considered as the underlying immediate cause of the epileptic discharge. The main indications therefore are: (1) Removal of factors producing cortical excitability and (2) modification of the latter when it exists.

1. The first is by far the most important. An effort must be made to place the patient in such conditions as to reduce to a minimum auto-intoxication. This can be accomplished by an appropriate régime and hygienic rules. It has been my experience that removal of **meat** from the diet is very beneficial. Starchy food and sweets should be avoided or at least considerably reduced in quantity. The patient should be instructed to have his meals at regular hours and not to take food between the meals. The latter should consist of milk, soft-boiled eggs, vegetables, fruit. These articles can be combined in such a manner as to suit the patient's taste and afford variations in the meals. Stimulants, also tea and coffee, should be avoided. The amount of food for each meal should not be abundant. The mastication must be thorough and the eating should be slow.

As I have shown with other observers (*New York Med. Jour.*, 1906), **table salt** is to be avoided entirely or taken in a very small amount. I have found that there is a decided relation between the intensity and the frequency of the attacks and the amount of table salts used with food. In order to avoid aversion for saltless food a gradual reduction of the amount of salt is recommended. Experimentally it has been shown (Paderi, *Archivio di Farm. sperim. e Scienze affin.*, 1911) that the effect of bromides on the organism is greater and more rapid when the diet is salt-free.

Proper **elimination** plays a prominent rôle. A purgative administered regularly once a week and oftener, if there is a tendency to constipation, is a necessity. Special emphasis should be laid on regular bowel movement, as there is almost a direct relationship between constipation and epileptic attacks. All sources of autointoxication must be removed. The patient is also instructed to keep his mouth and teeth in as perfect condition as possible.

The mode of living, occupation, sleep, etc., must be well regulated. The patient must go to bed early and get up late, so as to get a great deal of rest. An hour's sleep during the day is also desirable. He must take two or three walks a day, each of such duration as not to feel fatigued. Fatigue as a rule must be avoided. All sporty exercises are forbidden. The occupation must be of such a nature as to give a minimum of mental or physical strain. Crowded places, as theatre, church, etc., must be avoided. While complete seclusion is not advisable because of the mental depression it leads to, entertainments, society gatherings, games, etc., should be avoided as much as possible. Hydrotherapy is useful. Shower baths, warm or, better, cold, of half a minute's duration and followed by a gentle general massage—once or twice a day—are very beneficial. A sojourn in the country several times during the year is advisable. If the patient is a young boy or girl, they shouid be kept out of school. Mental work in general should be done as little as possible. Sexual intercourse should be avoided. Marriage is not to be advised, first because of the probability of sexual excesses, next in view of the offspring which, as statistics show, in the majority of cases inherit a neuroyathic tendency. Imbeciles, idiots and otherwise mentally deficient have come from parents suffering from epilepsy.

This is the régime to which I usually submit my patients suffering from essential epilepsy.

Careful examination should be made in regard to local irritation, as polyps of the nasal cavities, to errors of refraction, to intestinal parasites, to scars, to injured peripheral nerves, to visceral diseases. Correction or

removal of these factors should be made as early as possible in the course of the disease. Incases of toxic origin rapid elimination of toxic products must be insisted on. In cases of traumatic epilepsy a thorough search should be made for any depression of the cranium or for a localized pachymeningitis. Surgical intervention is of course necessary in such conditions. In traumatic epilepsy of long standing operative procedures should be undertaken. An effort should be made to localize the lesion. A Roentgen-ray examination should be of considerable assistance in such cases.

In absence of any localizing cause on the cranium or at the periphery **surgical procedures** should be undertaken in idiopathic epilepsy with great hesitation. When the seizures are rare and slight, also when medications improve the condition, no operation should be thought of. In grave cases which are rebellious to medical treatment and which by virtue of the intensity and frequency of the attacks endanger life, surgical intervention may be considered. Trephining and osteoplastic operations have been performed but without permanent success. Recurrences of attacks is the rule, although amelioration has been obtained in some cases. In one of my cases I had an osteoplastic operation performed over the motor area of one side and six months later over the other side. Considerable improvement followed not only with regard to the frequency of the seizures, but also the girl (twelve years of age) began to show considerable mental progress.

Operations on the cervical sympathetic nerve have been advised by Chipault and Jonneseo, but the results so far obtained do not apparently justify their use. Giacomelli (*Gaz. degli Osped. e delle Clin.*, 1912) recommends for essential epilepsy intra-spinal injections of stovain. After withdrawing by lumbar puncture about 8 c.c. of cerebro-spinal fluid he injects 0.08 grm. of stovain diluted in 1 c.c. of physiological solution of chloride of sodium. In one of his cases there was complete recovery, in other cases a considerable reduction of the number of seizures and of their intensity followed.

2. **Cortical excitability** will as a rule be diminished by the just outlined method of general management. However in some cases the latter may not be sufficient. Medications are very frequently necessary. **Bromides** is the most efficient remedy according to the majority of observers and among all the salts **sodium bromide** is the best tolerated by the patients. Its doses are given according to the age. I have had better results in giving small doses frequently repeated than large doses once or twice a day. I usually prescribe for an adult at the beginning gr. x every three hours and at the end of three or four days every two hours. If the attacks

are not controlled, the patient is told to take it every hour. When an improvement is noticed at the end of a few weeks, the same amount is given only every four hours. An excellent adjuvant to the bromides is one of the coal-tar products, and I always combine sodium bromide with antipyrin in gr. v doses. This treatment must be kept up for several months. If **bromide acne** appears, the drug must be discontinued for a few days and substituted by sulfonal or trional. The acne can be relieved considerably by administration of arsenic in two, three, or four drops of Fowler's solution. It should be continued as long as bromides are given. In the majority of my cases I persist with the bromides in spite of the acne and no special ill effects have I noticed. In severe cases the gr. x dose every two or three hours may not be sufficient. Fifteen or twenty grains may then be given. Bromides sometimes produce a marked general depression with impairment of memory, but the latter are only temporary phenomena. On the contrary, in a number of instances in my experience a prolonged administration of bromides was followed by a very marked lucidity of mind because the drug succeeded in removing the seizures for long intervals. The so-called bromide dementia has never been observed by me.

When a marked intolerance is noticed (which is exceptional) bromides may be substituted by other drugs, as extract of opium in doses not above fifteen grains a day, extract of solanum carolinensis in doses of from fifteen to sixty drops, adonis vernalis, digitalis, atropine, belladonna, camphor, valerian. Any of these drugs can be given conjointly with or in place of bromides. It should be, however, borne in mind that bromides are the most efficacious of all the drugs mentioned.

When a syphilitic history is present, mercurials and iodides are indicated.

Iodides are also useful in cases with a history of lead poisoning.

There is a certain class of epileptics which I found can be benefited by the administration of thyroid extract. These individuals show signs of hypothyroidization. I made a special study of them (*Therap. Gazette*, 1907) and the entire series of my patients benefited considerably from this medication. The epileptic seizures became less and less frequent and the mental condition improved to a remarkable degree.

Leubuscher (*Deut. Medi. Wchn.* No. 11, 1913) advocates administration of phosphorous in view of its good effect in tetany. He gives it in 10,000 parts oil. In his cases he obtained considerable reduction of the number of seizures.

The treatment of epilepsy must be kept up in the most rigorous and persistent manner and efforts be made to control the attacks. The

disease itself has a very deleterious effect upon the mental condition of the patient. In children it interferes with their intellectual growth. Mental arrest follows if the intervals between the seizures are not lengthened. When it is difficult to carry out the above outlined treatment concerning the general condition of the patient it is advisable to place him in a special institution for epileptics (colonies or state asylum).

Treatment during an Epileptic Seizure.—In the chapter on symptomatology mention was made of cases in which an attack can be cut short by quick constriction of a limb when the aura appears in that limb. I also mentioned a patient whose seizure was arrested at the very onset as soon as he put some food in his mouth. An attack can also be checked by inhalation of a few drops of amyl nitrite. If in spite of these means the attack continues, it is advisable not to interfere with it except in so far as to watch that no injury should occur to the patient when he falls. In view of the disturbed circulation, it is urgent to loosen the clothes, collars, neckties and the end of a folded towel, of handkerchief or else a piece of wood be placed between the jaws so as to avoid biting of the tongue. When sleep supervenes, the patient should not be awakened, as otherwise the post-epileptic depression increases.

CHOREA (OF SYDENHAM)

(St. Vitus Dance)

Symptoms.—The onset may be **rapid** or **gradual.** The former follows usually a severe shock. In the majority of cases the symptoms develop gradually. A few prodromal symptoms precede the appearance of the characteristic symptoms. The patient (who is usually a child) becomes irritable, morose, inattentive. Gradually he gets restless and awkward movements are noticed in the arms and legs. Objects fall out of his hands, grimaces are noticed on his face.

The characteristic choreic symptoms may begin in one leg or in one arm. They soon become generalized. The patient's musculature is continuously contracting. The movements are **involuntary, irregular** and **incoördinate.** When the upper extremity is affected, he is unable to take hold of an object and keep it for a certain length of time, is unable to feed himself, to write. In attempt to approach his hand to an object, a series of various incoherent movements will be produced before the hand reaches it. The fingers separate, approach, flex, extend. The entire limb supinates, pronates, is abducted or adducted. The shoulder is raised, lowered, pushed backward or forward. The leg is in constant motion, moves in every direction when the patient is at rest. The toes

flex, extend, the foot turns inward, outward, the legs bend or extend. When seated the patient crosses his knees, approaches or separates them. The gait is also irregular. The face is continuously agitated, so that various expressions are assumed by the patient. He closes and opens his eyes, rolls the eyes in every direction, the head rotates, the lips pout. Continuous contraction of individual muscles of the face and forehead is noticed one after another. The tongue is continuously moving from side to side, forward and backward. The speech is disturbed because of irregular contractions of the respiratory muscles, especially of the diaphragm. The articulation of words, emission of sounds, is difficult. Ziemsen observed with the laryngoscope irregular movements of the vocal cords. When the muscles of the palate and pharynx are affected, deglutition is difficult. The muscles of the trunk and pelvis participate in the movements.

To sum up, the muscles of the entire body are in a state of frequently interrupted involuntary contractions, which are **rapid, irregular, purposeless.**

Voluntary movements increase the contractions, although a reverse condition may also be observed. Emotion, excitement, may increase the twitchings, but sometimes they have an inhibitory effect. In writing the following conditions may occur: (1) The power of control over the movements may be complete. (2) General choreic movements may be very slight or else much incoördination may be present.

The movements usually disappear during sleep.

Sensory symptoms may be present in the form of paræsthesiæ and even tenderness of the muscles. Diminished objective sensibility is quite common.

Chorea is frequently accompanied by the following symptoms.

The reflexes may be normal, increased or diminished.

W. Gordon described the following phenomenon. When, the patient being in dorsal position, a short blow is struck over the patellar tendon, the leg will at first respond like in an ordinary knee-jerk, but instead of coming down immediately after, will remain suspended in the air some time and gradually come down. It is probably due to a prolonged contraction of the quadriceps muscle. This reflex is not absolutely constant, but it has never been observed outside of genuine chorea.

The pupils are often dilated. Inequality of pupils and hippus has been observed. Occasionally optic neuritis is met with, a fact which led to the view that acute chorea is of infective origin.

The pulse is rapid and quite frequently a mitral lesion is present.

Urea is increased. Phosphaturia is present.

The mental faculties are sometimes involved. Mental dulness, diminished attention, weakness of memory, excitability or else depression are not infrequent. In exceptional cases a delirium with confusion and hallucinations may develop. In some cases the mental symptoms may become permanent and develop into dementia.

The general nutrition suffers when the muscles of deglutition are involved.

Forms of Chorea.—Sydenham's chorea may be pronounced and very slight. Between these extreme forms there are many intermediary forms. It may affect one side of the body and in then called **hemichorea.** When there is a marked weakness or a paretic condition of the extremities, it is called **paralytic chorea.**

The loss of power and of voluntary movements in **paralytic chorea** may be generalized or confined to one or two extremities (mono-, hemi-, or paraplegic forms). The paralysis is flaccid and there is no involvement of reflexes, of sensations or of sphincters. The prognosis is good.

In the pronounced or **grave** variety of chorea the twitchings are so intense that the patient is obliged to be in bed. I have seen patients whose movements were so violent that they were thrown out of bed. In such cases walking is impossible. The agitation continues even during sleep. The violent muscular contractions lead to traumata and such a patient is often covered with ulcerations. Mental disturbances frequently accompany grave chorea. Death is the usual termination.

Chorea of pregnancy occurs usually in primiparæ during the first half of pregnancy. It is a grave affection, as the muscular twitchings are very severe and interfere with sleep. It is frequently complicated by cardiac diseases with fever, also mental disturbances. The gravity of this form of chorea lies also in the spontaneous interruption of pregnancy.

Course, Termination, Prognosis.—The average case of Sydenham's chorea lasts about one or two months. Periods of amelioration and aggravation are observed in the course of the disease. Recovery is the usual result. The younger the child the better is the prognosis. Chorea occurring in youths presents a serious outlook, as it lasts longer and has a tendency to become chronic. The **grave** form of chorea has an unfavorable prognosis: death is due either to exhaustion or to the mental symptoms. The more marked the latter are, the more serious the outlook is.

Chorea of pregnancy is a serious malady (see above).

Recurrences are very frequent in chorea. After the patient has made apparently a complete recovery, a shock, an emotion, promptly brings on another attack. Some patients have every year an attack of chorea

during several years. Chorea is sometimes associated with other neuroses, viz. hysteria, epilepsy.

Diagnosis.—The **rapidity, irregularity** and **incoördination** of muscular contractions are sufficiently characteristic signs for diagnosis. Occasionally hesitation is experienced and the only affections with which chorea may sometimes be confounded are: tic, myoclonia, athetosis and Hysteria.

Tic and **Myoclonia** are recognized from the suddenness and instantaneity of twitchings which are confined **only** to a certain portion of the body. In tic the movements are coördinate and purposive, while in chorea they are distinctly incoördinate. The tic movements can be controlled to a certain extent, choreic movements cannot be controlled. The former are not disabling, the latter are.

In **Athetosis** the movements are slow and regular and affect mostly the fingers or toes. In **Hysteria** the movements are coördinate and they usually develop suddenly after an emotion or during an hysterical paroxysm.

Etiology.—Sydenham's chorea is a disease of young age and affects children from the age of seven to puberty. Girls are more predisposed than boys. It may, however, occur at any age. The **predisposing** causes are a neuropathic hereditary tendency which can be traced in the majority of cases, constitutional diseases (tuberculosis, etc.), anæmia or a debilitated state originating from any cause.

Infectious diseases, among which acute inflammatory rheumatism occupies the first place, are not infrequently accompanied or rather followed by chorea. Scarlet fever, measles, diphtheria, smallpox may be followed by chorea. In secondary syphilis chorea has been observed and the former may be the cause of the latter. Recently Milian (Bull. et Mém. de Soc. méd. d. hôp. Paris, 5 Déc., 1912) reported fifteen cases in which stigmata of hereditary syphilis were present. Moreover, in eight cases out of thirteen Wassermann reaction was positive. He calls attention to von Bokay's case in which Salvarsan cured the chorea. Rheumatism, however, is most frequently followed by chorea. The cardiac condition which is so frequently met with in chorea is probably due to the preëxisting rheumatic affection.

Pregnancy is an important factor in causation of chorea, especially in cases with a history of previous attacks (see above).

Fright, emotion, traumatism are frequently the immediate causes of chorea, especially in predisposed individuals. Predisposed children being in company of choreic ones may sometimes develop the disease by imitation.

Chorea is rarely observed in negroes.

Pathology.—Hyperemia, punctiform hemorrhages and perivascular alterations have been observed by some writers. Hudovernig (see below) found cellular changes analogous with those of anterior poliomyelitis, especially in thalamus opticus. Proliferation of connective tissue was observed in the spinal meninges. It is possible that these changes are due to a toxi-infectious process. There are also many observations on record with absolutely negative findings.

Bignami and Nazari (*Presse Méd.*, 1911) report two cases of hemichorea, in one of which was found a solitary tubercle in the left superior cerebellar peduncle, in the other a hemorrhagic focus in the same spot as in the first case.

Pathogenesis of Chorea.—The post-mortem investigations present nothing definite for the localization of the disease. There is a tendency at the present time to consider chorea as **infectious** in nature. The bacteriological works, especially of Pianese, favor this view. This author found in the spinal cord a bacillus with the cultures of which he made successful inoculations. Microörganisms in the brain were also found by other authors. Poynton and Paine isolated from the cerebrospinal fluid a diplococcus which after an inoculation into a rabbit produced muscular twitchings. The diplococcus was found in the pia-mater and brain in choreic patients and in the rabbits.

The cytological examination of the cerebro-spinal fluid showed in a number of instances a marked lymphocytosis. In favor of the infectious origin speaks also the occurrence of chorea with infectious diseases. I have seen cases in which chorea developed subsequently to a localized inflammatory focus; the twitchings appeared at the time of formation of pus. The choreic movements disappeared with the removal of the infectious focus (*J. Amer. Med. Ass'n.*, 1910). That acute chorea is due to an infectious agent is highly probable. In this respect it is analogous to acute anterior poliomyelitis with this difference, that in the latter the motor cells are being destroyed, hence the paralysis; in the former the motor cells of the cortex and of the nuclei of the medulla undergo irritation, hence the twitching. The destructive effect in the latter and the irritating effect of the former point to a difference in the virus in the two affections. Hudovernig's pathologic report (*Arch. f. Psych.*, 1903) shows a very great resemblance between the cellular changes of both affections. It must not be forgotten that the bacteriological investigations cannot as yet be accepted as absolutely conclusive.

Charcot's and Joffroy's views cannot be neglected. According to them there is an inherent degenerative predisposition of the motor apparatus, which is brought in evidence as soon as some special cause

disturbs the latter. This cause may be an acute inflammatory rheumatism or any infectious disease.

Treatment.—It was mentioned above that a neuropathic tendency is found in the majority of cases. The indication is therefore to improve the general condition of the patient by proper dietetic and hygienic measures. In cases of anemia, tuberculosis or other constitutional diseases an effort should be made to improve these conditions. In an average case of chorea factors leading to emotion, excitement or depression must be removed. Mental strain is contraindicated. The child should not be sent to school. The patient's life should be so regulated as to give him a sufficient amount of rest, good sleep, proper and nutritious food in moderate quantities. I am in a habit to remove meats and all stimulants, including tea and coffee. Elimination must be taken proper care of. An occasional purgative is beneficial. When the twitching is marked and does not show any tendency to improve, I keep the patient in bed. This procedure has given me excellent results in many obstinate cases. Cool spongings followed by a gentle massage have a sedative action on the patient's nervous system. In cases with violent twitchings the patient must be guarded against unjury and being thrown out of bed.

Among all the medications the most reliable ones are: **arsenic** and **antipyrin.** It is advisable to commence the treatment with the first. Its administration should begin with very small doses and only very gradually increased, as otherwise intolerance will be exhibited very early. An average child of ten should be given ℳiii t. i. d. of Fowler's solution during the first two or three days. If after two or three weeks of treatment no improvement is noticed, the arsenic should be substituted by antipyrin. To a child of the same age gr. j of the latter can be given every two hours. I have frequently obtained very good results with antipyrin when arsenic failed. When none of these drugs yields results, the patient should be kept in bed and given **bromides.** The latter may be given conjointly with arsenic.

When there is a history of rheumatism, sodium salicylate, aspirin or salophen give very satisfactory results. As to individual doses of all medicaments, they will have to be modified according to the age and the intensity of symptoms, but it is always wise to commence with very small doses. A very gradual increase in the amount is always preferable to a rapid increase, as it establishes a satisfactory tolerance.

The patient must be frequently observed, and with the first signs of physiological intolerance, the given drug must be immediately discontinued and in a day or two substituted by another.

Among other drugs **chloral hydrate** is to be recommended for controlling the twitching when other remedies fail.

In chorea of pregnancy artificial termination of pregnancy may be considered. The latter should be undertaken when the indications are strong, viz. when life is endangered by exhaustion, cardiac or renal lesions or mental disturbances.

The latest researches of Loeb and J. B. MacCallum, also of W. G. MacCallum and C. Voegtlin, show a certain relationship between various twitchings and calcium metabolism also the function of the parathyreoid glands. A trial of calcium salts and of parathyreoids in chorea is therefore indicated.

Finally Marinesco (*Semaine méd.*, 1908), advises the use of intraspinal injections of magnesium sulphate (25 per cent.). He withdraws first a certain amount of cerebro-spinal fluid and injects the same amount of the drug. The amount injected is 1 c.c. to each 25 pounds of bodily weight. Marinesco obtained very satisfactory results in every one of his cases. He advises against the use of this drug in grave cases, in chorea of pregnancy and in cases of chorea dependent on an organic disease of the central nervous system.

CHRONIC CHOREA

HEREDITARY CHOREA

(Huntington's Chorea)

This disease, which has **no relation** whatever to Sydenham's chorea, was known before Huntington, but the latter was the first to call special attention to three important elements of the affection, viz. **heredity, onset** at the age of thirty or forty and **mental** symptoms.

Symptoms.—The clinical picture differs little from that of Sydenham's chorea. Like in the latter, the movements are arhythmical, irregular, incoördinate. The onset is slow and the twitchings at first appear on the lower half of the face. Gradually they spread to the upper and lower extremities, also the trunk. When the muscles of the palate and pharynx become involved, the deglutition is difficult. The tongue is particularly affected, so that the speech becomes indistinct and nasal in tone. When the diaphragm is involved, the respiration is disturbed. The reflexes are exaggerated. Muscular weakness is usually present, but there is no atrophy; neither are there changes in the electrical reactions. Sensations are intact.

The distinguishing features of Huntington's chorea are:

1. It occurs in adult life.

2. The movements are slower and not as frequent as in Sydenham's chorea.

3. The muscles of the eyeglobes are usually not involved.

4. The upper part of the face is rarely affected.

5. The gait is characteristic. It is analogous to that of an inebriate. The patient makes a few rapid and awkward steps, then stops suddenly; leans forward, looks at the ground and then again advances with small steps. All this is done rapidly and with variation.

6. Voluntary effort may repress the twitchings, so that at that time the patient is able to execute delicate acts, as writing or threading a needle.

7. Rest decreases the intensity of the twitchings.

8. As the disease advances the mentality suffers. Extraordinary irritability is constant and is frequently one of the earliest symptoms. **Defective power of attention** in execution of physical as well as of mental acts is one of the most essential characteristics. Gradually the memory for recent and old events weakens and the conceptions become retarded. The patient is depressed and the intellectual faculties become feeble. There is a tendency to suicide. Dementia is the ultimate result. The mental phenomena as a rule follow the motor phenomena, but in some cases they precede.

Course, Termination, Prognosis.—The disease lasts many years, develops slowly, but it is essentially progressive. Death occurs either from disturbance of deglutition and respiration or from the extreme mental hebetude or else from some intercurrent disease.

Diagnosis.—The differential diagnosis with **Sydenham's** chorea is given above.

In **tic** the movements are abrupt and always the same.

Etiology.—In the majority of cases there is a direct heredity or a general neuropathic taint. There are cases on record in which several successive generations were affected with this disease. Huntington observed that when one member of an affected family escapes, his offspring are free from the disease. In other cases there may be a family history of epilepsy or hysteria.

Both sexes are equally affected. The disease occurs in adult life between thirty and forty. **Emotions** have a very important influence upon the development of the malady. Traumata and pregnancy are equally exciting causes.

Pathogenesis of Huntington's Chorea.—Post-mortem examinations show an anatomical basis of the disease. Atrophy of the cortex and espe-

cially of the motor area, thickening and adhesions of the meninges, diffuse meningoencephalitis, have been found. Microscopically disseminated foci of round cells in the cortex and white matter have been seen quite frequently. Whether a parenchymatous degeneration of the neurons or a vascular alteration is the primary condition, it is difficult to say. There is a possibility of hereditary malformation of the central nervous system. The relation between the anatomical findings and the clinical picture of the disease is not entirely established.

Treatment.—When treated early the patients may derive some benefit from good hygienic and dietetic measures, from bromides, arsenic, chloral, antipyrin. As a rule all these means are only palliative. The disease is progressive and incurable.

ATHETOSIS

It is characterized by **continuous, slow, involuntary** movements, mostly of **fingers and toes,** occurring even during sleep.

Unilateral Athetosis (**Hemiathetosis**) stands in close relation to hemiplegia (see this chapter).

Bilateral Athetosis.

This is mostly a congenital condition appearing in infancy and accompanied by mental symptoms.

Symptoms.—The onset may be **insidious** or **rapid.** In the latter case it is preceded by a fright, or a convulsive seizure. In the majority of cases the course is progressive, affecting the face, extremities and trunk in successive order. The muscular movements are **involuntary, slow,** of **wide range.** When on the **face,** expressions of fright, joy, laughing or crying, of contemplation, etc., will be alternately observed. The eyeglobes, tongue, usually participate.

In the **upper extremities** the fingers are mostly affected. There will be a continuous display of flexion and extension, also of abduction and adduction. Sometimes the wrist, forearm and arm are similarly affected. The functional disability is therefore evident. In the **lower extremity** the toes and ankle are mostly involved. When the neck is affected, there will be an oscillation of the head in all directions. The trunk is rarely invaded.

Rigidity of the muscles is another characteristic feature of the disease. It becomes marked upon a voluntary act. The spastic condition causes deformities of the limbs and this is especially marked in the lower extremities. The gait is spastic and difficult. The reflexes are exaggerated. When the muscles of the lips and tongue are affected, the speech is difficult; it is usually slow and dragging. The muscles are hypertrophied from

continuous movements, but there are no changes in their electrical reactions. Relaxation of the joints and subluxation of the phalanges are sometimes observed.

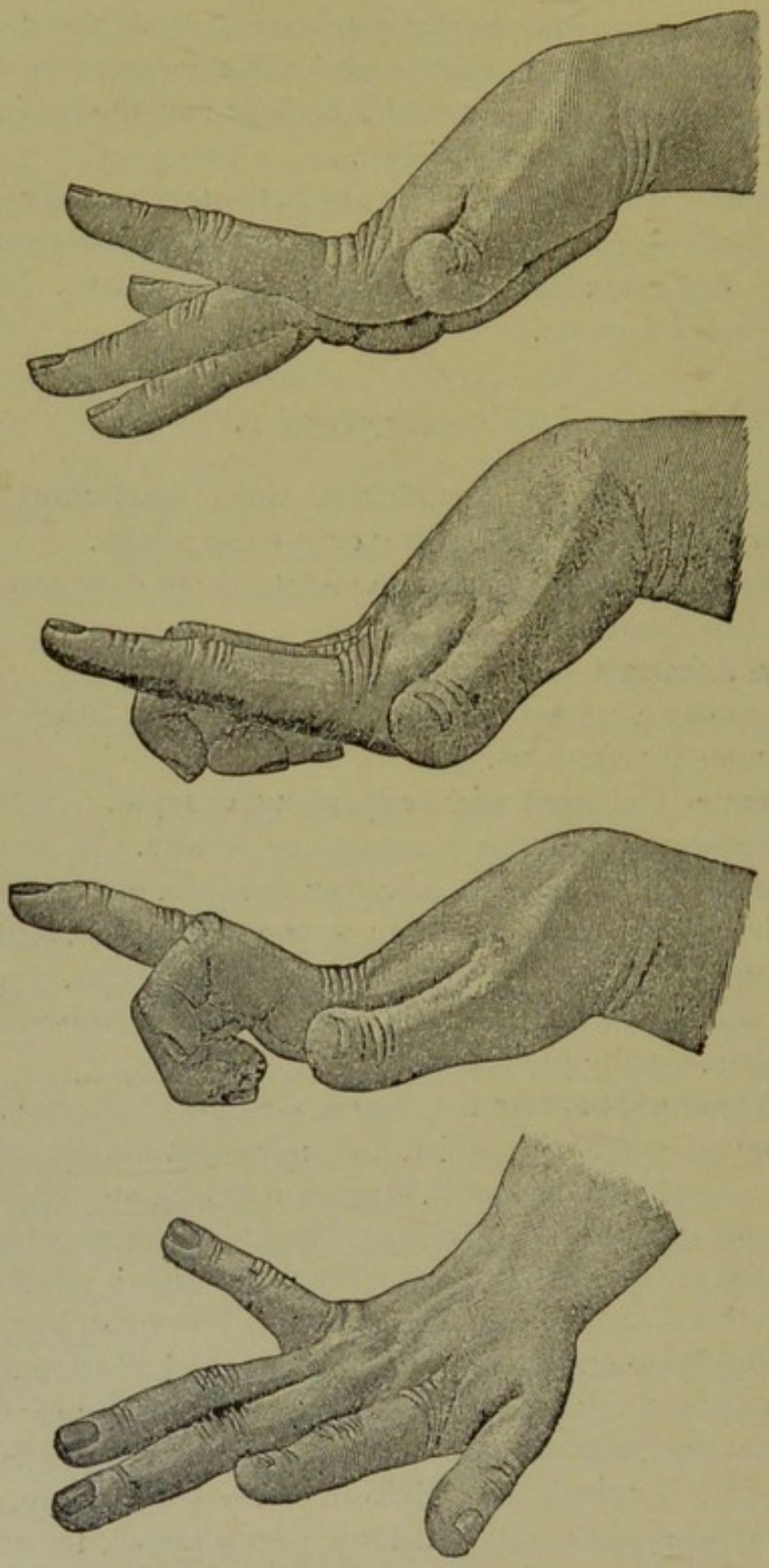


FIG. 151.—POSITION OF FINGERS IN ATHETOSIS. (*Strümpell.*)

Mental symptoms are the third characteristic sign of double athetosis. They are congenital and noticeable early in life. They consist of a feebleness of all the intellectual faculties. In exceptional cases the intelligence is preserved.

Course, Prognosis.—As all other functions of the body are intact, the disease does not endanger life. Death occurs from some intercurrent affection. The disease itself is stationary and lasts many years.

Pathogenesis.—The lesions found post-mortem have not been constant, so that positive conclusions as to the nature of the disease cannot be drawn. Cortical lesions, malformation of convolutions, pachymeningitis, asymmetry of the hemisphere, of the cerebellum, of the medulla, cerebral sclerosis, have all been observed in cases with positive findings. There are also cases in which the central nervous system was found to be normal. The consensus of opinion is that bilateral athetosis is caused by a bilateral irritation of the motor area or the motor pathway.

Etiology.—A hereditary neuropathic taint is present in the majority of cases. Syphilis, alcoholism, epilepsy, insanity, etc., are not infrequently traced in the family.

Premature or difficult labor is a potent factor in causation of cerebral disturbances and therefore of double athetosis. Infectious diseases and traumata occurring in early infancy are sometimes the direct causes of the disease. In some cases no appreciable cause can be found. The disease occurs in early infancy, although later development has also been observed.

Treatment.—Sedative medications (bromide, chloral), hydrotherapy, may be tried, but very little can be expected from them.

Systematic exercises, with the purpose of controlling the movements, kept up for a long time and carried out persistently and patiently, may yield satisfactory results.

DYSTONIA MUSCULORUM DEFORMANS (DYSBASIA LORDOTICA PROGRESSIVA)

Oppenheim in 1911 (*Neurolog. Centralblatt.*) described a symptom-group which he characterized as "a disturbance of muscle tone." Prior to him Ziehen (*Allg., Ztschr. f. Psych.*, LXVIII) observed an analogous condition to which he gave the name of "tonic torsion neurosis." The disorder, according to Oppenheim, has some resemblance to Huntington's chorea and more to athetosis, so that his diagnosis of the condition at first was between hysterical lordosis and idiopathic bilateral athetosis.

Symptoms.—The main features of the disease are: a deformity about

the pelvis and spasms of the muscles surrounding the pelvis, also twitchings in other muscles. The disease may begin with twitchings in the upper limbs, but it is in the muscles of the pelvic girdle that they are most marked. The twitchings are evident while standing or walking, but not in a lying position. The deformity, which is persistent, consists of a marked lordosis of the dorso-lumbar region with a lateral inclination of the pelvis. The gait is especially peculiar, it resembles the movements

FIG. 152.—DYSBASIA LORDOTICA PROGRESSIVA OR DYSTONIA MUSCULORUM DEFORMANS. (*Oppenheim.*)

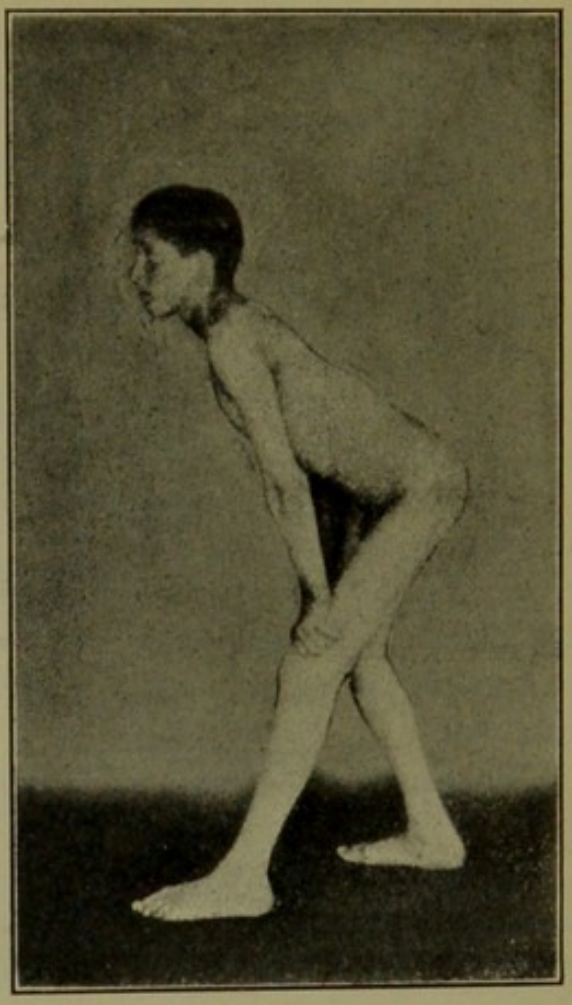


FIG. 153.—SAME AS FIG. 152.

of a quadruped. While the patient walks, he is affected with movements of a clownish character; the fatigue and strain caused by such movements brings on a perspiration and rapidity of pulse. The muscular twitchings are either a rhythmical tremor, or rhythmical clonic contractions, especially in the lumbo-abdominal muscles. Tonic contractions are seen especially in the upper extremities. On passive movements a distinct **hypotonia** is observed even in the muscles which are affected with tonic contractions. (Figs. 152 and 153.)

The disease presents no evidences of organic involvement of the nervous system. The tendon reflexes are sometimes diminished. There is no paralysis, no muscular atrophy, no electrical change. Sensations, sphincters, cranial nerves, psychic sphere—are all intact. The course is progressive. The disease occurs in children from eight to fourteen years of age.

Treatment.—Oppenheim obtained relief from metallotherapy in one case. J. Fraenkel obtained satisfactory results from intraspinal injections of sulphate of magnesium and from reëducation movements.

The nature of the disease is unknown.

TIC

It consists of **abrupt involuntary** contractions of a muscle or groups of muscles.

Symptoms.—The sudden involuntary contraction of muscles has a convulsive character and it may be **clonic** or **tonic.** In the first case the individual contractions are separated by intervals of rest. In the second case the contractions are so near each other that they give the impression of a prolonged contracture. Unlike chorea the tic is characterized by **coördinate** movements, by **systematized** movements. At the beginning of their development these movements consisted of muscular contractions executed for a certain definite purpose, but in an exaggerated manner. For example, tic of the eyelids produces exactly the act of their sudden closure done to protect the eye from penetration of a foreign body. Little by little, when these movements are frequently repeated, they become a matter of **habit** and **necessity.** Tic is therefore a disease of habit, a habit which through its persistency acquires a morbid character.

Tic may affect one muscle, if this muscle by itself has a certain functional purpose. What is important is the fact that the invaded area does not correspond to a well-defined anatomic distribution of a certain nerve or nerves contrary to what is seen in spasm. In the majority of cases several muscles contract simultaneously, as their associated action is necessary for execution of certain acts. Occasionally a certain portion of a muscle may be affected by tic; it occurs in those muscles various portions of which have different functions (deltoid, trapezius, etc.). The form, intensity and rapidity of the twitching vary from patient to patient and in the same patient. Tic has a tendency to spread and invade other functions, so that the twitch of the face may be accompanied by a sudden protrusion of the tongue or by a laryngeal noise, by a scream or by a certain gesture in other parts of the body, such as sudden kick of the foot.

Gilles de la Tourette describes various tic movements accompanied by **coprolalia,** viz. enunciation of profane words. Another characteristic feature of tic is the possiblitiy with a certain effort of retarding or suppressing the muscular contraction. The reason of it lies in the cortical nature of the phenomenon.

Tic usually disappears during sleep.

Sensations, reflexes, sphincters are intact in tic.

The affected muscles may sometimes become hypertrophied, but this condition is only functional in nature.

Forms

Tic of the Face.—The most frequent tic of all the muscles of the face is that of the **eyelids** (**palpebral tic**). It is usually bilateral. The neighboring muscles frequently participate. The eye-globes frequently participate (**nystagmoid tic**). The latter symptom is not observed in blepharospasm. Next to the eyelids the **lips** are the most frequently involved. Grimaces are the result of tic of the lips.

FIG. 154.

FIG. 155.

FIGS. 154 and 155.—MENTAL TORTICOLLIS AND CORRECTION GESTURE. (*Brissaud.*)

Any other muscles of the face, including the **platysma,** may be affected by tic. When the **tongue** is involved, all sorts of noises may be heard from the movements of the tongue. When the **masticatory** muscles suffer from tic, abrupt and repeated lowering or raising of the lower jaw will be noticed.

Tic of the Neck.—Sudden and repeated rotation, flexion or extension of the head will be noticed according to the muscles involved. The flexors are more frequently affected than the extensors. Tic of the neck is frequently associated with tic of the face or of the shoulders.

Torticollis in the form of tic (**mental torticollis** of Brissaud) or **spasmodic torticollis** may present itself as sudden simple rotatory movements, rotation with flexion or rotation with extension. The tic may consist of a single movement or of a series of successive movements. Among all the muscles of the neck the sterno-mastoid is the most frequently affected. During the contraction the face is turned to the opposite side, the head is inclined on the same side and the ear touches the shoulder. When the upper part of the trapezius and splenius muscles are involved, together with the sterno-mastoid, the head, besides being inclined, is also drawn backward. When the muscles of the anterior part of the neck are in-

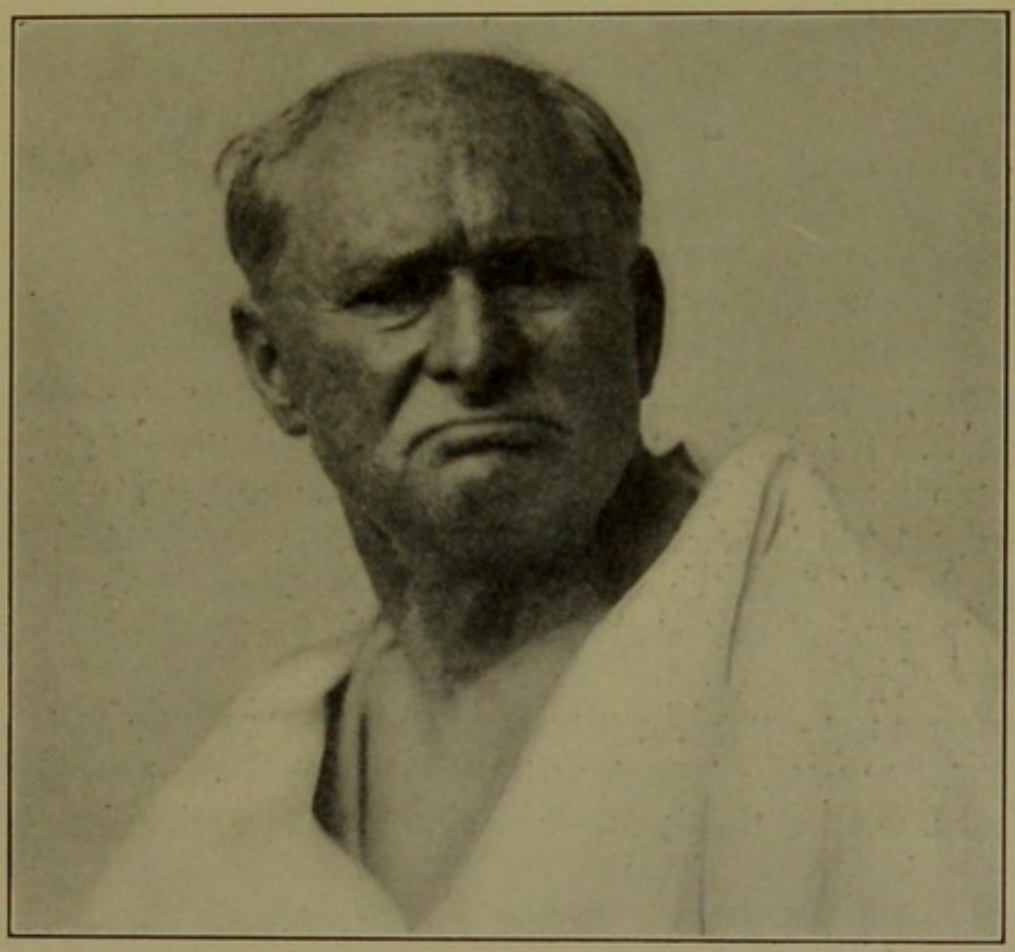


Fig. 156.—Spasmodic Torticolis. Patient Seen in Attempt to Correct Position of Head.

volved (which is rare), the head falls forward so that the chin touches the chest. Rotatory tic of the head is often combined with elevation of the shoulder.

The patients frequently complain of pain or of a drawing sensation in the neck. This is of course due to the repeated muscular contractions. Rest, physical and mental, decreases the intensity of the tic. Fatigue and emotions increase it. The patients use all sorts of subterfuges to

remove the torticollis. The most frequent attitude is to keep a finger on the chin. The onset of the affection is usually insidious. The disease originates from some false position adapted at first for the purpose of relieving pain in the neck or from unavoidable positions in certain occupations which require special gestures or, as some pretend, from visual disturbances. The act is repeated a number of times, at first voluntarily, and finally involuntarily. The condition is thus established. In cases of long standing the involved sterno-mastoid muscle may become hypertrophied, and the muscle of the opposite side atrophied for want of exercise.

Tic of the shoulder consists mostly of a sudden raising of the shoulder. The habit is acquired through some discomfort felt from the clothes covering the shoulder or else from some pain in that region.

Tic of the arms, hands and fingers occasionally occurs.

Tic of the Trunk is a rare condition. In a case reported by me (*Amer. Medicine*, 1906) the tic consisted of abrupt salutations: the recti muscles of the abdomen contracted.

Tic of the Lower Extremities may be manifested in sudden bending of the knees, jumping, kicking, changing of the steps.

Respiratory Tic.—It presents itself as abrupt inspiratory or expiratory movements. It is frequently associated with contractions of the muscles of the naso-pharynx. The sounds of snuffing, of snoring, are heard.

Laryngeal Tic is manifested either in sudden laryngeal sounds, as grunting, barking, etc., or shouting certain syllables or words (Verbal Tic). The latter is particularly encountered in a symptom-group known under the name of

Tic Convulsif.—This condition was described first by Guinon and Gilles de la Tourette. It begins with the muscles of the face. Blinking of the eyes, pouting the lips, protruding the tongue, grimacing the face, blowing, whistling, chattering of the teeth—all these acts are done with extreme rapidity, convulsive-like. Frequently the tic does not remain confined to the face; the neck, upper and lower extremities and trunk participate. Thus the patients will also exhibit sudden raising and lowering of the shoulders, propulsion, retropulsion or lateral movements of the trunk, rubbing of the hands, raising of a finger to the nose, ear or mouth, stamping of the feet, jumping, dancing, etc. All these movements are spasmodic, abrupt, rapid, but they **systematically** succeed each other. They may be arrested temporarily, but at the expense of a painful struggle and intense anxiety. Voluntary acts arrest the movements. They disappear during sleep.

Another characteristic manifestation of tic convulsif is **coprolalia** or

echolalia. The first consists of a sudden use of obscene words in the midst of an ordinary conversation. In echolalia the patient repeats sounds, words or syllables heard around him. Sometimes he repeats movements seen by him (**echokinesis**). All these acts are done under the influence of an irresistible impulse. The patient is always fully conscious of his uncontrollable movements. To mask them he frequently executes some voluntary acts in rapid succession, but it is not an easy task to overcome the involuntary movements. For the same reason the patient is seen doing the most peculiar acts, as spending considerable time in counting the buttons on his coat, the number of stores on the street, his own footsteps, etc. Here the motor phenomenon of Gilles de la Tourette's disease may be absent, but imperative conceptions compel the patient to utter certain words or sentences or perform certain acts. This is a **Psychic Tic.**

Tic of Salaam (Spasm nutans).—It is a rare affection and occurs mostly in infants. It consists of the act of flexion of the head and the upper part of the trunk repeated a great many times—from twenty to fifty in a minute. It is, otherwise speaking, a convulsive salutation. The affection is also spoken of as **"head nodding."** In some cases the phenomenon assumes the character of epilepsy. Thus it may be preceded by a sudden pallor of the face, staring and dilatation of the pupils; there may be also a loss of consciousness. In some of these cases genuine epileptic convulsions develop later.

Course, Duration, Prognosis.—The tendency of tic is to spread and invade other portions of the body. The duration is uncertain. Recurrences are frequent. The younger the individual, the more is the case hopeful as to recovery. Rest, mild diversion, quit life have a beneficial effect. Fatigue, emotions, aggravate and prolong the condition. The longer the tic lasts the more obstinate is the case. Among all the forms **Tic convulsif** has the gravest prognosis. It has an essentially progressive course. The mental symptoms which accompany the motor phenomena may terminate in dementia.

Tic in the aged presents also an unfavorable prognosis.

The other forms of tic are amenable to treatment if properly managed.

Diagnosis.—Although the symptoms of tic are typical, nevertheless it may be confounded with some other affections. It is **spasm** that is sometimes difficult to differentiate from tic.

In tic the movements are purposive, only in an exaggerated form. In spasm one fails to find the least tendency to reproduce physiologic acts.. Spasm of the face, for example, has no resemblance to ordinary mimicry In tic the muscles involved do not correspond to a well-defined anatomical

distribution of a nerve. In spasm, on the contrary, the convulsive movements are produced solely in the area of distribution of a nerve. Tic disappears during sleep, spasm does not. An effort of will or attention is capable to arrest or inhibit tic, but not a spasm of the muscles. In the majority of cases of tic we find a hereditary predisposition to some nervous disorder. This is not observed in cases of spasm.

Paramyoclonus multiplex is recognized by its clonic contractions, which are sudden, quick in succession, irregular, arhythmical; they may involve not only an entire muscle, but also a portion of it and usually they affect symmetrical muscles. They occur more frequently in the lower extremities than in any other portion of the body; the face never participates. They cannot be controlled by the patient. They can be easily brought on by stimulation of the skin.

Myokymia is characterized by fine fibrillary contractions, also by vaso-motor and sensory disturbances.

In **chorea** the movements are incoördinate, irregular and not convulsive.

Pathogenesis.—As said above, tic is a functional disturbance in which repetition of the act is influenced by an imperative desire, so much so that if the patient resists the act he suffers, but after the movement is produced there is great relief and satisfaction. It is therefore evident that the mentality plays a certain rôle in the phenomenon. It is because of a deficient will power that the twitchings become automatic, that a habit is established. In fact tic occurs usually in individuals with peculiar ideas, desires and tendencies. They are eccentric, not stable, and frequently develop fixed ideas, obsessions. For this reason Brissaud considers tic as a "psychic" malady.

Etiology.—Tic can be considered as a degenerative neurosis. A neuropathic heredity plays an important rôle. Insanity, epilepsy, hysteria, tic, tuberculosis, diabetes, organic nervous diseases, are not infrequently traced in the family histories of individuals affected with tic.

Bad hygienic surroundings, habits, excesses, undue cerebral fatigue, are predisposing causes of tic.

Local irritation and local lesions (eyes and naso-pharynx) are the exciting causes.

Tic is rare in very young children. It usually occurs at the age when great physiological changes take place, viz. puberty, menopause, old age.

Treatment.—In view of the neuropathic make-up of individuals suffering from tic, the first indication is to improve their general health with proper hygienic and dietetic measures. Such patients must lead a life free from excitement or emotions of any sort.

As to the tic itself, Brissaud, Meige, Feindel and others obtained satisfactory results from special **physical** and **psychic** methods. The first consists either of **voluntary immobilization** followed by **systematic exercises.** When **immobilization** is used, the patient is taught to immobilize the affected muscles for a gradually increasing period of time. The sittings are held daily, at first only for a few seconds and only two or three times a day. Gradually the number of exercises is increased and the duration of each is prolonged. Patience and perseverance are required. A child must be placed in charge of some trained person. An adult can be taught how to proceed. He is advised to have before him a mirror so that he can watch the procedures. When a certain amount of control has been obtained by the patient, the next method is taken up, viz., **voluntary movements.**

Exercises consist of replacing the involuntary movements of the tic by **voluntary** and **correct** movements. By being voluntarily contracted, but in a slow, deliberate and correct manner, the affected muscles fall under control and thus become trained. Here again the first séances and their duration must be brief. Gradually they are increased. These exercises should be made under control of an observer or the patient can train himself gradually in the performance. In the latter case he places himself before a mirror and is thus able to acquire a certain skill in the treatment.

The **psychic** method plays an enormous rôle. It consists of continuous encouragement and of pointing out to the patient the necessity of training his will power in overcoming the involuntary movements.

Isolation, rest in bed, removal from usual surroundings, together with the above physical and mental training will give in a number of cases satisfactory results.

As to medications there is none to be relied upon. The usual sedatives may be employed.

The most recent researches of Loeb and J. B. MacCallum, also of W. G. MacCallum and C. Voegtlin, show a certain relationship between abnormal motor phenomena and calcium metabolism, also the function of the parathyreoid glands. A trial of calcium salts and of parathyreoids in tic is therefore indicated.

In spasmodic torticollis operative procedures have been attempted. They consist of excision of a portion of the spinal accessory nerve on the affected side and of section of the posterior primary divisions of the upper cervical nerves on the opposite side. I have seen failures from this operation.

FACIAL SPASM

Symptoms.—Spasm of the face commences with clonic contractions which as they advance gain in rapidity and at the height of the attack are replaced by tonic contractions. As the latter subside, clonic contractions reappear and remain until the attack is over. The entire cycle lasts but a minute. The following individual features are observed during the paroxysm. The forehead on the affected side is wrinkled, the orbicularis palpebrarum closes the eye; there is a simultaneous con-

Fig. 157.—Facial Spasm on the Left.

traction of the frontalis and orbicularis palpebrarum. This phenomenon is called by Babinski paradoxical synergia. The zygomatic muscles deviate the angle of mouth. The nose is curved toward the affected side and the chin presents a characteristic depression on the affected side. The muscular contractions may be either fascicular, tremulous or coarse, of a wider range (Figs. 157 and 158).

The muscles involved in facial spasm correspond to the well-defined anatomical distribution of the seventh nerve and according to the case the convulsive movements predominate in the upper or the lower portion of the nerve. Facial spasm occurs during sleep. No effort of will or attention is capable to arrest or prevent an attack.

The most frequent form is hemispasm, but **double spasm** has also been observed. Meige described a **median facial spasm,** which consists of bilateral convulsive movements predominating near the median line of the face. Both orbiculares palpebrarum are most markedly involved.

Pathogenesis.—The stimulation of the muscles supplied by the seventh nerve may originate in the seventh nerve itself or in its nucleus or else in any of the sensory fibers of the fifth nerve. Blepharospasm is an ex-

Fig. 158.—Facial Palsy on the Left Produced by Injection of Alcohol into the Left Facial Nerve.

ample of stimulation of the orbicularis through an irritation of the sensory fibers of palpebral mucosa. Facial spasm has been observed in attacks of neuralgia, in connection with carious teeth. Not infrequently the spasm is found limited to both upper and lower eyelids. It is called **blepharospasm.** The contractions may be **tonic** or **clonic.** In the first variety the eyelids are persistently closed during the paroxysm, which may last many minutes. It is due to some irritation of ocular branches of the fifth nerve. The clonic variety consists of rapid winking movements.

The majority of cases show that the cause of facial spasm lies in the peripheral portion of the seventh nerve. The occurrence of facial spasm in cases of facial palsy speaks in favor of peripheral cause of the spasm.

Neuroma on the facial nerve has been found in such cases (A. Thomas, *Rev. Neurol.*, 1907). While an irritation of the peripheral end of the nerve is the most common cause, nevertheless facial spasm has been observed also in organic lesions of the central nervous system. In cases of meningo-encephalitis, of pseudo-bulbar palsy, of the pons, facial spasm has been observed. Finally the above-mentioned cases of involvement of the fifth nerve show that a lesion of any portion of the reflex arc (sensory fibers, nucleus of the seventh nerve, motor fibers of the facial nerve) may be the cause of facial spasm.

Course, Duration, Prognosis.—It is usually rebellious to medical treatment. It may have intermissions. It lasts ordinarily an indefinite time. It has no effect on patient's life.

Treatment.—If a local cause of irritation can be detected, its removal is necessary. Freezing of the face on the affected side has been recommended by Weir Mitchell. The most effective method of treatment is injection of a few minims of 80 per cent. alcohol into the nerve at its exit of the stylomastoid foramen. It has given me the most gratifying results. The spasms ceased for periods ranging from eighteen months to three years. The facial palsy which follows immediately the injection disappeared at the end of five or six weeks in every case. In some cases returns of spasm were treated with repeated injections. No bad results followed repetition of injections. An experimental study (*Trans. Amer. Neurol. Ass'n.*, 1913) on dogs has shown me that injection of alcohol into a motor nerve is followed by very slight pathological changes in the perineurium, but the nerve fibers themselves remain unaffected.

In cases of double facial spasm the injection should be made into each nerve separately and only after the palsy disappeared on one side. Alcoholic injections into the nerve may be tried even in cases of organic diseases of the nervous system.

MYOCLONIA

The disease is characterized by **sudden unsystematized, involuntary clonic** contractions similar to those produced by an electric shock. They may be localized or disseminated. The following varieties belong to the group **myoclonia.**

I. Paramyoclonus Multiplex of Friedreich.
II. Familial Myoclonia with Epilepsy of Unverricht.
III. Myokymia.
IV. Electric Chorea of Bergeron-Hénoch.
V. Dubini's Chorea.

Pathogenesis of Myocloniæ.—The symptom myoclonia may be encountered in the course of various organic and functional nervous diseases. That it may be an independent affection is true. That it may be merely a hysterical phenomenon is also correct. It is accepted by the majority of observers that myoclonia is to be considered as an episodic manifestation in neuropathic individuals.

As to the physiological basis for myoclonic twitchings, the views are divided. The majority believe that the condition is due to an irritation of the cells of the anterior cornua of the spinal cord.

The latest researches, particularly of Loeb and J. B. MacCallum, also of W. G. MacCallum and C. Voegtlin, show a certain relationship between various twitchings and calcium metabolism, also the function of the parathyreoid glands.

I. Paramyoclonus Multiplex

Symptoms.—The myoclonic contractions, which are **sudden** and **lightning-like,** appear first in the lower extremities, but they may become generalized. The face as a rule is respected. The contractions are **unequal, irregular** and **arhythmical.** They may affect individual muscles or groups of muscles. Their frequency may vary: they may occur every few minutes or every half hour. They may be mild or so violent as to move the affected part. When in the lower limbs, the locomotion is disturbed. When in the upper limbs, movements of flexion, extension, supination, etc., are observed so that the usual occupation of the patient is impossible. Usually symmetrical muscles on both sides of the body are affected. The muscles of the limbs are more frequently affected than those of the trunk.

If the muscles of the pharynx, larynx and diaphragm are affected, disturbance of deglutition and of respiration, respectively, will be observed.

The nutrition of the muscles as well as their electrical reactions are intact. Sensations are normal. The reflexes are exaggerated.

The muscular contractions cease during sleep; they may be sometimes arrested or lessened by a voluntary effort, although in some cases a reverse condition is observed. Emotion increases their intensity and frequency.

Course, Duration, Prognosis.—The disease is progressive and its onset is insidious. It may last indefinitely. Cases of recovery have been reported. Recurrences are very frequent.

Diagnosis.—The **sudden lightning-like clonic contraction** is sometimes sufficient for the diagnosis.

In chorea the movements are not so abrupt and their range is wider than in myoclonia.

It is with tic that the differential diagnosis presents sometimes great difficulties. However in tic the movements are coördinate and systematized.

Etiology.—Paramyoclonus is found sometimes associated with organic and functional nervous diseases, also insanities. A neuropathic heredity exists in the majority of cases. Infectious diseases, intoxications, fatigue, emotion and traumatism are among the exciting causes.

Treatment.—When the condition is only a symptom of other diseases, the first indication is to treat the latter. As in the majority of cases myoclonia develops in neuropathic subjects, much attention should be given to the general health. Hydrotherapy, moderate exercises, proper mode of living, avoidance of excitement and worry, nutritious food, etc., are necessary (see chapter on Neuropathy). For controlling the muscular contractions sedatives may be employed, viz. bromides, antipyrin, chloral and others. Arsenic has been advised. Confinement to bed may sometimes be of service. Not much can be expected from medications.

The above-mentioned remarks concerning calcium metabolism and the function of the parathyreoid glands (see Pathogenesis) indicate the use of calcium salts and of parathyreoids in myoclonias.

II. Familial Myoclonia with Epilepsy

(Unverricht's Type)

This form presents an association of paramyoclonus multiplex with epilepsy. The latter may occur only early in life and then disappear, to be substituted by myoclonic twitchings, or else accompany the myoclonia. The occurrence of the same condition in several members of the same family is a striking feature of the disease. In Unverricht's first case (1891) five brothers and a sister were thus affected.

III. Myokymia

It is characterized by **continuous fibrillary contractions.** The muscles of the extremities are most frequently involved, although other parts of the body may be also affected. Sometimes pain and hyperhidrosis accompany the muscular twitchings. In one of my cases myokymia of the right lower half of the face was associated with myoclonia of the upper half of the face. The least mechanical irritation increased the contractions. The affected muscles presented a decreased faradic and galvanic irritability.

IV. Electric Chorea (Bergeron-Hénoch)

It consists of **sudden twitchings** rapidly, but rhythmically, repeating themselves. An attempt to control them increases their intensity. They disappear during sleep.

They may affect any portion of the body. The movements are so frequent and intense that the patient is obliged to give up his usual work. The nutrition of the affected muscles is not disturbed. The prognosis is usually good.

In a large number of cases the disease was associated with gastric disturbances and improvement of the latter was followed by disappearance of the muscular twitching. Autointoxication is therefore supposed to be the cause of the affection. In some cases it may be a manifestation of hysteria, as instances of recovery from suggestion prove.

V. Dubini's Chorea

By its manifestations it resembles the electric chorea of the preceding chapter, but by its course, duration and termination it differs.

The onset, which is abrupt and sudden, is accompanied by **pain.** The latter affects the head, neck and lumbar region. The twitchings are rapid, appear first in the upper extremities and soon spread. Although they are rhythmical, nevertheless they are constant. In the course of the disease not infrequently are observed genuine **convulsive seizures** without loss of consciousness. **Fever** is also present in the majority of cases.

The disease is progressive. Gradually the twitchings and the convulsive seizures increase in intensity and frequency, a comatose state supervenes and death follows. The duration of the affection is from several days to four or five months.

The sudden onset, the pain, the accompanying fever and the associated pulmonary diseases (which are quite frequent) are in favor of an **infectious origin** of the affection. Post-mortem investigations have shown in a number of cases congestion and inflammation of the meninges, also of the cerebral tissue.

TETANY

It is characterized by **bilateral, intermittent, painful cramps,** especially in the hands.

Symptoms.—The muscular spasms occur in attacks. The latter are

usually preceded by a few premonitory symptoms, viz. paræsthesia (tingling, numbness, etc.), general malaise and sometimes by a mental depression and vertigo or headache. In the majority of cases cramps appear first in the fingers. The **attitude of the hand** is then very characteristic: it is either in a **writing** position or in an **obstetrical** position, viz. the fingers are extended, the first phalanges are flexed, the thumb is against the palmar surface of the other fingers, the entire hand is flexed. Variations in this attitude are observed. When the interossei and lumbricales are affected, the hand is in a claw-like position (main en griffe.)

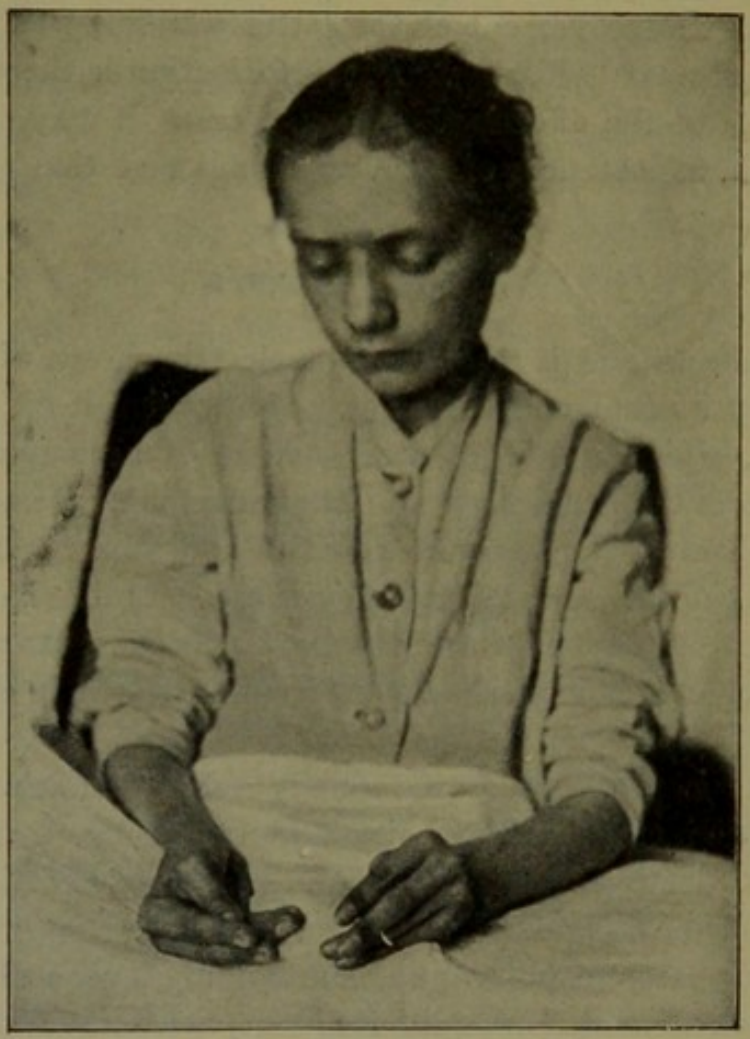


FIG. 159—A CASE OF TETANY DURING AN ATTACK. (*Oppenheim.*)

When the contracture spreads and involves the arm, the latter is in a forced flexion and applied to the thorax. If the lower extremities are affected, the flexors of the foot and toes are found mostly in a state of tonic contraction. The toes are flexed and adducted, the feet are arched; the attitude of the foot is equino-varus.

Tetany may also affect the muscles of the trunk, abdomen and neck and in rare cases the ocular muscles. When the neck muscles are involved,

the head is bent forward and the chin touches the chest. Rarely the dorsal muscles are involved: the head is then drawn backward and the body is in opisthotonos. The muscles of the face, of the tongue, of the ocular muscles, are occasionally affected. The diaphragm, larynx, may participate and then the patient is threatened with suffocation. If the sphincter of the bladder is affected, retention of urine will be present. The tetanic contractions are usually very painful and the least attempt to move the affected parts increases the pain. Voluntary movements are impossible. During an attack the temperature is slightly elevated and the pulse is accelerated. The spasm may persist during sleep, although as a rule it is less severe.

In addition to the above clinical picture the following characteristic symptoms are observed in tetany.

1. **Trousseau's Sign.**—This observer found that compression of the biceps or immediately below the inferior insertions of the deltoid and in the lower extremities upon the internal surface of the thigh a tonic contraction of the corresponding muscles will be produced. V. Frankl-Hochwart has shown experimentally that compression of the nerve-trunks is the cause of the contracture. Trousseau's phenomenon is pathognomonic of tetany.

2. **Chvostek's Sign.**—Percussion or any mechanical irritability of a motor or mixed nerve or the muscles of the face produces vivid muscular contraction. This so-called **facial phenomenon** is observed in the domain of the seventh nerve. The symptom is not constant.

3. **Hoffmann's Sign.**—Pressure upon sensory nerves produces marked pain or paræsthesias. Their electrical excitability is also increased.

4. **Erb's Sign.**—The electrical excitability of motor nerves is increased so that a very **mild** galvanic or faradic current gives a prompt and marked muscular contraction. The contraction is not brief as in normal condition, but prolonged, and may not subside until the current is interrupted. The anodal closure or opening contracture is more prompt than the cathodal closure contraction. Increased response to galvanism is more frequently observed that to faradism. Erb believes that not all the nerves are equally apt to be easily irritated. The ulnar nerve is the most frequently responsive.

5. **Schlesinger's Sign.**—If the extended lower limb is forcibly flexed over the pelvis, a spasm will appear in the extensors of the knee and the foot is placed in the position of extreme supination.

Among other symptoms, although not constant, may be mentioned vaso-motor and trophic disturbances, such as hyperhidrosis, herpes, œdema, falling out of the nails, muscular atrophy. During the attack

the face may be flushed, the extremities are cyanosed, the hands are red. These symptoms disappear at the end of the attack. The reflexes are usually normal, but an increase or diminution is sometimes observed. The objective sensations are not modified except when hysteria is associated. Indicanuria is so frequent in children that it may be considered pathognomonic. Glycosuria has also been observed.

Occasionally epilepsy, exophthalmic goiter and myxœdema have been found associated with tetany.

Course, Duration, Prognosis.—The attacks may occur every day or only after more or less prolonged periods: the intervals may be hours or days. The individual spasms may last from a few minutes to several hours. The entire disease may consist of but a few attacks or else may last weeks or months. Recurrences are not infrequent and in some cases they appear at regular intervals during a number of years. The prognosis in the majority of cases is favorable. It depends, however, upon the cause. Cases with gastric dilatation or with exophthalmic goiter present an unfavorable prognosis. Moynihan has shown that gastric tetany can be relieved by gastro-enterostomy. When the respiratory muscles are affected, life is threatened. Children particularly are in danger of asphyxia due to spasm of the glottis.

Diagnosis.—The symptoms of tetany are ordinarily so typical that the diagnosis is made without difficulty. In **tetanus** the spasm begins with the muscles of the jaws and neck, the hands and fingers usually escape, the temperature is elevated from the beginning. A **pseudo-tetanus,** in which there are generalized contractures with albumin in the urine, is met with frequently in children and bears an unfavorable prognosis.

In **hysteria** tonic muscular contractions (**pseudo-tetany**) are unilateral, painless and not accompanied by Trousseau's and Erb's signs.

Latent cases of tetany without spasms have been described. Here the only symptoms of the disease will be Chvostek's and Erb's signs, also some numbness or tingling of the hands.

Etiology.—Certain **occupations** play a predisposing rôle. According to Frankl-Hochwart's statistics, among 314 patients 141 were shoemakers and 42 tailors. Certain **countries** are more affected than others. The disease is frequent in Sweden, Austria and Germany.

Epidemics of the affection have been observed. Men between fifteen and twenty-five are particularly attacked. In women the disease occurs during pregnancy or lactation and puerperal state.

Infection and **intoxication** play a predominant part in the causation of tetany. In the course of infectious diseases, such as typhoid fever,

grippe, scarlet fever, measles, malaria, or during convalescence tetany may occur. Among toxic conditions gastro-intestinal disorders are very frequently the cause of tetany. Gastro-enteritis is almost the only cause of tetany in children. Tetany has also been seen in association with albuminuria. Extirpation of the thyroid gland has been followed by tetany. Here also a toxic element is the cause of the disease. It is probable that in such cases the parathyroids were removed together with the thyroid gland. Removal of the **parathyroids** has been seen to be followed by tetany.

Rickets, osteomalacia, intestinal worms, use of alcohol, ergot, inhalation of alcohol, may be also accompanied by tetany.

Local irritation, as the use of a stomach pump or passing of a sound, percussion of the region of the stomach, cold, emotion, exertion, are the exciting causes.

Pathogenesis.—No reliance can be placed upon the findings at the autopsies. The results are contradictory and in the majority of cases negative. Mention can be made of some poliomyelitic changes in the cells of the anterior cornua of the spinal cord. The consensus of opinion is that in the majority of cases the disease is due to a **toxic** or **infectious condition.** The occurrence of it in connection with gastro-intestinal disorders, with removal of the thyroid or parathyroid glands, with infectious diseases, speaks in favor of the above view. Experimental researches and close observations on operative cases are strongly in favor of parathyroid insufficiency as being the pathogenetic factor in tetany. But what toxic element is at work, it is at present difficult to ascertain.

Gastric Tetany.—In this form of tetany an ulcer of the stomach with resulting pyloric stenosis and gastric dilatation have been found. There is usually hyperchlorhydria. The typical symptoms of tetany are present. The intensity of the spasms may vary from case to case. Sometimes they are so slight or occur so rarely that tetany may be overlooked. In the majority of cases the spasms are severe. The only radical measure in such cases is gastro-enterostomy. As to the pathogenesis of gastric tetany, it may be presumed that a toxic element from the stomach is added to the already existing parathyroid insufficiency. Langmead has recently called attention to tetany in association with dilatation of the colon. The spasms occur regularly and appear to be in intimate relation with the character of the feces. The condition is met with in children of arrested physical and intellectual development.

Treatment.—Removal of the cause is the first indication. Gastro-intestinal disorders should be remedied by intestinal antiseptics, enemas and emetics if necessary, but the stomach-pump must be avoided (see

reasons above). In cases of operations on goiter care must be taken to avoid total thyroidectomy. When the latter is unavoidable, thyroid or parathyroid extract must be administered internally.

When exposure to cold is the cause, warm baths and diaphoretics are of benefit. The spasms can be relieved by sedative medications, such as bromides, morphine, chloral. Rest in bed is an excellent measure in some cases. Trousseau advises application of ice to the spine. On the other hand, tepid baths administered several times a day for ten to fifteen minutes may be of great benefit. Galvanism may sometimes render good services. In one of my cases absolute rest with milk diet gave me very satisfactory results. In the gastric form of the affection, which is rebellious to treatment, gastro-enterostomy should be undertaken without delay, as excellent results have been reported. In cases of asphyxia due to spasm of the glottis, hypodermic injections of pilocarpine or application of a wet cloth to the neck may be useful. Digitalis is advised by Gowers.

The researches of Loeb and J. B. MacCallum show that there is a great relationship between a tetany and reduction of calcium salts in the organism. An analysis of blood taken from a dog during tetany shows an amount of calcium which is only about a half that of a normal dog on the same constant diet. It is also known that the parathyroids control the calcium metabolism, so that upon their removal a rapid excretion deprives the tissues of calcium salts. For these reasons administration of calcium salts or of parathyroids is indicated in tetany. The intravenous method is the best for administration of calcium salts. Forty to 80 grains of calcium lactate are diluted in 400–500 c.c. of normal salt solution. The injection can be repeated in twenty-four hours if necessary. The parathyroids may be administered by mouth, intravenously or by grafting. The intravenous method gave the best results. Krabbel (*Beitr. z. klin. Chir.*, 1911) has recently reported excellent results from implanting parathyroid bodies in the tibia of one patient and in the preperitoneal space of another patient.

MYOSPASM FROM INTENSE HEAT

This condition was first described in 1904 by Edsall (*Amer. J. Med. Sciences*) and later from a larger number of cases by Cameron (*J. Am. Med. Ass.*, 1909).

Symptoms.—In working men exposed to intense heat (140°–235° F.) such as iron-workers or men employed in firerooms of vessels, a very painful tonic spasm of the muscles develops spontaneously or upon the least voluntary effort. An attack lasts from half a minute to a minute and occurs

very frequently during the illness, the duration of which is about twenty-four hours. A sense of exhaustion and soreness with tingling in the muscles remains for some time. Between the individual spasms a fibrillary contraction of the affected muscles is distinctly noticeable. The muscles of the forearms and legs, also the abdominal muscles, are usually involved. The condition grossly resembles tetany, but the special symptoms characteristic of the latter are absent, viz. Erb's, Trousseau's and Hoffmann's sign. The mechanical irritability of the muscles is increased. The reflexes are normal, there are no changes in the general objective sensibility (touch, pain and temperature), in the sphincters and in the pupils.

Etiology.—The spasms are due to the intense heat of the atmosphere in which the work is done. They occur, according to Cameron, usually after the men have been working for some time. Men on the day turn would be attacked toward the close of the morning's or afternoon's work, and the night-turn men toward midnight. **Overwork** is probably one of the predisposing factors. **Alcoholism** is probably another predisposing cause. Cameron observed that the spasms occurred mostly in hard drinkers. Lowered muscular tone from other causes is also one of the predisposing elements.

Prognosis.—One attack usually predisposes to others. Recovery is possible. However, fatal cases have been reported. They may be due to a spasm of heart muscle.

Pathogenesis.—Nothing definite is known as to the nature of the disorder. Disturbances of metabolism producing some degenerative condition of the muscles have been suggested.

Treatment.—It must naturally be symptomatic. Pain may be relieved by the usual remedies or by a general anæsthetic. A mild interrupted faradic current gave Cameron some satisfactory results.

THOMSEN'S DISEASE

(Myotonia Congenita)

Symptoms.—The chief symptom is a sudden tonic contraction of a group of muscles when an attempt is made to make a forced movement. At first there is an inability to continue the movement, but gradually and very slowly the muscles relax and the affected limb is able to perform the act. The stronger the contraction is the longer is the relaxation. When later an attempt is made to stop or to modify the movement, a new spasm occurs. As the lower extremities are the usual seat of myotonia, it is in the act of walking that the phenomenon is mostly observable. All the muscles may become affected, especially those of the extremities, but

those of the trunk and neck are less frequently involved than those of the extremities. Exceptionally the muscles of the tongue and the masticatory muscles are involved. The muscles of deglutition and ocular muscles are very rarely affected.

The myotonic spasm is **increased** by **reflex acts,** such as sneezing, coughing, etc., by cold, exertion and especially by an **emotion.** On the contrary it is **decreased** by heat, physical and mental rest.

The muscles affected by **myotonia** are generally **hypertrophied,** but their **power is diminished.** The following special signs are characteristic of the myotonic **muscles** and of the **nerves** distributed in them.

Erb called attention to the fact that mechanical irritability of the **nerves** is normal or diminished. Their electrical reactions are as follows. The faradic and galvanic irritability of the **nerves** are quantitatively normal when a moderate degree of stimulation is applied. Should the stimulation be prolonged, a persistent tonic contraction will follow. Mechanical irritability of the **muscles** is increased. The electrical contractility presents here a special feature.

With the **galvanic** current, the KCC and AnCC are equal. The contractions are **sluggish** and **continue long after the excitation has ceased.** With the faradic current the contraction also lasts a long time (about a half of a minute) and **undulation** of the muscle is observed. These phenomena constitute the so-called **"myotonic reaction."**

Psychic disturbances and epilepsy are sometimes associated with Thomsen's disease. Muscular atrophy and multiple neuritis have also been reported in connection with myotonia congenita.

Course, Duration, Prognosis.—The disease makes its first appearance in infancy, but becomes well developed at the age of twenty. Exertion aggravates it. It may be arrested, but frequently recurs. Life is not threatened, but the affection is incurable; it lasts all life.

Diagnosis.—The special characteristics of the disease are sufficient for diagnosis. Eulenburg described a **"paramyotonia congenita,"** which is recognized by a symmetrical muscular rigidity without Erb's myotonic reaction; it usually follows exposure to cold.

Etiology.—Heredity plays a predominant part. Thomsen traced over twenty members in his own family. Males are more frequently affected than females.

Pathogenesis.—The consensus of opinion is that the disease is a form of **myopathy** (see this chapter). Histological studies show hypertrophy of muscular fibers and proliferation of their nuclei. The central nervous system is intact. Congenital abnormal development is probably the true nature of the malady.

Treatment.—All the factors that are apt to increase the muscular tonicity (see above) should be avoided. Moderate exercises and rational gymnastics may be beneficial.

OCCUPATION NEUROSES

(Occupation Spasms)

Under this name is understood a motor disturbance consisting of a sudden cramp in a group of muscles used in certain acts and brought on **exclusively** during the execution of those acts.

This functional disturbance may occur in any portion of the body, but more particularly in the upper extremities. The most typical form of these neuroses is

Writer's Cramp

It develops slowly. At first the patient notices a certain fatigue and stiffness in the fingers while writing, so that he is obliged to rest for a while. Soon he finds that the interval of rest must be increased; the difficulty of writing appears as soon as the act is commenced. He is forced to have recourse to various positions, use both hands, etc., and finally give up completely the act of writing.

The phenomenon may present itself in the **spastic, paralytic** and **tremulous** forms.

Spastic.—A sudden extension of the index and flexion or adduction of the thumb or *vice versa* is the initial manifestation. Sometimes the medius and the other fingers suddenly flex. In some cases the condition extends to the forearm, arm and shoulder. This is due to the fact that in the act of writing participate not only the muscles of the hand, but also those of the forearm and arm.

The cramp will persist as long as the act of writing is insisted upon. In advanced cases **pain** is present, but only when an attempt is made to write.

The characteristic feature of the condition is the appearance of a spasm in the muscles of the hand only in the act of writing, but absence in any other act executed by the same muscles. However in cases of long duration any fine and delicate act of the fingers may bring on an attack.

Paralytic Form.—It consists of a sudden sensation of fatigue and numbness in the hand while writing. The hand remains applied to the paper. As soon as the penholder is removed, the sensation disappears. A true paralysis of the adductors of the thumb has been observed.

Tremulous Form.—Instead of a **spasm** or **paresis** there may be only a **tremor** in the fingers or in the entire arm during the act of writing.

The **course** of writer's cramp is usually chronic. Periods of amelioration and aggravation are observed. It may last an indefinite time. Recurrences are frequent. Recoveries are possible.

Other Occupation Spasms

Pianist's Cramp.—It is mostly of the paralytic form. It manifests itself as a sudden functional inability to continue playing. The right hand is more frequently affected than the left, although both hands may become involved.

Violinist's Cramp may present itself in the paralytic and spastic forms and mostly in the left hand.

Telegraphist's Cramp presents the same course and the same varieties as writer's cramp. In one of my patients there was also pain along the nerve-trunks of the right arm.

Shoemaker's, Tailor's, Seamstress's Cramp occurs in the muscles which are exercised in the act of sewing or cutting with large scissors.

In **blacksmiths** the cramp is localized in the biceps and deltoid muscles.

The **lower extremities** are rarely affected.

Dancers' Cramp occurs usually in the thigh muscles.

The **face** is also rarely involved. In **watchmakers** and **trumpet-players** a spasm of orbicularis palpebrarum has been observed.

Pathogenesis.—Various opinions have been expressed concerning the nature of occupation neuroses. Some believe in a **muscular** origin, others that the condition is due to an irritation of the **peripheral nerves** and still others in a **central** cause.

The fact that a muscle or a group of muscles will be thrown into a state of spasm only when they are called upon to execute a certain given act and contract normally in other acts speaks **against** the muscular theory (muscular traumatism and myositis). On the other hand, there are many individuals who write daily a great deal, pianists who play long hours, and still do not become affected with writer's or pianist's cramp. It is evident that only a certain class of persons is subject to this nervous disorder. The latter is met with in **predisposed** or **neuropathic** individuals and the disturbance is dependent upon a mental cause. It is sometimes associated with other mental disturbances, as phobias, abulias (see Neurasthenic Insanities). Not infrequently it is observed in several members of the same family. Some writers believe with Duchenne that there is a disturb-

ance in the center of coördination. Others think that the cramp is due to a neuralgia caused by molecular changes in the sensory fibers.

Treatment.—Abstention from work which causes the cramp is the first indication. General hygienic measures with massage and hydrotherapy are beneficial in view of the neuropathic make-up of the patients. Local massage may be of use. Electricity has given no appreciable results. No special internal medication is to be mentioned. I obtained very satisfactory and sometimes perfect results from Bier's method. In a series of cases (published in the *Therapeutic Gazette*, 1908) I applied this treatment systematically in every case to the exclusion of all other treatment and the patients were allowed to continue their work moderately. I found that in some cases a few applications of the bandage above the elbow—for an hour twice a day—gave the patients great relief. Complete recovery also followed in some cases. It is apparent that the circulation in the affected muscles has somewhat to do with the cramp. **Reëducation** is a good method. The patient is advised to practice his usual exercises for a very brief time every day and gradually increase the duration and the number of the séances. Benedikt (*Wiener klin. Wochenschr.*, 1911) obtained very satisfactory results from injections of 2 per cent. solution of phenol. He believes that the original source of occupation neurosis is in the tendons and muscles. Tender points can always be found in the tendons of the wrist and hand or in the heads of the tendons in the elbow or shoulder. Injection of weak phenol solution at these tender points restores the condition to normal. Sometimes several injections are necessary.

PARALYSIS AGITANS (PARKINSON'S DISEASE)

Shaking Palsy

Symptoms.—Tremor, attitude, gait, facies, are the elements presenting special features in the disease.

Tremor.—It is present in the majority of cases. It is **passive** in character, viz. it is present when the body is at rest. It usually disappears upon voluntary movements, but returns if the latter are sustained.

The tremor may affect the entire body, but more frequently the upper extremities and particularly the hands and fingers. Sometimes all the fingers are agitated, but the **thumb** is especially affected. It moves to and fro over the palmar surface of the other fingers in a continuous and slow manner; its oscillations remind the act of rolling pills or crumbling bread. The tremor is **rhythmical.**

The tremor decreases from the distal end toward the root of the limb, so that it is not perceptible at the shoulder.

In the lower extremities the foot is particularly affected. When the patient is seated, the toes are held against the floor, but the heel keeps on striking the floor in a continuous and rhythmical manner.

The tremor of the head is usually transmitted by the arms. When there is a primary tremor of the head, it is the result of a contraction of the neck muscles. The tremor is then perceptible on the lips. The patient gives then the impression of muttering silently.

The tremor of the extremities as a rule does not interfere with ordinary active movements, provided the latter are not prolonged. Writing is difficult.

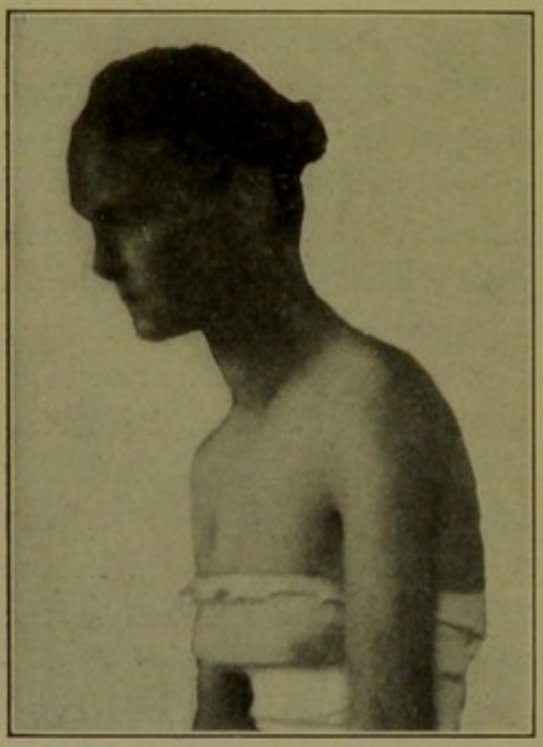


FIG. 160.

The tremor disappears during sleep. Emotions and exertion increase it. A continuous vibration of the body occurring, for example, in traveling decreases the intensity of the tremor and renders the patient more comfortable.

Attitude.—In a typical case the patient's head is inclined forward and as if fixed to the trunk, the back is curved (kyphosis). He holds himself rigid, turns, walks, sits down, gets off his chair as one rigid mass (rod-like). This condition is due to the **muscular rigidity** which is so characteristic of Parkinson's disease. Upon passive movements considerable resistance is felt. Although in the majority of cases the body is in a state of semiflexion, there are also cases of **extension** type; the head is held backward, and instead of kyphosis there is lordosis of the spine. The movements of the body are naturally slow and monotonous.

It is well to remember that the muscular rigidity is not that of organic nervous nature, as for example is spastic paraplegia or hemiplegia. It is a rigidity of a cadaver, as Bloch has well said. The rigidity produces a certain degree of muscular weakness, which disables the patient for work or even for ordinary voluntary acts. A true paresis may occur only in advanced stages. The patient is in a state of restlessness; there is a tendency to change position.

Gait.—In mild cases the only peculiarities noticeable are **small quick steps** and **slow gait**. In advanced cases the following is observed. When the patient attempts to walk, he inclines the body forward, steps first on his toes and then for fear of falling he is obliged to accelerate his gait and

run. In some cases there is only an accelerated gait (**festination**), in others a distinct tendency to fall forward (**propulsion**). The latter is easily noticed when a slight push is given on the back. The patient will keep on running until an obstacle is met. The same phenomenon is observed when the patient is pushed backward (**retropulsion**) or laterally (**lateropulsion**).

Facies.—It is typical. It is characterized by immobility of features. It is mask-like. It gives the impression of astonishment, surprise, fright. This expression is independent of the inner feelings of the patient. The conditions of the facies as well as the fixed expression of the eyes are due to the rigidity of the facial and ocular muscles, respectively.

Other Symptoms.—The **speech** is not infrequently changed. Monotonous voice and rapidity of words are its characteristics.

Sensory disturbances are only of a **subjective** nature. The patients frequently complain of rheumatic pain in the limbs, of muscular fatigue and numbness. Sensation of **heat** is continuously present and sometimes accompanied by abundant perspiration. The tendon **reflexes** are either normal or diminished or more frequently exaggerated. Ankle-clonus, Babinski and paradoxical phenomena are absent.

Trophic disturbances are usually absent. In some cases there is a thickening and hardening of the skin, especially on the face. Rheumatoid deformities of hands and feet are also observed. Muscular atrophy occurs in the last stages of the disease.

Vaso-motor Disturbances have been mentioned above. They consist of a feeling of heat and hyperhidrosis. Cyanosis of the extremities, also localized œdemata, may occur.

The **mentality** is usually intact. However there is a certain degree of indifference, apathy and even depression. There is an intellectual fixation alongside the physical fixation. This mental condition varies from time to time in the same individual. In some cases there may be mental failure and even a certain degree of dementia.

Course, Duration, Prognosis.—The course of paralysis agitans is slow. In some cases it develops suddenly, as for example in cases following a trauma or emotion. In the majority of cases it commences with a tremor in one extremity, most frequently in one of the upper extremities. Gradually the lower extremity of the same side becomes affected. Later the opposite side is invaded. The development of the symptoms is slow, but progressive. Several years elapse before the disease is generalized. Remissions with amelioration of the symptoms may occur, but no complete disappearance of the latter.

The disease may last many years, from ten to thirty. Death usually

intervenes from some intercurrent disease, particularly from pneumonia. If this is not the case, the disease progresses until complete physical disability occurs. The patient is then confined to bed; bed-sores and cachexia hasten death.

Forms.—The above clinical picture presents the most frequent variety of paralysis agitans. There is also a form, in which the tremor is absent and the disease is then recognized by the rigidity, facies and attitude. In another variety the tremor alone is present or else the rigidity is extremely slight. In some cases the tremor is not only passive, but also intentional (similar to that of multiple sclerosis). There are also cases in which instead of flexion of the trunk and extremities there is extension.

Diagnosis.—The symptoms as a rule are so typical that an error in diagnosis is rare. The variety without tremor may present some difficulties.

The **tremor** may lead sometimes to an erroneous diagnosis, especially when besides being passive it is also intentional. **Multiple sclerosis** should be then thought of. But in the latter disease there are also nystagmus, scanning speech and absence of passive tremor.

In **paresis** the tremor is also intentional, but there are also changes in speech, in the pupillary reflexes; finally mental symptoms.

A **senile** tremor is usually passive and intentional, but it is not associated with other symptoms characteristic of paralysis agitans. Besides, in the senile form the head is generally affected early.

Hysteria may simulate paralysis agitans by its tremor. In one of my cases it was limited to one hand, but the movements were somewhat of wider range, they were present when the arm was at rest and upon voluntary movements; the characteristic attitude, facies and gait of Parkinson's disease were absent.

Organic hemiplegia may sometimes be accompanied by a tremor on the same side and thus simulate a form of paralysis agitans in which the tremor is confined to one hand. In the majority of such cases the diagnosis will not be difficult in view of the characteristic symptoms of both affections, but there are cases which present almost insurmountable difficulties in differentiating them.

Pathogenesis.—The post-mortem findings present nothing definite as to the nature of the disease. While some autopsies are negative, others show perivascular sclerosis in the gray and white matter of the brain or spinal cord. The most recent work on the subject is that of G. Maillard, who considers the disease as due to arteriosclerotic changes in the **red nucleus.** On the contrary a great many observers found changes only in the muscles, viz. nuclear proliferation in the sarcolemma, atrophy of

some fibers, diminution of muscle-spindle, and they place the disease among myopathies (see this chapter). In the peripheral nerves increase of interstitial tissue and slight degenerative changes in the fibers have been found in some cases.

Some writers believe that the disease is due to some toxic agent produced by the parathyroid bodies (Lundborg, Berkeley and others). They base their opinion on this observation that the symptoms following parathyroidectomy resemble much those of paralysis agitans. Until a solid anatomical basis is found paralysis agitans should be considered as a functional nervous disease. This is the opinion of the majority of authors.

Etiology.—The exciting causes are **traumatism** and **emotions** of a depressive character.

A neuropathic tendency plays an important predisposing part.

The disease affects both sexes, but men more frequently than women. The usual age at which it occurs is beween forty and sixty, although it may develop before twenty, as some records show.

Treatment.—The **tremor** is sometimes ameliorated by trepidation in a carriage or train. One of my patients obtained great relief by riding on a train two hours every day. For the same reason I treated another of my patients with a very frequently interrupted faradic current and succeeded in diminishing the intensity of the tremor.

Internally **hyoscine hydrobromate** in gr. 1/100 doses two or three times a day relieves sometimes the tremor as well as the rigidity. Among other drugs may be mentioned: cannabis indica, codein, opium, arsenic, bromides, veratrum viride.

Warm Baths are useful to control the rigidity. Massage gently applied may do some good. Systematic exercises sometimes give satisfactory results in decreasing the rigidity.

Rest, which is so beneficial in other neuroses, is contraindicated here. However violent exercises or undue fatigue must be avoided.

According to the latest researches, particularly of Loeb and J. B. MacCallum, also of W. G. MacCallum and C. Voegtlin, there is a relationship between various twitchings and calcium metabolism and the function of the parathyroid glands. There is consequently a therapeutic indication for the use of calcium salts and of parathyroids in paralysis agitans.

AKINESIA ALGERA

Under this name Moebius in 1891 described a symptom-group characterized by an inability to move about because of pain. If some slight movements are possible, the condition is called "**dyskinesia algera.**"

At first only forced movements are painful, but gradually the most insignificant displacements become intolerable. The pain appears upon voluntary movements, persists after the movements ceased. Little by little any movement becomes impossible. There is, however, **no genuine paralysis.**

Pain is present not only in the muscles executing movements, but it extends to the remotest parts of the body. Thus **headache** may develop. The latter may appear upon the least mental exertion. Sometimes mental fatigue provokes pain in the entire body, especially in those muscles that are called upon to contract.

In some cases a **light** causes pain (**dysopsia algera**). In Oppenheim's patient the sight of white objects caused pain. In analogy with painful motions the light phenomenon can be called **"painful photophobia."** It is particularly noticeable upon attempts of reading.

Some patients feel a fatigue in the head and severe headache upon the least effort to read, write or speak. It is the so-called **apraxia algera.** In Erb's case **hearing** caused pain, so that the patient could not converse. In another of Oppenheim's patients **ingestion of food** awakened pain, while there was no disease of the stomach.

Neftel described an analogous form under the name of **"atremia,"** where the patient while lying can move around without pain, but as soon as he attempts to get up, he suffers in the back and head.

Course, Duration, Prognosis.—In the majority of cases the disease is rebellious to treatment and may last an indefinite time. Ameliorations occur, but recurrences are frequent. Complete recovery is also possible. Mental disturbances, as delusions and hallucinations, not infrequently develop in the course of the disease. Some patients die in a fully developed psychosis. The prognosis is bad in the majority of cases.

Etiology and Pathogenesis.—Functional nervous diseases (neurasthenia, hysteria, hypochondria) play a very important **predisposing** rôle. Their association with akinesia algera is quite frequent.

The consensus of opinion is that the condition is a neurosis and should be placed alongside other well-known functional nervous diseases, that the **pain is central** with complete integrity of the nervous tissue. It is a fixed sensation, a disease of attention, a painful hallucinatory obsession.

Treatment.—Suggestion and autosuggestion are the main elements of treatment. What a patient can accomplish by exercising his will power and with a continuous effort to help himself can be seen from Erb's case, in which the patient after remaining in bed nineteen years finally learned how to convince himself that his trouble was not real, that it was only hallucinatory. The patient improved considerably.

Reëducation of movements with a great deal of persistence and persuasion tactfully carried out, aided by gentle massage, can in some cases accomplish much good. Oppenheim's patient mentioned above recovered by wearing blue glasses, also by taking arsenic.

HEADACHE

Cephalalgia

Headache is not a disease but a nervous symptom accompanying various diseases of various organs. It is a very frequent phenomenon and in the majority of cases has no special diagnostic significance. Sometimes, however, it is so conspicuous by its intensity and character that its diagnostic value is enormous.

Diseases and Conditions that Cause Headache

1. **Circulatory Disorders. Anæmia, Hyperæmia.**—In all forms of **anæmia** headache is a common occurrence. It is particularly observed when an effort is made by the patient. In aortic diseases cerebral anemia occurs. The headache is then accompanied by vertigo.

In **passive hyperæmia** pressure on the neck (tumors, tight collars, etc.), in **active hyperæmia** certain drugs (caffeine, nitroglycerine, alcohol), undue physical and mental effort, violent emotions, overheated room, cardiac diseases, especially mitral lesions, are the causes of pain in the head.

2. **Meningitis** (localized or diffuse) and **meningeal hemorrhages,** also **acute encephalitis.**

3. **Toxæmia** in **infectious diseases. Perverted metabolism,** such as met with in diabetes, gout, chronic rheumatism, nephritis, albuminuria, uræmia, gastro-intestinal disorders (constipation); **intoxications,** such as lead, alcohol, tobacco, opium, carbonic acid gas.

4. **Syphilis** through a degenerative condition of the blood vessels or gummatous deposits in the meninges.

5. **Increased Intracranial Pressure** through tumors, aneurisms of the brain (see Tumors of Brain).

6. **Hysteria, Neurasthenia.**

7. **Traumatism** of the head.

8. Diseases of the skull and of its cavities (nasal, frontal, etc.).

9. **Reflex irritation,** such as errors of refraction, naso-pharyngeal adenoids, diseases of genitalia, etc.

Pathogenesis.—It is generally conceded that headache is directly due to an irritation of the sensory filaments distributed in the meninges of

the brain, especially the dura. In **organic** cases the irritation is caused by some intracranial disease. In the **functional** cases some irritant (toxic or other) circulating in the blood vessels reaches the sensory ends of the meningeal nerve-supply.

Character of Headache in Various Diseases

1. Headache due to anæmia, chlorosis or loss of blood is usually **diffuse** and accompanied by a sensation of **pressure.** It is severe and aggravated upon an effort. Vertigo is frequently present.

In hyperæmia (passive or active) the headache is diffuse, continuous and of a **throbbing nature.** Horizontal position aggravates it. In cerebral congestion caused by a cardiac lesion, there is a venous stasis and ædema of the brain. The headache is accompanied by somnolence and even delirium.

2. In **Meningitis** the headache may be localized or generalized. It is usually continuous and presents exacerbations. In this disease the headache is of diagnostic importance, especially in the epidemic cerebro-spinal or tubercular form of meningitis, in which it is a very early symptom and of great severity.

In tubercular meningitis headache is less intense than in the epidemic meningitis. In the latter the pain extends down to the neck and spine. In both forms the headache is persistent.

In **Pachymeningitis** the pain in the head is at first localized.

3. In **infectious diseases** headache appears in the prodromal period and usually persists through the entire course of the disease. It is usually dull and continuous.

In **influenza** the headache is diffuse and particularly marked in the frontal sinuses. This disease is **frequently** followed by headache of unusual severity.

Headache caused by poisons is usually **frontal, dull** and **quite severe.** It persists even after the other symptoms have disappeared.

4. Imperfect metabolism of gastro-intestinal origin (**constipation**) produces a temporal or occipital headache.

5. **Syphilitic** headache is mostly nocturnal in character. It is dull, continuous, with nocturnal exacerbations. It may be general or confined to one side of the skull. It is frontal or parietal most frequently. In the latter case the skull is tender on percussion. Headache is the earliest symptom of cerebral syphilis. It is due to involvement of the meninges by gummata. It is usually relieved by antisyphilitic remedies, but recurs readily.

6. Headache is a very significant symptom in intracranial diseases, as tumor, abscess, etc. It is of unusual severity and persistent. Percussion or deep pressure on the skull provokes pain. In case of abscess due to an ear lesion, a disease of the nose, of frontal sinus, etc., the pain will be localized in the corresponding regions.

In tumors of the cerebellum the headache is dull, boring and sometimes sharp, but always intense. It is localized frequently in the occipital region.

7. In **Neurasthenia** the headache is in the form of continuous pressure or constriction; it resembles a sensation of a tight iron band around the head. It is usually diffuse, but it may be also localized. Neurasthenics often complain of various distressing sensations in the head, as heat, emptiness or else fullness, which alternate with or accompany headache.

In **Hysteria** the headache is in the vertex. It is boring and localized, as "clavus," viz. a sensation of a nail being driven into the head.

8. In **traumata** of the head and diseases of the skull the pain is usually circumscribed and corresponds to the injured or diseased area, although it may spread from the latter and become diffuse.

9. **Reflex Headache** is quite common. When it is due to errors of refraction, it is frontal or temporal. Nasal diseases give headache in the vertex or temporal region. Persistent headache is present in diseases of the auditory apparatus. A thorough examination in each case of headache for possible reflex causes is necessary.

Treatment.—Determination of the cause of headache is the first indication before the treatment is instituted. The above described etiological factors should be borne in mind. Every case must be scrupulously examined, as the complete removal of the headache presents frequently great difficulties. As soon as the cause of headache is ascertained, all medical or surgical measures should be employed for its prompt removal.

In cases with circulatory disturbances the latter must be relieved. In anæmia rest, absolute or partial, according to the case, is necessary. Good nutritious food, iron and arsenic are appropriate. In hyperæmia, besides the removal of the original cause, purgatives are indicated. In meningitis purgations and local abstraction of blood are useful.

In gastro-intestinal disorders their immediate correction is necessary. Regulation of diet, avoidance of nitrogenous food, of intoxicants, light but nutritious food according to the case, daily evacuations, also administration of hydrochloric acid in case of diminished secretion in the stomach, and of an alkaline in case of increased secretion of hydrochloric acid, administration of a stimulant stomachic in case of sluggish digestion—all these means are indicated for treatment of headache. Headache caused

by certain poisons will be treated by measures of elimination and antidotes, also by removal of the patient from exposure to the poisons. In cases of lead poisoning besides systematic elimination, iodides are beneficial.

Symptomatic Treatment of Headache.—Relief of pain can be obtained from coal-tar products. Aspirin, sodium salicylate, antipyrin, bromides, are useful. A combination of aspirin, gr. v, phenacetine, gr. ii, and caffein citrate, gr. j, administered every two hours, has given me very satisfactory results. In case of malaria quinine is advisable. For headache of syphilitic nature salvarsan, mercurials or iodides or both will give relief.

Local application of extreme heat or cold, counter-irritation, application of menthol, alcohol, chloroform, ether, may give some relief.

Complete isolation with absolute rest sometimes gives relief.

In organic headache (tumor, abscess, etc.) surgical measures are the only means, as it is unusually rebellious to internal medications.

Morphia should be avoided, as the pernicious habit is easily acquired. In infectious diseases headache is caused by a slight inflammation of the meninges, which is indicated by increase of pressure and of albumen in the cerebro-spinal fluid. In such cases a lumbar puncture, and withdrawal of 10–20–30 c.c. of cerebro-spinal fluid, may give considerable relief. Recent observations of Roger and Baumel (*Revue. de Méd. No.* 1, 1913) are very conclusive.

Muscular or Indurative Headache

This form of headache is due to a rheumatic infiltration, a chronic myositis of the muscles of the neck. The cervical muscles are swollen and tender to touch. The tenderness exists at the insertions of the tendons, spinous processes, mastoid processes, clavicle and the muscles themselves. In subacute myositis, the swelling of the muscles has an elastic consistency. In chronic myositis hard nodules are felt in the muscles. The skin covering the swollen muscles is infiltrated and thick. Histological studies show that the infiltrations are due to a proliferation of the connective tissue, at first œdematous, later fibrous and accompanied by endoperiarteritis (Lorenz and Stockman). It is therefore a trophic disturbance.

The above mentioned **tenderness** is present not only in the insertions of the tendons but also in the infiltrated masses. Müller (*Deut. Ztschr. f. Nerv.*, 1910) calls attention to **hypertonicity** of the affected muscles: the muscles of the neck are rigid and when the patient wishes to turn his head, the entire body moves with it. The pain produced by muscular induration

may be **diffuse** and affect the entire head, or **migrainic** affecting one side of the head or else **neuralgic.** These three varieties may alternate in the same individual.

Pathogenesis.—The headache can be explained either by an involvement of the sensory fibers passing through the infiltrated muscles, or by irritation of the sympathetic. In fact Nörstrom and Hartenberg observed in migrainic patients a swelling of the upper and middle cervical sympathetic. It stands to reason that in swelling of the muscles of the neck the sympathetic is being irritated, hence the headache. Müller also believes that the swollen muscles compress the jugular veins, the return circulation is interfered with, a cerebral congestion is the result, hence the headache.

Hartenberg believes that the chief cause of these muscular infiltrations lies in an insufficiency of arterial, venous and lymphatic circulation which is the result of an insufficiency of muscular activity. **Cold,** which decreases the nutritive and circulatory conditions, is capable to produce the lesion.

Treatment.—Avoidance of cold, general hygiene, hydrotherapy, are measures not to be neglected. Galvanism applied to the affected muscle in the beginning is very useful. Massage should be employed only after the acute symptoms have subsided; it is contraindicated when the muscles are very tender. Internally cannabis indica has been advised by Hartenberg in very rebellious cases.

Migraine. Hemicrania

Under this name is known a variety of headache which occurs in **paroxysms,** is confined to **one side** of the head, and is accompanied by nausea, vomiting, vertigo and ocular phenomena.

Symptoms.—The onset is usually, but not always, preceded by **prodromal** symptoms. They may be either depression, apathy, somnolence or, on the contrary, exaltation with a ravenous appetite.

The **pain** sets in ordinarily in the morning, when the patient gets awake. At first it is dull, but gradually increases as the day advances. It soon becomes severe and even intolerable. The patient compares it to a tearing, breaking, boring. The least motion of the head or the act of coughing, sneezing, exaggerates it. **It is confined mostly to one side of the head** and to the left most frequently. It may also spread to the other side. It may also alternate in various attacks. It may appear first in the orbital or temporal region and from this point spread to the entire half of the head.

During the attack an irritation of any of the special senses aggravates the pain. A loud sound, a sharp odor, a bright light, become intolerable.

For this reason the patient instinctively isclates himself, closes up the windows, the doors, and seeks quietness. When the headache reaches the maximum, **vomiting** ocurs. This is a frequent symptom and it may take place also at the onset of an attack. It is accompanied by vertigo. The patient rejects a bilious, mucous fluid. It is brought on by the least amount of fluid or food and sometimes it occurs before breakfast. There may be several attacks of vomiting. Sometimes an attack of vomiting ends the attack of migraine. The patient then feels considerably relieved and the headache rapidly disappears. In a large number of cases the final vomiting spell is followed by a deep sleep. When the patient gets awake, he is totally relieved and feels well. Sometimes, however, there is a feeling of lassitude and a dullness in the head which persist for several days.

As **accessory symptoms** of migrainic attacks can be mentioned lachrymation, unilateral or bilateral, sweating, polyuria, coldness of the extremities and exceptionally hemorrhages.

Forms of Migraine

1. **Ophthalmic Migraine.**—It is characterized by **visual** disturbances **at the onset** of an attack. The most important among them is a **scotoma** (scotoma scintillans). It is a dark-grayish spot in the visual field, surrounded by a bright border of various colors. Sometimes instead of a scotoma the visual field is covered by glaring zig-zag lights. In other cases there is a distinct blindness of the visual field in the form of homonymous hemianopsia. There may be also a transitory amaurosis, photophobia with interocular pain. In some cases the migraine may be accompanied or followed by temporary ophthalmoplegic symptoms such as strabismus, diplopia, ptosis, loss of accommodation. In exceptional cases they remain permanent.

Ophthalmic migraine may be associated with **motor, sensory** and **psychic** phenomena.

A **paresis** of the side opposite to the seat of head-pain, **paræsthesias** on one or both sides of the body are sometimes observed.

Aphasia (motor) with agraphia is occasionally observed. One of my patients had three attacks of a mild right hemiplegia with slight aphasia. During these attacks the right knee-jerk was increased and the paradoxical reflex was evident. These manifestations lasted but a few hours.

Cerebellar ataxia with its characteristic disturbance of equilibration was observed by Oppenheim.

All these phenomena are usually transitory and last a short time;

they may precede the headache and vomiting or persist during the entire attack of migraine.

The **psychic** symptoms are various. There may be only a temporary **amnesia, visual hallucinations, depression, apathy,** which disappear with the attack. There may also develop true **psychoses,** as Krafft-Ebing, Mingazzini and myself have shown.

In my recent study (*Journal of American Medical Association*, January 5, 1907) of the subject, I described twelve personal cases of typical psychoses observed in migrainic attacks. In all of them I have invariably found three mental states, viz. (1) **Confusion,** (2) **stupor,** with hallucinations and unsystematized delusions, and (3) **delirium.** The hallucinations were mostly visual, although auditory and gustatory were also found in some of the twelve cases. The confusional state predominated in all my patients. It was quite frequently accompanied by illusions of identity, incoherence of thoughts and disturbance of orientation. The delusions were of a fleeting character. In the majority of cases the mental symptoms developed during the attacks when the headache reached its climax and disappeared with the headache.

2. **Hemicrania Sympathico-tonica (White Migraine).**—It is characterized by symptoms of **irritation** of the cervical sympathetic nerve, viz. pallor of the integument of the head on the side of the pain; the temporal artery is of high tension, the local temperature is lowered; there is also retraction of the eyeglobe and mydriasis.

3. **Hemicrania Sympathico-paralytica (Red Migraine)** is characterized by symptoms of **paralysis** of the cervical sympathetic nerve, viz. redness of the integument of the head on the affected side, lachrymation, photophobia, contraction of the pupil, elevation of the local temperature, unilateral hyperhidrosis; the temporal artery is distended.

4. **Migraine in its Relation to Epilepsy.**—It is well known that both neuroses may be associated and that the first may be **equivalent** to epileptic seizures. That the analogy between them is considerable can be seen from this fact that in both the attacks are sometimes preceded by **auras** (motor, sensory, psychic), that the onset may be sudden, that an attack is followed by a sense of exhaustion and sometimes by unilateral paralysis or paresis. Finally the abortive cases of migraine (see below) are almost identical to attacks of petit mal.

5. **Abortive Migraine.**—It consists of incomplete attacks (without vomiting or vertigo) or else sudden attacks of vomiting without headache. Sometimes it is only an attack of vertigo. This form alternates frequently with the typical attacks of migraine.

Course, Duration, Prognosis.—A typical attack commences in the

morning, gradually increases in severity and terminates in the evening with vomiting and sleep. When he wakes up, he feels refreshed and well. An individual attack may last from a couple of hours to twenty-four or forty-eight hours. In some patients there is a great regularity in the periodicity of attacks. In such cases it comes on once every month or every two months. In women it may coincide with menstruation. In other cases the attacks are irregular and develop only after some excess (alcoholic or other) or after some indulgence in eating. The disease lasts many years or even the entire life. Usually with advent of old age it decreases in intensity and disappears. Sometimes an intercurrent disease, a trauma or shock, makes migraine disappear completely or for many years. In such cases some other neurosis (hysteria, epilepsy, etc.) develops instead of it. In some patients after many years of periodical and regular attacks the interval between them becomes shorter and shorter and finally the headache becomes continuous. A sort of **migrainic status** is thus established. The **Prognosis** is unfavorable as to recovery, but not to life.

Diagnosis.—The symptoms ordinarily are typical enough for a correct diagnosis.

In **cerebral tumors** there are also severe headache and vomiting, but there is no interval of "well being" characteristic of migraine. Besides, the changes in the eyegrounds will aid in the diagnosis. **Tabes** and **Paresis** may present migraine as one of their initial symptoms. Syphilitic and malarial migraine will be promptly relieved by mercurials and quinine, respectively. The diagnosis between ophthalmic migraine and **neuralgia** of the ophthalmic branch of the trigeminus is not always easy. The following differential signs may be useful. In migraine there is nausea, vomiting. In neuralgia they are absent. In migraine the pain is continuous for several hours. In neuralgia it is in paroxysms. In migraine remissions are of long duration—hours or days. In neuralgia they are very brief. In migraine photophobia, but not in neuralgia. In migraine scotomata, but not in neuralgia. In migraine the pain often spreads to the other side of the head. In neuralgia the pain is strictly confined to one side.

Etiology and Pathogenesis.—The most important element in the etiology is **predisposition.** The disease is very frequently found to be **hereditary.** A **neuropathic** personal or family history is frequently found. An association of migraine with an arthritic diathesis is a common observation.

The most recent researches in the domain of physiologic chemistry lead to the view that migraine finds its explanation in **autointoxication.** Whether it is uric acid or a special ferment or ptomain is not completely elucidated. The fact is that in a large majority of cases there is an ele-

ment of gastro-intestinal disorder and that arthritic, gouty, asthmatic, obese and constipated individuals are most frequently affected.

Our recent studies on the function of the ductless glands permit also to suppose that their derangement is apt to play a certain part in the causation of migraine.

Suffice it to mention the fact that in pregnancy, for example, there is a functional hyperactivity of the thyroid gland and migrainic patients are frequently free from attacks of migraine at that time. General lassitude, anorexia, constipation, obesity, falling of hair, are observed in migrainic individuals, also in cases with diminished thyroid function.

A poison, whatever its source may be, circulating in the blood vessels irritates the sensory filaments of the dura and produces the syndrome of migraine.

As exciting causes may be mentioned: disturbances of digestion, excesses, masturbation, irregular sleep, physical and mental fatigue, emotions, lack of fresh air, certain odors, loud sounds. Pregnancy may provoke or arrest the attacks. Women are more often affected than men.

Treatment.—A patient suffering from migraine should be energetically treated **between** the attacks. Proper measures taken at that time will in a great many cases succeed in increasing the intervals between individual attacks and in some cases accomplish a complete cure.

As said above, migraine is due in the majority cf cases to autointoxication. It is therefore toward the latter that our therapeutic efforts must be directed. Each migrainic individual should first of all be put on a special diet and follow a certain mode of living. It has been my practice to remove from the diet meats, stimulants of any kind, including tea and coffee, sweets, pastry. Starchy food is allowed only in extremely small quantities. Milk is of course desirable, but it is not tolerated by every patient; in the latter case I substitute it by either butter milk, skimmed milk, kephir, koumyss or else by plain water. In a great many cases, however, I succeeded in having the patient accustomed to milk by giving it at first in very small quantities and then **very gradually** increasing the amount. The amount of food of each meal should be moderate and taken regularly. Fruit, eggs, milk, green vegetables, crackers, custards, junkets, gelatin, are the only articles allowed. As drink, plain water or some gaseous mineral water, like apollinaris, lemonade, are permissible. Saline purgatives should be administered at first twice a week and later once a week for a long period.

Hydrotherapy in the form of cold shower baths of a minute's duration and followed by massage twice a day is very beneficial.

Daily walks two or three times a day of an hour each are advisable.

A quiet life, free from undue emotions, retiring early, are also necessary.

Tobacco must be used very moderately and, if possible, abandoned.

As to medications, a stomachic, as nux vomica together with hydrochloric acid and gentian, taken regularly before meals is of benefit. Any dyspeptic disturbance should be corrected and treated accordingly. A thorough examination of all the organs must be made as early as possible and treated if necessary. In case of anæmia, iron and arsenic should be given. Arsenic is highly praised by Oppenheim. The urine should be frequently examined. Any diseased condition of nasal, pharyngeal and other cavities, errors of refraction, diseases of the ears, etc., must be taken care of.

When an attack of migraine occurs, all food must be immediately withdrawn, the patient put to bed, the room made dark and all noises avoided. An ice cap or else a hot water bag are to be applied to the head. Application of menthol, a spray of ether or alcohol to the forehead or temples may give some relief. The vomiting is sometimes controlled by internal administration of small pieces of ice or chloroform water.

All these means should be tried before medications in every case of migraine. The following are the remedies advised for combating the pains: Coal-tar products, aspirin, salicylates, phenacetin, antipyrin. A combination which has given me satisfactory results in many cases is: Aspirin, gr. v, phenacetin, gr. ii, caffeine citrate, gr. j, to be taken every hour or two hours until relieved. Aspirin and codein form a good combination. Bromides succeed sometimes when other drugs fail.

In the sympathico-tonic form of migraine (see above) nitro-glycerine is useful. In the sympathico-paralytic form (see above) ergot is advisable. Amyl-nitrite in inhalations (five drops on a handkerchief) sometimes cuts short an attack. **Morphia should be avoided.**

In the discussion on the pathogenesis of migraine its relation to disturbances of thyroid function was indicated. In a series of cases studied and reported by me in the *Therapeutic Gazette*, 1907, I showed the very satisfactory results obtained in the treatment of migraine by **thyroid extract,** when every other medication failed. This treatment will yield good results only in, so to speak, "thyroid cases," viz. in cases with a deficient thyroid function.

VERTIGO

Vertigo is not a disease but a symptom. It may be encountered in various diseases and conditions. In Ménière's disease it is the most conspicuous symptom.

Nature of Vertigo.—The relation of the body to surrounding objects determines the so-called **"sense of space."** Should this relation be disturbed, our **orientation** and **equilibration** in the space will become irregular and **vertigo** will ensue.

In vertigo the orientation is first lost and the loss of equilibrium follows.

Orientation is the result of centripetal function. The pathway controlling it is composed: (1) of **sensory** fibers going from the periphery through the spinal ganglia and posterior columns, also direct cerebellar tract of the cord to their termination in the cortex of the cerebrum and cerebellum, respectively; (2) of **auditory** fibers originating in the labyrinth and ending either in the temporal lobe of the brain (cochlear nerve) or in the nucleus of Deiter in the medulla (vestibular nerve).

Equilibration is a centrifugal function. Its pathway consists of fibers of the pyramidal tract and of the descending cerebellar tract, also of Monakow's bundle, which all end in the anterior cornua of the spinal cord.

Disturbance in the harmonious function of those **pathways** or **centers** of orientation and equilibration results in vertigo.

Diseases and Conditions in which Vertigo Occurs

1. **Disorders in the Hearing Apparatus.**—Any disease of the ear, from a grave affection of the labyrinth to a simple accumulation of cerumen in the external ear, may produce vertigo. The most important form of aural vertigo is the so-called **Ménière's disease.** Suddenly the patient hears a noise in the ear, which is immediately followed by dimness of vision or double vision and sometimes nystagmus. Vertigo sets in at once and the patient falls. At that time he feels the floor sinking and himself revolving in a circle. He is nauseated and begins to vomit. Headache, coldness of the skin, pallor, accompany the vertigo. After the attack is over, the noise in the ear persists. If the attacks are frequent, hypoacusia or deafness gradually develops and vertigo becomes chronic.

The paroxysms usually last a few minutes. They may occur every day, week, month or even at longer intervals. The disease may last an indefinite time, but when recovery takes place, total deafness is established.

The disease is probably due to a labyrinthine involvement (progressive degeneration of the nerve-ends); a lesion of the cochlea causes deafness, and of the semicircular canal vertigo. The labyrinth is innervated by the vestibular branch of the eighth nerve. This branch is connected with the cerebellum, center of equilibrium. The ocular symptoms are due to the anatomical relation between the vestibular nerve and the nucleus of the

oculomotor nerve. To sum up, **vertigo, tinnitus auris, hypoacusia,** or **deafness** constitute the chief characteristic symptoms of Ménière's disease. Nausea, vomiting and nystagmus are not necessarily always present.

Diseases of the inner ear, of the middle ear, of the external ear, of the acoustic nerve, injuries of the petrous bone, meningitis, tabes, syphilis, gout, anæmia, arteriosclerosis, may be accompanied by Ménière's symptom-group, but it has also been observed in persons free from any disease. In such cases a vaso-motor disturbance in the labyrinth is probably at fault.

Forms of Ménière's Disease. (a) Charcot described a **continuous form** of the disease. The auditory disorder and the hypoacusia, also the vertiginous state are permanent, so that the patient's gait is that of cerebellar type (gait of an inebriate). In addition to this continuous condition the patient has paroxysms of the acute form.

(b) In the **mild form** of Ménière's disease, which is the most frequent, all the typical symptoms are present but they are exceedingly slight Here the manifestations are not always referred to the real cause. Stomach, kidney, uterus, anæmia are frequently incriminated.

(c) **Incomplete forms** (formes frustes) were described by Frankl-Hochwart. One or two chief elements of the disease may be wanting. Thus we observe: a form with hypoacusia, a form without tinnitus, a form with a mild vertigo.

(d) **Apoplectic form** is probably due to a labyrinthine hemorrhage. Its onset is sudden. It is usually accompanied by a complete and irremediable loss of hearing. This is the form described originally by Ménière. Loss of consciousness is sometimes present. It is usually met with in individuals suffering from syphilis, tabes, arteriosclerosis, nephritis, infectious diseases. In **traumatism** of the ear labyrinthine hemorrhage and therefore apoplectic form of Ménière's disease may occur.

In making a diagnosis of Ménière's disease, syncope, hysterical or epileptic attacks, indigestion, should be thought of. To determine labyrinthine involvement, the tests described in the chapter on Nystagmus must be taken into consideration (see page 363.)

2. **Disorders in the Visual Apparatus.**—Ocular palsies, diplopia, nystagmus, sudden passage from obscurity to light, are common causes of vertigo, and if the latter is severe, nausea and vomiting may follow.

3. **Disturbances in the Central Nervous System.**—Tabes, multiple sclerosis, may be accompanied by vertigo. In the latter affection it is quite frequent and it is an indication of bulbar involvement.

In diseases of the brain (tumors) and especially of the cerebellum vertigo is one of the most important diagnostic symptoms. In cerebellar conditions the vertigo is of a rotatory type. Softening of the brain, circulatory changes, atheromatous condition of the cerebral blood vessels, are accompanied by vertigo. Diseases of the medulla, cerebellar peduncles and vestibular nerve, all partaking in the control of equilibrium, will cause vertigo.

4. **Disorders of Metabolism (Toxic Causes).**—Nephritis, gout, diabetes, gastro-intestinal disturbances, migraine, are not infrequently accompanied by vertigo.

Infectious diseases, intoxications (alcohol, lead, coffee, tobacco, quinine), intestinal parasites produce vertigo.

5. Hysteria and neurasthenia, exophthalmic goiter, mental diseases, may be accompanied by vertigo.

6. **Visceral Disturbances.**—In cardiac diseases, especially diseases of the aorta, vertigo is not infrequently present. Dilatation of the stomach may cause dizziness, but here the latter is probably due to auto-intoxication. Reflex vertigo may originate in the uterus, bladder, liver, kidney, larynx, nasal cavity.

7. **Sea-sickness,** traumatism, insolation are accompanied by vertigo.

8. **Paralytic vertigo** (Gerlier), which has been observed in Switzerland, is characterized by vertigo, weakness of the limbs and of the muscles of the neck, ptosis. It occurs in paroxysms. Nothing is known of the nature of this endemic affection.

Treatment.—The treatment of vertigo is closely connected with the management of the diseases in which it occurs. An effort must be made first of all to remove the cause.

In Ménière's disease Charcot's advice has given me the best results in some cases. Quinine hydrochlorate given in small doses, but frequently repeated, with milk diet and avoidance of stimulants yield very satisfactory results. The administration of quinine is based on this fact, that vertigo subsides with increase of deafness. Quinine weakens the hearing and it may therefore be useful. At first it will increase the noises of the ears and even the vertigo, but if persisted in, it will eventually relieve the vertigo. Aspirin, sodium salicylate, pilocarpine in hypodermic injections (5–10 minims of 2 per cent. solution every other day) may be useful. Purgatives, hot foot-baths and bleeding may be beneficial. Babinski obtained some relief from lumbar punctures. Krause, in 1902, and Frazier, in 1909, attempted intracranial division of the auditory nerve for relief of intractable tinnitus and aural vertigo respectively. The results have been encouraging.

CHAPTER XXVI

TRAUMATIC NEUROSES AND PSYCHOSES

RELATION OF ACCIDENTS TO FUNCTIONAL NERVOUS DISEASES AND PSYCHOSES; MEDICOLEGAL CONSIDERATIONS

The modern requirements in every sphere of human activities are highly contributory to accidental injuries. Traumata may lead to surgical and nervous disturbances. As we will be concerned exclusively with the latter, it is necessary first to emphasize what factors are the most frequent causes of this disorder.

Among all accidents, those caused by conveyances (railroads or trolley cars) give the largest contingent of victims. Organic injuries, such as fracture or dislocation of the vertebræ, tearing of the spinal cord or of peripheral nerves, fracture of the skull, followed by tearing of or hemorrhages in the cerebral tissue are discussed in their respective chapters. Functional nervous disorders and psychoses exclusively will be discussed here. The subject is an extremely important one for two reasons: (1) traumatic functional neuroses are exceedingly frequent; (2) the neuroses are very frequently misunderstood, and therefore a proper estimate of their value is not always given.

The functional nervous diseases produced or perhaps brought out by such accidents are: hysteria, neurasthenia, chorea, paralysis agitans, amnesia. A review of the clinical pictures is necessary for a proper appreciation of the medicolegal questions.

Hysteria.—When a collision, for example, occurs, the shock and the fright into which the passenger is thrown are sufficient causes to disturb the workings of the entire central nervous system or of its main centers. Supposing he or she during that time is thrown even not violently against the seat of the car, the anticipation alone of a possible severe injury or of a fatal injury, or else of immediate death, is capable of putting out of order the function of the nervous system. During the first few days, or even weeks, the patient still dreads that his life is in danger.

The essential features of hysteria are:

Sensory Symptoms.—Hyperæsthesia in areas or along the spine; loss of sensations in the distant segments of the limbs (glove-like and stocking-like anæsthesias); anæsthesia confined to an entire half of the body (hemi-

anæsthesia); anæsthesia of the pharynx, of the conjunctivæ, of the retina. The latter will be manifested by a contraction of the visual field. The special senses may also be affected. Sudden partial blindness or deafness (without material changes in the eyes or ears), vomiting occurring immediately after the accident and continuing for several days or weeks without any relation to the food and with an excellent appetite—all these symptoms occur quite frequently.

Motor Symptoms.—Palsies, contractures confined to segments of limbs, are not infrequent, but they are never accompanied by disturbances of reflexes usually seen in organic nervous diseases. The absence or exaggeration of knee-jerks, toe phenomena, muscular atrophies with reactions of degeneration accompany organic but not hysterical palsies.

The function of the viscera may also be disturbed. Hysterical aphonia, anuria, polyuria, retention of urine, are well known phenomena. In a case published by me in the *Medical Record*, August, 1900, a woman became anuric after an emotion.

Psychic symptoms are quite frequent. Temporary amnesia, capriciousness, inconsistencies in the ideas and conduct, dissociation of personality, hallucinations are possible occurrences in hysteria. Hysterical subjects are easily influenced to change their thoughts, to do certain acts, to acquire certain sensations, to execute or to adopt certain motor phenomena. Suggestion or auto-suggestion plays an enormous rôle. I have also seen cases of hysterical paroxysms following railroad accidents. A young girl of seventeen, while lying in bed, contorted herself, assumed the position of opisthotonos, was animated with a generalized tremor, so that she was unable to utter a word distinctly.

These are the characteristic manifestations in the motor, sensory, and psychic spheres of a hysterical individual. Of course, not all of them are simultaneously found in every case, but when some symptoms are present in the form and intensity as described above, the diagnosis of hysteria can be made without hesitation.

Neurasthenia.—This neurosis frequently follows accidents. Its main features are: physical exhaustion and undue irritability, so that the patient cannot stand the least contradiction or annoyance. The neurasthenic feels fatigue upon the least exhaustion. Mental processes are also sluggish; the least mental effort disables him from continuing his work. The patient complains also of backache, headache, and insomnia. The symptoms are mainly subjective. The objective symptoms are very few; tremor of the hands, increased reflexes, cold, clammy skin, and the special facies. The latter is that of a tired and depressed individual and one full of anxiety.

Chorea, with its irregular and incoherent muscular movements rapidly following one another, is too well known to dwell upon. It does not frequently follow accidents. Within the last three years I have seen only two cases, and then there was a history of previous attacks. The shock of the collision was immediately followed by a new attack of chorea.

The same remarks can be made about **Paralysis agitans.** In one case of a middle-aged woman the onset of the shaking palsy was traced to twenty-four hours after a trolley accident. In two other cases (a man and a woman) the shock of the accident was the direct cause of aggravation of the previously existing symptoms, which from that time kept on increasing in intensity. As it is well known, the disease is characterized by a continuous tremor affecting mainly one or both hands when the latter are at rest, and by a fixed attitude of the body, mask-like expression of the face.

Amnesia.—This term is applied in practice to an acquired diminution or loss of memory. There are two classes of amnesias: functional and organic. The characteristic feature of organic amnesia consists of its permanency and progressive evolution. Here, by virtue of an organic alteration of nervous elements, the memory for recent events is first affected, as new impressions can no more be associated and preserved. The gradually old intellectual acquisitions become effaced.

Functional amnesia is due to the profound emotional shock caused by trauma, viz. psychic trauma. The sudden commotion of the nervous system thus created disturbs the mechanism of association of ideas with regard to time and place and produces amnesia. Functional amnesia presents by far more variations than organic. It may concern only a certain group of ideas; it may be general, in which all past events are lost; it may be partial, as in cases of double personality, a striking example of which was reported by me in the *American Journal of the Medical Sciences*, 1906; it may be localized, when it concerns only a certain principal fact of life. Sometimes, in addition to the latter, a certain period of time immediately preceding it is forgotten. We then speak of retrograde amnesia. When the period of time following the principal event is forgotton, there is an anterograde amnesia. These two forms may be combined. Within the last three years I have seen two cases of retro-anterograde amnesia which followed a fall from a trolley car in which the patients struck their heads against the ground. In one of them there was loss of consciousness; the patients were confused for only ten minutes. A careful examination revealed in both cases no symptoms of an organic lesion. Subsequent events showed the diagnosis in both cases to be correct, as the patients made an uneventful recovery.

Medicolegal Considerations.—In the presence of an individual who, having sustained a trauma, complains of disturbances of a nervous or psychic order, the following medical problems must be solved: (1) Is the alleged disorder genuine or simulated, and what is its nature? (2) Should the symptoms observed be attributed to the traumatism? (3) If the latter is correct, what is the degree of incapacity for work, what are the prognosis, duration, and termination of the malady?

1. The determination of simulation is as a rule not difficult. When an affection is characterized by objective signs, they will be easily recognized. It is absolutely impossible for anyone to simulate an anæsthetic pharynx, and anæsthetic conjunctiva, a contraction of the visual field, a genuine hemianæsthesia, a genuine plus reflex. These stigmata taken together constitute the typical picture of hysteria. This malady is a well-defined morbid entity, which, except in rare cases, cannot be confounded with any other nervous affection. It is true that an hysterical paroxysm with its screaming, laughing, and various motor and psychic phenomena, can be to a certain extent simulated, but an experienced physician, familiar with the disease, will have no difficulty in recognizing it. The psychic symptoms of hysteria are not ordinary symptoms; they present a special physiognomy, the main points of which can never be guessed by the simulator. It is the entire picture of the attack that should be taken into consideration, not individual elements.

To illustrate to what errors a non-familiarity with the disease may lead, the following is a case in point. An attorney requested me to examine one of his clients, who claimed to have developed a hemiplegia after a broken trolley wire struck him. The physician in charge pronounced it an apoplectic stroke (there was a partial loss of consciousness) caused by the electrical current of the wire. The patient presented a limping of his left leg and some inability to use the left arm. The condition persisted almost two months. Examination showed a state of affairs totally different from typical hemiplegia which is due to a brain lesion. The knee-jerk was not exaggerated, and the abnormal reflexes (toe phenomenon, ankle-clonus) usually found in such cases were absent. The entire left side was also wholly anæsthetic. It was unquestionably a case of hysteria. The case was settled out of court, and the patient made a complete recovery shortly after he got his money. In fact, I cured him with a few séances of a strong suggestive static breeze.

A woman was in a slight railroad accident. She was only thrown on the floor of the car and there was no surgical injury. When picked up she could not hold herself in an erect position nor could she walk. The legs gave way under her. The condition was pronounced grave and a diagnosis

of complete paraplegia due to a hemorrhage in the cord was made. I saw the patient since then on several occasions. There was no paralysis, as she could move the limbs easily while in bed, but could not use them when standing or walking. The reflexes were normal, sensations normal, and the sphincters intact. It was a case of hysterical astasia-abasia, from which she fully recovered after a course of rest treatment with good feeding.

It is highly important to always bear in mind the possibility of suggestion during an examination. Suggestion is also one of the causes of hysteria. The examination therefore must be conducted very carefully. Sensations should be investigated by having the patient blindfolded. No question should be asked of her while the test is being made with a pin over various areas of the skin. There is no doubt that she will make some defense movement if the prick causes pain. Or else the patient may be asked to say "yes" the moment she feels pain or the touch of an object. No mention should be made to the patient of possibilities of loss of power in one or more limbs or segment of a limb, of loss of memory, of a grave outlook, etc. Various disturbances of function in the motor, sensory, vaso-motor, vegetative and psychic spheres may be suggested to a hysterical patient with great facility.

Far more difficulty is encountered in the medicolegal consideration of **neurasthenia.** This malady is characterized almost exclusively by subjective manifestations. When a patient, having met with an accident, complains of fatigue, backache, headache, insomnia and irritability, there are no means of verifying the presence or absence of each of these symptoms.

Nevertheless, when the disease is pronounced, the general aspect of the individual will aid in forming an opinion as to the veracity of his complaints. There is usually loss in weight, loss in strength, pallor of the face, depression, cold and clammy skin, tremor. When the case is mild, the latter symptoms are absent, and then one has to rely upon the first series of subjective disturbances. I have seen time and again that, in spite of an enormous shock following for example a collision or a fall from a height, only a few subjective symptoms would be complained of. In such cases the expert's opinion before the jury and court should be expressed thus: "If the subjective symptoms the plaintiff claims to suffer from are correct, he is neurasthenic; but I have no means of verifying them."

In such a manner justice is done to both sides, defendant and plaintiff. The expert's rôle, I believe, is to present medical facts and explain their meaning to a lay jury. The decision as to the relation of these symptoms to the accident is left entirely to the impartial judgment of the twelve men.

In the majority of traumatic cases hysteria and neurasthenia are combined. This facilitates considerably the problem, as objective signs are almost always present, and if there is a certain hesitancy in the mind of the physician in accepting the subjective symptoms, due credit should be given to the objective phenomena of the victim of the accident.

Chorea and **Paralysis agitans** developed after a trauma present no difficulty in being recognized. Simulation in such cases is an impossibility and the objective symptoms are too evident to be contested.

Considerable difficulty will be encountered when **amnesia** is the only symptom produced by the accident. If the loss of memory is associated with hysteria, the symptoms of the latter will render sufficient aid, but when hysterical stigmata are absent, the difficulty becomes very great in deciding the question whether the amnesia is genuine or not. In such cases the examination should be repeated, the individual must be tested with great perseverance and patience, he must be questioned as closely as possible and for a prolonged period of time. Great skill and tact are necessary in investigations of this sort, and in some cases one may arrive at a positive opinion.

2. The second medicolegal problem is, as I said above, to determine whether the maladies discovered in an individual are attributable directly to the accident. While a shock, physical or mental, is a frequent cause of functional nervous diseases, one cannot nevertheless be affirmative in a given case unless the state of health prior to the accident is known to the examiner. Hysteria, neurasthenia, chorea, or paralysis agitans may have existed before the plaintiff sustained the shock. It should, however, not be forgotten that if any of those affections existed before, the newly sustained shock will aggravate it. A person, for example, is supposed to be in the process of recovery from a neurosis. Should he at that time sustain an injury, the primary disease is likely to return and present itself in a more pronounced form than in its first attack.

Individual predisposition should also be taken into consideration in giving an account before court and jury. Neuropathic individuals are more apt to suffer from any of these neuroses than normal individuals. They are predisposed, and the least shock, a comparatively slight trauma is sufficient to disturb the workings of their nervous system with the greatest facility. Individuals whose vitality, and therefore resisting power, has been lowered following a protracted infectious disease, lead intoxication, alcoholism, syphilis, are inclined to respond with unusual promptness to the effects of a shock. The neuroses will find a fertile soil for their development. It is consequently important to surround one's self with this precaution in giving an estimate of the degree of the individual's

suffering. Not only the neuropathic tendency or the existence of a previous serious disease of the victim of the accident must be known to the physician, but also the knowledge of the previous conduct and habits of the injured individual is absolutely necessary. Sexual or alcoholic excesses, sleepless nights, irregular mode of living in every respect—are all predisposing elements for neuroses. The question of just compensation consequently can be decided after a complete knowledge of all factors concerned in any given case.

In view of the fact that trauma may actually incapacitate an individual for a long time, the possibility of **malingering** should never be overlooked. In cases in which the symptoms are chiefly subjective there may sometimes be some difficulty in differentiating true neurasthenia from malingering. Here the knowledge of the victim's previous habits and conduct is essential. However a malingerer usually exaggerates especially objective manifestations, such as paralysis or hyperæsthesias. He will often speak of loss of consciousness during the accident and at the same time give a correct account of all happenings. Such an individual should be thoroughly scrutinized and with a skillful manner of questioning and of eliciting the objective symptoms there will be no great difficulty in the majority of cases to arrive at a proper diagnosis.

3. The last proposition of our problem is to determine the degree of incapacity caused by the accident, the consequences, duration, and termination of the latter.

Hysteria and neurasthenia are not synonymous of simple nervousness, as some pretend. They are well-defined diseases of the nervous system. They do incapacitate for mental or physical work, but only to a certain extent. A neurasthenic with his chronic physical and mental fatigue is certainly not able to accomplish much. The disease presents variations in its intensity. If the symptoms are marked, the patient must go to bed, as rest is the most essential element of the treatment. Then there is no question of work. When the case is mild, and the fatigue is not particularly marked, and the backache with the headache only occasionally disturb the patient, a certain amount of work can be done by the patient, and with the proper regulation of the patient's mode of living he can be made to feel quite comfortable.

Hysterical patients are not very much disturbed in their daily life when the psychic symptoms are not present. One may bear the anæsthesias, hyperæsthesias, and tremor, also contraction of the visual field without being particularly annoyed. But, as a rule, there are psychic phenomena, viz. great emotionality, irritability, crying spells, restlessness, impressionableness, etc. Such patients are unfit for work. Their mental

concentration and application are of a very short duration. Whatever they commence, they are unable to finish. Their association of ideas is incomplete because of instability and want of depth of mental processes. For this very reason good work cannot be expected from those in whom psychic disturbances are marked. Even in those cases which are apparently free from mental symptoms, the least emotion or undue exertion will bring forward the above psychic manifestations so characteristic of hysteria, because the nature of the disease predisposes them to an unusual responsiveness. The degree of incapacity will depend upon whether the psychic symptoms are present or not, also upon the intensity of the latter.

Chorea and paralysis agitans incapacitate for physical work considerably, but only partially for mental work.

When amnesia occurs, a disability will ensue if it is of a generalized character. If it concerns only a certain past period of life (retrograde or anterograde form), there is no disability whatever. The individual is perfectly able to earn a living, as the memory is good for all events except that one.

A proper discrimination as to degree of incapacity in all the neuroses is always possible when the above elements are taken into consideration.

Not infrequently we are asked about the future of the victims of accidents. My answer is that while the neuroses are curable affections they nevertheless may last an indefinite time. The results depend upon many circumstances, viz. treatment, surroundings of the patient, previous general health, ability or inability to carry out certain instructions, outside influences, and finally the individual make-up. In exceptional cases the disease may last for years in spite of treatment. It should not be forgotten that recurrences are possible, and in fact are not infrequent. In the majority of cases the patients make a good recovery. The latter is possible in hysteria, neurasthenia, chorea, and amnesia, but not in paralysis agitans.

Psychoses.—Mental disorders present a very important chapter from a medicolegal standpoint. A shock caused by an accident is likely to produce mental disturbances, but between the latter and distinct, well-defined, mental affections there is a great difference.

Hysteria is, properly speaking, a psychic disturbance, and its mental phenomena described above belong by right to this chapter. They have been sufficiently emphasized on the preceding pages. **Amnesia** is another psychic phenomenon. It is also familar to us. When after a trauma **concussion** of the brain is suspected, the period of unconsciouness may be followed by some **mental symptoms,** viz. dullness and sometimes con-

fusion, inability to recall the circumstances of the accident (retrograde, anterograde or retroanterograde amnesia), inability to exercise the power of attention; disturbance of judgment, marked indecision in undertakings. All these symptoms are aggravated by insomnia which is usually present in such cases. These patients are ordinarily conscious of their mental deficiency and we observe that anxiety and worry over their condition and their future are added to the above disorder.

When the immediate symptoms of brain injury are severe, there is usually a cerebral hemorrhage or fracture of the skull. A cerebral contusion, followed perhaps by minute disseminated hemorrhages, is apt to be followed by mental symptoms, such as vertigo, delirium, confusion, loss of memory, and coma. In such cases the patient either dies or gradually recovers his mental faculties. **Korsakoff's psychosis** may be the result of a severe injury. We then have a marked confusion, illusions of identity, disorientation of space and time, loss of memory for recent events and fabrication. Alcoholism is usually the underlying cause (see for details page 347).

In infancy and childhood the condition is somewhat different. When a child in the process of development undergoes the effect of a severe cerebral injury, the result may be very serious. If **epilepsy** follows, there is no doubt that the mental growth will be interfered with. Faulty cerebral development may follow a grave injury to the head, irrespective of epilepsy. The prognosis, therefore, is guarded when arrest of intellectual development is observed shortly after a cranial injury.

The relation of **paresis** to traumata is a question of great importance. There are some alienists of note who believe that an injury is apt to develop paresis, others find no relation between the two. Paresis is an incurable disease; it is one of the most serious of mental affections. If its symptoms are noticed first after an accident, one is naturally apt to attribute them to the latter. At the Congress of the French Neurologists and Alienists in 1906 this question was the subject of a special report, so important it was considered. It was discussed at length by the most brilliant minds. It is frequently brought forward as of a special import, as courts and lawyers are waiting for our decision.

In the light of our present knowledge concerning especially the complement fixation test it may be safely said that paresis is not and cannot be caused by an accident. It is a mental disease of a slow, but progressive evolution, and characterized pathologically by a gradual degeneration of cerebral tissue. If its symptoms become sometimes conspicuous after a trauma, is it reasonable to suppose that a cerebral degeneration sets in immediately and at once produces the symptoms the development of

which require a long period of morbid changes in coritcal cells? In my judgment the disease existed before the accident, and the latter served only as an exciting cause for its more rapid development. Medical records are abundant with examples of this nature in other affections.

The mental manifestations of paresis, in its expansive or depressive forms, the physical signs, such as irregular and unequal pupils, the disturbance of the pupillary reflexes, characteristic speech, tremor of the tongue and hands, changes in the reflexes (loss or increase), apoplectiform or epileptiform seizures—it is not conceivable that these symptoms will explode shortly after an accident without being in existence prior to the accident. We are now in possession of bio-chemical means to determine the nature of paresis, so that in doubtful cases they render the most valuable aid in diagnosis. Positive Wassermann reaction of blood serum and cerebro-spinal fluid, increase of albumen in the latter, finally lymphocytosis—are all pathognomonic of paresis. The following peculiarities concerning these reactions are worth mentioning. (1) If the blood serum does not give a positive reaction, the cerebro-spinal fluid will be negative. If the cerebro-spinal fluid gives a positive result, the blood serum does the same. (2) If the serum gives a positive result, but the cerebro-spinal fluid a negative, the disease is not paresis, but cerebro-spinal syphilis. (3) The examination of the cerebro-spinal fluid is more important in paresis than that of blood serum. (4) Mercury, iodides, salvarsan may render the Wassermann reaction negative for a certain time .

If after a trauma these reactions are found to be present, there can be no doubt that paresis existed before the trauma. The latter *per se* cannot create any of the four reactions mentioned.

EPILEPSY IN RELATION TO TRAUMA

It is a well-known fact that trauma may cause epilepsy. A fracture of the skull may occur without any material evidence on the scalp. In such cases the inner table alone may be injured and a fragment may press directly on the cortical layer of the motor area. In other cases there may be some cicatricial tissue with thickening of the meninges producing an irritation of the motor area. In still other cases no appreciable lesion of the motor cortex had been found and still epilepsy developed after a trauma (Raymond). According to Kocher and Pitres the cause of epilepsy in such cases is the increased pressure of the cerebro-spinal fluid. In every case of traumatic epilepsy one must hear in mind the possibility of preexisting epilepsy or of the individual being predisposed

to it through alcoholism or syphilis. In such cases the trauma plays the rôle of a contributory factor. The following case of epilepsy is of interest fron a medicolegal standpoint.

The man I was called upon to examine fell off a ladder while working at a building. He struck his head and became unconscious. In the evening of the same day he had an epileptiform seizure with generalized convulsive movements. He was taken away from work, kept at home, and treated for epilepsy. Since then his mental faculties became obtunded. His memory began to fail. He was unable to give an account of himself. He would make grave mistakes. Would ask for a fork when he wanted a knife; would forget his wife's name. He would try to get in his neighbor's house instead of in his own. He also had outbreaks of extreme furor, in which he would use profane language, and even strike, so that the lives of his relatives became endangered. After the explosion of this passion subsided, he had no recollection of what occurred.

When I saw him months later he presented marked tremor of lips, increased reflexes, pronounced confusion, and a vague expression of the eyes with dilated pupils. He could not give me an explanation of those outbreaks of passion, of which he had no recollection. The patient had evidently been suffering from epileptic dementia. The attacks he presented were psychic in form, but of epileptic nature.

The question arose in court whether the trauma was the direct cause of his mental condition. A very careful search into the man's previous life, with data obtained from his wife, who was separated from him before the accident, also from his relatives and friends, revealed the fact that while the man never had a seizure prior to the trauma, and was mentally clear as he worked in the same place for seven years, he nevertheless had a syphilitic infection and used alcohol to an unusual excess.

My opinion, based upon all the facts of the case, was formulated as follows: "The trauma was an exciting cause to the epilepsy with its dementia, but the patient was predisposed to the disease. Any other cause would have produced the same condition. It is also possible that the mental affection would have developed without any apparent cause, as chronic alcoholism and syphilis are sufficient etiological factors in epilepsy."

An accident may be the exciting cause of a **delirium** or a **delirium tremens** in an individual profoundly intoxicated with alcohol. Similarly I have seen a confusional state developed in individuals with a previous alcoholic history. A thorough investigation of the personal antecedents is absolutely indispensable in cases of this category.

The previous study of trauma in relation to nervous and mental disorders leads to the following conclusion. In cases of railway or other injuries caused by neglect of those who have in charge the management of transportation cars, it is no more than just that the injured person should be compensated for disability. On the other hand simulation or exaggeration of incapacity should be condemned. The physician is indispensable to the law. In the name of justice he must be invariably reserved in his statements. His opinion must be formed after a thorough study of each individual case. He must not forget that, while some severe traumatisms may produce mild symptoms, some insignificant traumata may cause marked disturbances of the nervous system. The degree of the disability and the prognosis of the affection vary in each individual case. The recognition of the affection, the recognition of the influence of the accident upon its manifestations, finally the discrimination of a genuine malady from a simulated one—all these elements can be acquired only when the physician is properly prepared.

CHAPTER XXVII

DISEASES OF THE SYMPATHETIC NERVOUS SYSTEM

TROPHONEUROSES. ANGIONEUROSES

Exophthalmic Goiter

(Grave's or Basedow's Disease)

THIS affection is characterized by four cardinal symptoms: **enlargement of the thyroid gland, exophthalmos, tachycardia** and **tremor.** In the majority of cases they are all present.

Symptoms.—In typical cases the disease develops slowly. In exceptional cases all or almost all the characteristic symptoms appear suddenly or rapidly.

1. The most constant and earliest symptom is **tachycardia.** The least muscular effort or emotion brings on an attack of palpitation which may be so pronounced that the heart beat can be perceived at a distance. Arhythmia with signs of asystoly (cyanosis, œdema, vertigo) is observed sometimes. Precordial pain is frequently present. The carotid arteries are full and beat violently. The pulse is about 120 and more per minute. Examination of the heart shows nothing abnormal in most cases, although in advanced stages it may become dilated and a mitral insufficiency develops.

2. Gradually the neck begins to get large and a **goiter** develops. The enlargement may affect the entire thyroid gland or only one lobe. The goiter is soft and vascular. A systolic bruit is often heard on auscultation. The goiter is not painful, but when it increases rapidly, it produces symptoms of compression and threatening suffocation.

3. **Exophthalmos** is very frequent, but not constant. It is usually bilateral and occasionally unilateral. It may be more marked on one side than on the other. The protrusion of the eye globe gives the face an expression of fright and anger. Closing of the eyelids is impossible. The exposure of the conjunctiva and cornea leads to inflammation and ulceration. Lachrymation is frequent. The palpebral fissure is sometimes very wide because of a contraction of the levator palpebræ and the patient is then unable to wink. This is **Stelwag's** sign. There are other signs that accompany exophthalmos. **Von Graefe's** sign consists of an

inability of the eyelid to follow the movements of the eyeglobe; when the eye is in the act of moving upward or downward, there is a delay in the movement of the eyelid. **Möbius' sign** consists of difficulty of convergence.

External ophthalmoplegia has been observed. The visual acuity is diminished. Hyperæmia of the retinal blood vessels is sometimes noticed.

FIG. 161.—EXOPHTHALMIC GOITER.

The eye-grounds are usually normal. Amblyopia, diplopia, are very rare occurrences.

4. **Tremor** is very frequent and it is particularly significant in the incomplete forms. It is a fine, vibratory tremor. The oscillations are rapid (eight to ten per second). It is localized or generalized. It affects more frequently the hands, but also the head, trunk and the lower extremities. It is present during rest, but more marked on voluntary acts and emotion.

Besides the above four typical signs there are others which may occur now and then in the course of Grave's disease.

Motor Symptoms.—They are: paresis of the lower extremities or of the facial muscles, hemiplegia, monoplegia, but they are all transitory. Cramps, contractures, tetany, epilepsy, occur occasionally. The reflexes are usually unaltered, but they may be increased or decreased.

Sensory Symptoms.—Pain of a **neuralgic** character, especially in the eyeglobes, face, neck, also in the precordial region, in the arms, is not infrequent.

Vasomotor and Trophic Disturbances.—Flushes of heat in the head, profuse perspiration, slight elevation of temperature several times during the day, are quite commonly observed.

The skin may be the seat of various eruptions. Urticaria, œdema, pigmentation, vitiligo, occur sometimes. Association of Grave's disease with scleroderma has been reported. Falling out of the hair is not infrequent. Increase of galvanic reaction in the skin and various tissues is frequently observed. Nutritive changes are present. There is usually emaciation and general weakness.

Urinary Symptoms.—Polyuria, albuminuria, glycosuria, occur. The first is quite frequent.

Digestive Disturbances.—Anorexia or else polydipsia, vomiting, diarrhœa are observed. Excessive salivation and jaundice are rare occurrences.

Respiratory Disturbances.—Dyspnœa, dry cough, inability to execute forced inspiration (Bryson's sign) are observed.

Disturbed Genital Function.—Atrophy of the organs and of the mammary glands, impotence, diminution of sexual desire, amenorrhœa, occur.

Psychic Disturbances.—Restlessness, irritability, instability, changes from gay and happy mood to depression, sadness, and anger are frequently present. Insomnia is the rule. Hypochondriacal ideas frequently develop. In some cases delirium and confusion with hallucinations occur.

Forms.—The above clinical picture is that of **typical** cases of exophthalmic goiter. There are cases in which not all of the four chief symptoms are present (**incomplete forms**). Grave's disease **without goiter** or else **without exophthalmos** is not very rare. Other forms are marked by a slow development of symptoms (**chronic**). Others (**acute**) are conspicuous by rapidity of development.

Association of Grave's disease with hysteria is frequent. Sometimes it is seen with epilepsy and occasionally with tetany, chorea, tabes, syringomyelia. I reported a case of exophthalmic goiter associated with

paralysis agitans (*New York Med. Jour.*, December 31, 1904). Finally it may be also met with in diabetes, myxœdema and insanity.

Course, Duration, Prognosis.—The symptom-group is rarely complete, but the phenomenon which is never wanting and which appears at the onset of the malady is the cardiac disturbance. The acute and chronic varieties have been mentioned. The course is rarely regular: it varies from individual to individual. Amelioration and aggravation of the symptoms occur frequently.

In the majority of cases the duration is protracted. It may last many years or indefinitely. Recoveries rarely occur. The **prognosis** depends upon the severity of the symptoms. Anorexia, diarrhœa, albuminuria, produce cachexia and hasten death. Sometimes death comes on very rapidly. Compression of the trachea by the goiter, asystoly, may be the cause of rapid death. In some cases an intercurrent disease leads promptly to a fatal termination. Pulmonary tuberculosis is not rare. In the most favorable cases some traces of exophthalmos, of goiter, remain.

Diagnosis.—The typical form will be recognized from its cardinal signs. Difficulties will be encountered in the abnormal or incomplete forms. In such cases the constant presence of tachycardia and very frequent presence of tremor will aid in tracing the affection. Hysteria associated with chlorosis may simulate Grave's disease, but the cardiac disturbance in the chlorosis is not as continuous as in exophthalmic goiter.

Simple goiter with tachycardia may also embarrass the diagnosis. It is the general picture of the disease that should always be taken into consideration while making a diagnosis.

Etiology.—A neuropathic heredity can be traced in the majority of cases. Acute **infectious diseases** (typhoid fever, inflammatory rheumatism, grippe, scarlet fever, pertussis) may be followed by Grave's disease. Syphilis, lead intoxication, tuberculosis, ordinary goiter, are all **predisposing** factors.

Among the **exciting** causes may be mentioned violent emotions, traumata, excesses (sexual and others), pregnancy.

Pathogenesis.—The pathological investigations show changes in the thyroid gland, thymus, cervical sympathetic nerve and medulla. The **thyroid** gland may present many morbid varieties from a simple congestion to the most pronounced lesions. Generally there is hypertrophy of the entire gland, of one lobe or of a portion of a lobe. The vesicles are increased in size, the epithelium is hypertrophied; the intravesicular colloidal substance is usually diminished in quantity; in the parenchyma of the gland there are **lymphoid masses** which are considered character-

istic of exophthalmic goiter. The connective tissue of the gland is abnormally developed. The blood vessels are dilated and their walls are thickened.

The same condition has been found in the **thymus.**

The cervical **sympathetic** ganglia and nerve have also been found altered (proliferation of connective tissue, multiplication of blood vessels, atrophy of cells).

In the **medulla** dilatation of blood vessels and hemorrhages, atrophy of the restiform bodies have been reported by competent observers.

In view of such a great variety of pathological changes the nature of Grave's disease remains as yet obscure.

The most favorable view held at present is that the thyroid gland is the only cause of the affection. The theory is as follows. The function of the thyroid gland consists of extracting from the system a toxic product and neutralizing it before it is thrown into the general circulation. The neutralization is performed by a special product secreted by the gland. In Grave's disease there is an excessive secretion of the gland (hyperthyroidization). The toxic product thrown into the circulation irritates the cervical sympathetic nerve, which then produces the symptoms of the eyes, of the heart and the goiter. This theory cannot satisfactorily explain why, if a toxin is at fault, it excites only the cervical sympathetic nerve and not also the other sympathetic nerves. It also fails to explain cases with enormous exophthalmos with insignificant thyroid enlargement, cases with enormous goiters and insignificant exophthalmos, cases of unilateral Grave's disease.

On the other hand there is a number of cases on record showing that many nervous diseases become complicated with symptoms of Grave's disease when in the later stages bulbar symptoms make their appearance. Such are the observations on tabes, amyotrophic lateral sclerosis, polioencephalitis, pachymeningitis cervicalis, syringomyelia. Experimental physiology (Filehne, Bienfait, Durdufi), post-mortem examinations collected by H. Klein (*Deutsche Zeitschrift f. Neurologie*, 1904) show changes in the medulla and pons, especially hemorrhages.

In a case observed by me (*N. Y. Med. Jour.*, 1905) a woman suddenly developed a paralysis of the third, fourth and sixth nerves unequally distributed on both sides. A few days later she noticed a gradually coming-on prominence of both eyes. A week later a goiter with tachycardia and tremor began to develop. There was also von Graefe's sign (Fig. 161).

All these considerations tend to prove that the phenomena of Grave's disease are due to a bulbar disturbance: the vaso-dilators of the head

and heart which have their deep origin in the medulla and situated in the cervical sympathetic nerve, undergoing irritation, send a constant afflux of blood to the thyroid and to the retrobulbar vessels; the filaments going to the heart give rise to tachycardia.

Treatment.—The first indication is to place the patient in a condition of keeping his nervous system free from emotions or shocks. Rest, proper hygiene, regular and quiet mode of living and removal from the usual surroundings—better in the country—are all beneficial means. He should avoid exertion, climbing stairs, mountains. The tachycardia which is always present, and which is distressing, will thus be ameliorated.

All stimulants, including tea and coffee, the use of tobacco, must be avoided. Sexual intercourse is forbidden. Marriage is also contra-indicated.

The diet should be nutritious, but not abundant. Milk is an ideal food in such cases. Constipation is to be avoided, but powerful purgatives should not be administered.

Internally the following remedies can be tried, but not much reliance can be placed on any of them: Bromides, salicylates, digitalis, strophanthus, iron, quinine, belladonna, iodides and injections of iodine into the thyroid gland.

Lancereaux reported very brilliant results from the use of quinine sulphate with and without ergot. Forchheimer obtained very good results from neutral salt of quinine hydrobromate in gr. v doses three or four times daily. The drug should be administered for a very long time, viz. months. In case of persistent tinnitus, which the drug is apt to produce, it is advisable to discontinue it for a certain time and then resume it.

Opotherapy sometimes gives satisfactory results. Thyroid extract, thyroidin, iodothyrin, antithyroidin, of Moebius, extract of parathyroid glands, of thymus or of suprarenal capsules are administered internally. Milk of horses whose thyroids had been extirpated (Lanz), thyroidectin, viz. desiccated blood of thyroidectomized sheep, a serum prepared by Rogers and Beebe by the use of nucleoproteid and thyroglobulin from normal and pathological glands, finally injection of serum of thyroidectomized dogs (Ballet and Enriquez) have also been advised. Rogers and Beebe's investigations with their serum concerned a series of 480 patients in 1909. The results were as follows: 15 per cent. totally cured; 10 per cent. cured from their subjective disturbances, but exophthalmos or goiter remained unaltered; 50 per cent. improved; 17 per cent. derived no benefit; 8 per cent. died from the progress of the disease. The results are better in acute than in chronic cases.

It is impossible as yet to form a definite opinion as to the therapeutic value of these specific preparations.

Electricity, especially galvanism with the negative pole on the goiter, stabile galvanization of the sympathetic may sometimes render some service. X-ray and radium therapy are reported to be beneficial. While radiotherapy may have a favorable effect on the general health and the nervous manifestations, it has no effect on the goiter, exophthalmos and very slight effect on the tachycardia. On the other hand radiotherapy may produce perithyroid adhesions which may be a disturbing element in ulterior operative procedures. Moreover, X-rays may produce a thyroid insufficiency with myxœdema or else may aggravate the preëxisting manifestations, as some observations tend to show.

While the medical treatment rarely gives permanent results, **surgical intervention** meets with somewhat greater success. Operations should not be performed in every case. In grave or advanced cases secondary organic changes develop in the thyroid, myocardium, periocular tissues, so that complete disappearance of symptoms cannot be expected. Patients who reach a state of cachexia should not be operated upon.

Surgical intervention is indicated in cases which continue to progress in spite of the best medical treatment; in cases in which there are evidences of compression of the trachea, œsophagus and recurrent laryngeal nerve; in cases with a very acute onset; in cases with grave nervous and mental symptoms; finally in cases with a marked lymphocytosis according to Kocher.

In the majority of cases exophthalmic goiter is rebellious to medical treatment. However, an attempt with internal medications should be made almost in every case. Horsley recommends about six weeks of such treatment and if the results are meager, surgical intervention should not be postponed. On the other hand the best results are obtained if the patients are operated on very early in the course of the disease.

Thyroidectomy and ligation of the thyroid arteries are very serious operations and exceptionally give favorable results. Complete recoveries have been reported. If under recoveries is understood a total disappearance of all subjective and objective symptoms, they are exceptional. In the majority of cases considerable amelioration is obtained. Thyroidectomy should always be **partial** in view of the great danger following removal of the parathyroid glands. In ligation of thyroid arteries there is danger of including the recurrent laryngeal nerve while tying the inferior thyroid artery. The danger is in producing laryngeal paralysis and inhalation pneumonia. Ligation of the superior thyroid artery is, according to Mayo, safe.

Removal of the cervical sympathetic ganglia with their cord gives, according to the statistics of Jonnesco, the most satisfactory results. He advises bilateral extirpation of all the three ganglia with the cords. The results obtained by him have been uniformly perfect. Chalier (*Lyon Chirurgical*, 1911) in an extensive study confirms Jonnesco's statement, viz. that operations on the sympathetic are in every respect superior to thyroidectomy.

All reflex irritation of the sympathetic from some remote affection, as fibroid uterine tumors, nasal polyps, etc., should be removed.

If the syndrome of Grave's disease occurs during the course of a spinal or other organic nervous disease, no operation should be performed.

FIG. 162.—MYXŒDEMATOUS IDIOCY. (*Bourneville.*)

MYXŒDEMA (CACHEXIA STRUMIPRIVA)

This disease was first described by Gull in 1873 and in 1877 by Ord, who gave the above name.

Symptoms.—There are **three** cardinal symptoms that characterize the affection, viz. (1) **swelling of the skin,** (2) **atrophy of the thyroid gland** and (3) **mental deficiency.**

1. The **swelling,** which is due to a mucous or mucoid infiltration of the skin, is first noticed on the face. The latter is large, round and its skin is of a yellowish tint (wax-like), dry and without hair. On palpation the skin feels hard, not depressible and thick.

The nose is enlarged, the eyelids are swollen and droop, the lips are thick, the forehead is wrinkled.

The facies is without expression and appears stupid.

In other portions of the body the skin is equally thick. The trunk is less affected than the extremities. The fingers and toes are large, the nails are thick, hard and brittle.

The hair of the body atrophies and falls out, perspiration ceases, the skin is dry and scales off.

In the subclavicular region and in the axilla pseudo-lipomatous masses are found.

The **mucous membranes** are equally swollen, pale and dry. The tongue is thickened and if the pharyngeal and laryngeal mucous mem-

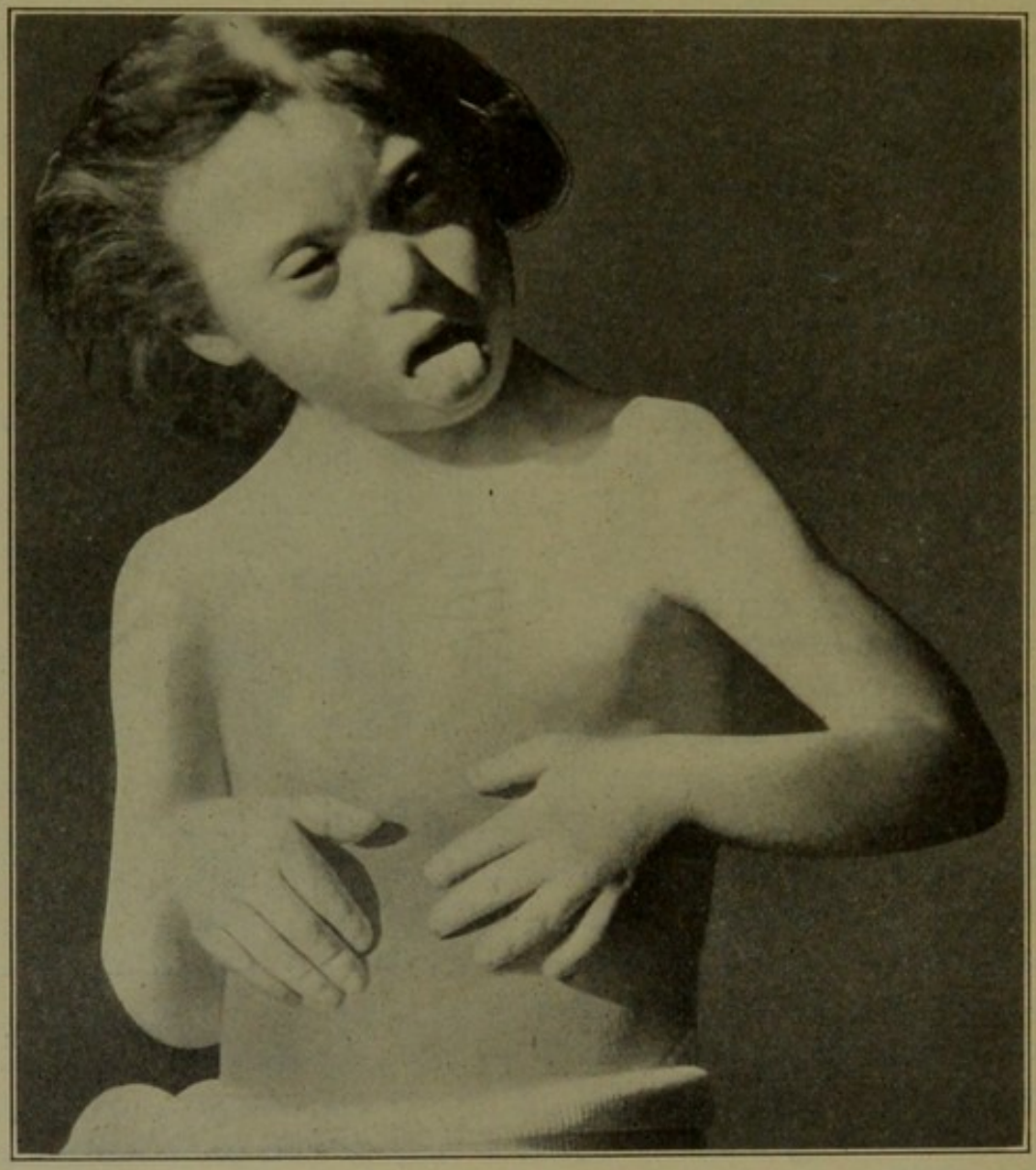


FIG. 163.—INFANTILE OR CONGENITAL MYXŒDEMA.

branes are swollen, dysphagia and changes of the voice are present The teeth also suffer in their nutrition and fall out.

2. The **thyroid gland** is usually atrophied. Very exceptionally it is hypertrophied.

3. The **mental** condition is marked by deficient intellect. The patient is somnolent, apathetic, his answers are slow, memory decidedly impaired. He is usually irritable. In some cases delirium and hallucinations develop.

Other Symptoms.—The heart-beat is weak, the pulse is small and irregular. Hemorrhages, and especially uterine, are frequent. The temperature is below normal. Constipation is the rule. Patients often complain of pain in the extremities and neck, of deafness, tinnitus aurium, vertigo and headache. The reflexes are not altered. There are no objective marked sensory disturbances except some diminution of sensations.

The patient is unable to do any work. He is slow in words, actions and thinking. The gait is hesitating. The torpitude is general.

Forms.—The clinical picture just given presents a fully developed type of myxœdema of adults. But there are also **incomplete forms** in which only some features of the typical form are present and in which thyroid medication proves to be beneficial.

Infantile or Congenital Myxœdema.—What characterizes this form is the **arrest** of physical and intellectual development.

Physically these patients are dwarfs. The head is narrow in front, the anterior fontanelle persists. The dentition is delayed. The mouth is open, the saliva dribbles continuously. They begin to walk and speak late. The genital organs are not developed. The usual signs of pubescence are absent. These patients eat abundantly and are much constipated. Intellectually they are **idiots.** When they grow up, they have the appearance as described in the first chapter, except that intellectually they show they have never developed. Such children are usually born from tubercular, alcoholic or syphilitic parents.

Infantile myxœdema may present various degrees. It depends upon the amount of thyroid gland preserved. **Brissaud's "infantilism"** belongs to this class. Here we find an arrest of physical development (small size, undeveloped genitalia, dry skin without hair, etc.), but there is some degree of intellectuality sufficient for certain occupations. These individuals are small adults. They constitute the so-called "partial myxœdema" cases.

Cases of infantilism described by Lorrain do not enter here. They are small individuals, but do not possess the attributes of infancy.

MYXŒDEMA STRUMIPRIVA (OPERATIVE MYXŒDEMA)

This form may follow extirpation of the thyroid gland or of a portion of it.

The symptoms begin to develop some months after the operation, although they may appear sooner. The initial symptoms are: a lassitude and weakness in the limbs. Gradually the skin becomes infiltrated

and the hair begins to fall out. At the same time the characteristic mental hebetude (see above) makes its appearance. The intensity of the symptoms varies from case to case and depends also upon the age at which the operation is performed. The younger the individual, the graver the symptoms are. A point of great importance is the fact that partial thyroidectomy is followed by far less grave symptoms than total extirpation of the gland. Gley and Vassale have called attention to the great relationship of the parathyroid glandules to physical and mental conditions. Preservation of these glandules is never to be forgotten in operations on the thyroid gland.

Sporadic Cretinism.—It presents, physically and mentally, the same infantile myxœdematous type as described above. The only difference is found in the thyroid, which is here hypertrophied instead of being atrophied. The hypertrophy coexists with insufficient function: instead of glandular tissue there is proliferation of sclerotic tissue. Consequently from the standpoint of function it is identical to an atrophic thyroid.

This type is particularly observed in certain localities of Switzerland, Austria, Italy and France. Climate and geological conditions, the water and the air, have probably to do with the disease.

Course, Duration, Prognosis.—In the typical form the course is slow and progressive. The physical and intellectual infirmities gradually increasing lead to a profound deterioration. The skin gets very hard, the functions of the viscera are more and more disturbed. The temperature goes down, the secretions become less and less. However, remissions occur, but these periods of amelioration are only temporary. Eventually the mental hebetude increases and a pronounced cachexia sets in. Death is the ultimate result. Death may occur from some intercurrent disease, among which pulmonary tuberculosis is the most frequent. The prognosis is therefore unfavorable. In the operative myxœdema, as mentioned above, the prognosis depends upon the integrity of the parathyroid glandules and upon the amount of thyroid gland removed and finally upon the age at which the operation is performed. Grave immediate symptoms may also occur after the operation, viz. **convulsive seizures** and **tetany.** The latter particularly may persist for months or cause death. These phenomena are due to the removal of the parathyroid glands.

In infantile myxœdema the individual cannot be considered diseased; his health is as a rule good. His condition does not lead to cachexia. He simply presents a morphological or functional anomaly which does not compromise his existence.

Diagnosis.—In the majority of cases the characteristic symptoms of

the skin, of the thyroid gland, of the intellectuality, of the attitude, are sufficiently typical for the recognition of myxœdema. Some difficulty may be encountered in the diagnosis of the **incomplete** forms. Infantile myxœdema will be recognized from the arrested development; there is no cachexia.

Etiology.—Heredity plays a certain rôle. Alcoholism, tuberculosis, diabetes and syphilis in the parents are considered as predisposing causes. Pregnancy, lactation, menopause, which are accompanied by congestion of the thyroid, perhaps have a predisposing influence.

Direct causes are: infectious diseases which may lead to an inflammation of the thyroid gland ending in its sclerosis and atrophy. Tumors of the gland may produce the same condition.

Acquired myxœdema may occur at any age, but more frequently in children than in adults. Females are more predisposed than males.

In infantile myxœdema the cause of congenital absence of the thyroid gland is unknown.

Pathogenesis.—Pathological investigations show that in congenital infantile myxœdema the thyroid gland is totally absent. In the adult form of myxœdema there is degeneration of the normal tissue of the thyroid gland, proliferation of connective tissue. The subcutaneous tissue is infiltrated with mucin. There is hyperplasia of adipose tissue.

The suppression of the function of the thyroid gland is the cause of the disease.

Normally the thyroid elaborates a special substance which, thrown into the circulation, destroys the usual toxic elements of the organism. When the thyroid is diseased, its antitoxic substance is either absent or perverted. The result is myxœdema. Internal administration of thyroid extract supplies the organism with the necessary elements and amelioration of symptoms follows. That the parathyroid glands participate in this useful function there can be no doubt (see above).

Treatment.—The foregoing remarks lead logically to the therapeutic indication. Thyroid extract administered internally is almost the only remedy for the condition. As it may produce tachycardia, cerebral irritation, headache, insomnia, dyspnœa and sometimes albuminuria, its administration should be watched. It is advisable therefore to commence with small doses—for an adult gr. j or ii t. i. d. and for a child a fraction of a grain. The initial dose can be gradually increased. General hygienic measures should not be neglected. A vegetable diet is preferable to any other.

Good results have been obtained with thyroid not only in the acquired form, but also in the congenital form.

ACROMEGALY

This disease was first described by Pierre Marie in 1885.

Symptoms.—The essential and characteristic signs of the disease are **enlargement** of the osseous and other supporting tissues, especially noticeable in the distal ends of the extremities (hands, feet) and the head.

The **hands** become first affected. They are thick and wide. The bones and the soft parts are involved. The skin is hard and free of œdema. The thenar and hypothenar eminences are very large. The fingers are enormous, but not deformed and their function is preserved. The fingers may be only wide (**transverse type**) but also long and wide (**longitudinal**

FIG. 164.—ACROMEGALY. (*Marie.*)

type). The size of the hand is out of proportion with the rest of the upper limb which is only slightly enlarged. The feet are equally enlarged. The toes and the soft tissues are thick and wide. Here again the contrast between the size of the feet and the legs is striking; the latter are only slightly involved. On the **head** the occipital protuberance is prominent. The cranial bones are irregularly thickened. It is particularly the **face** that shows hypertrophy. It is elongated, the forehead is low, but the orbital and zygomatic arches are very prominent, the eyelids are thickened. The nose is enormous. The lower jaw is very much hypertro-

phied, the chin is projected (**prognathism**) and large. The lips are heavy and the lower one is everted. The tongue is thick (macroglossia) and broad. The palate, tonsils, uvula and larynx are all hypertrophied. The vocal cords are thick. The mucous membranes are hypertrophied so that difficulty of deglutition may occur and the voice is deep. The skin of the face is dry and brownish. The hair of the head is thickened.

The **vertebral column** presents a cervico-dorsal kyphosis.

The **sternum** is thickened and projected; the clavicle and the ribs are large; the costal cartilages are ossified.

Besides the deformities there are other constant symptoms: **Headache** and **Amenorrhea.** They often appear at the onset of the disease.

Other Symptoms.—Not infrequently **ocular** disturbances occur, viz. scotomata, diplopia, strabismus, diminution of visual acuity; also **optic neuritis** and **optic atrophy.** Blindness and hemianopsia with Wernicke's pupillary reaction are occasionally observed. In a large number of cases simple amaurosis with pupillary immobility is observed.

The **viscera** sometimes participate in the hypertrophy. Cardiac hypertrophy is particularly troublesome. Palpitation, arhythmia and dyspnœa, also attacks of syncope are quite frequent.

The **abdomen** is large, the mammary glands and the uterus are atrophied. The male genitalia are enlarged, but impotence is present. In the female the clitoris may undergo hypertrophy.

Sterility is frequent.

Polydypsia, polyuria glycosuria are quite frequent.

Sensations are normal, but **pain** in the extremities and spine is frequent. It is increased by fatigue, pressure and exposure to cold. The pain may be of neuralgic or rheumatoid type.

The reflexes may be normal, increased or diminished.

There is a general sense of continuous fatigue, although the movements of the limbs are not involved.

The patient is as a rule apathetic and of slow mentality.

Focal epilepsy has been observed in exceptional cases.

Course, Duration, Prognosis.—The course is usually slow but progressive. The disease may follow an **acute** or **chronic** course. The latter is the most frequent. Amelioration and temporary arrest occur. It usually lasts twenty or thirty years. Cachexia and death are the habitual termination, unless the patient dies from some intercurrent disease. Men are more frequently affected than women.

Diagnosis.—The differentiation with **Myxœdema** is comparatively easy. In the latter disease the enlargement of the extremities is due to a mucoid infiltration of the skin. The round, puffy face of a myxœdema-

tous is essentially different from the oblong face and prognathism of an acromegalic.

In **Elephantiasis** only the skin and the cellular tissue are involved. It affects only one side and an entire limb.

Hypertrophic Pulmonary Osteo-arthropathy (Marie) is characterized by paw-like hands, the ends of the fingers are enlarged and the nails are thick and brittle. The lower jaw is not involved. The disease is usually associated with cardiac or pulmonary diseases.

In **Osteitis deformans** (Paget) there is a marked deformity of the legs, but the hands and feet are intact. The cranium is deformed, while in acromegaly the face is involved.

Etiology.—Heredity plays a small part. Diseases of the nervous system (chorea, hysteria, tabes), mental diseases, infectious diseases, alcoholism, syphilis, gout have all been considered as predisposing factors. As exciting causes can be mentioned trauma and violent emotions. The disease occurs mostly at the age of between twenty and thirty-five. Females are somewhat more frequently affected than males.

Pathogenesis.—Pathological investigations show that in a large majority of cases the **pituitary body** was found diseased (degeneration, tumor, hypertrophy). In a number of cases the thyroid gland, the thymus, suprarenals and pancreas were altered. Although in some observations the pituitary gland was found intact, the consensus of opinion at present is that a disturbance in function of this glandular body is the only cause of acromegaly (hyperpituitarism). There are clinical and experimental reasons to believe in physiological interrelation between all those glands.

The nutrition of the body and cartilaginous tissues is supposed to be controlled by the pituitary body. Therefore any disturbance in its normal function is followed by the manifestations characteristic of acromegaly.

Treatment.—The foregoing remarks lead to therapeutic indications. The pituitary body, thyroid and thymus glands have been administered in acromegaly. Some favorable results have been reported, particularly from the use of the pituitary gland. Iodides, mercury, iron, ergot and arsenic may be tried. Other manifestations of the disease are treated symptomatically. General hygienic measures should not be neglected. Operative procedures for the purpose of removing the hypophysis date from 1907. Schlosser was the first to extirpate a tumor in a man. Surgeons have since attempted operations through different routes. The latter are: (1) nasal route combined with resection of the upper maxilla and orbit; (2) buccal route with resection of hard palate; (3) transverse supra-

hyoid pharyngotomy; (4) temporal intra-cranial route; (5) trans-frontal method (Frazier), viz. through anterior cranial fossa.

GIGANTISM

A condition allied to acromegaly is gigantism. Under this name should be understood a condition which is characterized not only by a size of the body above the normal, but also by physical and mental anomalies.

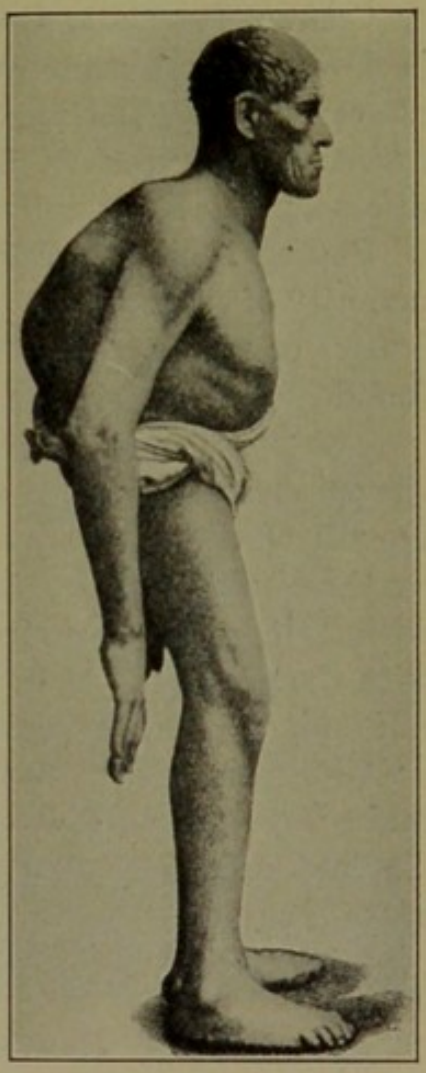


FIG. 165.—ACROMEGALIC GIANT. (*Brissaud and Meige.*)

Symptoms.—The most important feature is the **excessive size** of the body. The height may reach nine or ten feet. The unusual growth of the body commences at puberty and in exceptional cases in childhood and continues above the average age.

In spite of their enormous size the giants complain of **muscular weakness.**

As a sign of abnormality should be also mentioned **atrophy of genital organs,** the consequence of which are impotence, sterility, amenorrhea.

The **mentality** is also below normal. Such patients are apathetic, childish, without initiative and with a poor memory.

As additional manifestations they infrequently present continuous **headache, visual** disturbances, polyuria and especially **glycosuria.**

Gigantism and acromegaly is one affection. If the progressive increase in size makes its appearance in childhood or youth, the result will be gigantism. If the size continues to increase beyond the age at which growth ceases normally, the result will be associated with acromegaly (see this chapter). The individual is therefore an **acromegalic giant.** Acromegaly is consequently the gigantism of the period of full development. Gigantism is acromegaly of the developmental period. The association of gigantism and acromegaly may occur not only in two successive periods, which is the rule, but also simultaneously long before the epiphyseal cartilages become ossified, at a period very near birth and even before birth. In the latter case the hypophyseal hypertrophy, which is very

probably the common cause of both gigantism and acromegaly, may sometimes be congenital. Gigantism may be associated with **infantilism.** In such individuals the attributes of an infant are present, as, for example, absence of ossification of the cartilages between the diaphysis and epiphysis. Moreover, there are also atrophy of the genitalia, undeveloped musculature, tender skin, absence of hair of the pubis and axilla. The mentality is that of a young child. An **infantile giant** may also become acromegalic.

Course, Duration, Prognosis.—In the majority of cases the course is slow, although acute cases have been reported which ended fatally at an early age. Ameliorations have been observed. The condition is essentially progressive. The patients usually die from some intercurrent disease, particularly pulmonary tuberculosis.

Pathogenesis and Etiology.—Pathological investigations show in the majority of cases a diseased condition of the pituitary body, either hypertrophy or tumor. In some cases hypertrophy of the thyroid gland was found.

The prevalent opinion is that gigantism is due to the persistence of the epiphyseal cartilages beyond the normal age and continuation of the growth of the skeleton. Ossification of cartilages is controlled mainly by the pituitary body, but also by the thyroid, testicle and other glands with an internal secretion. A morbid condition of the latter will interfere with the process of ossification. Speaking generally gigantism is by itself an indication of a degenerative make-up.

Gigantism may follow infectious diseases, intoxications. It affects males more frequently than females.

Treatment.—Pituitary or thyroid gland may be tried, although no genuine satisfactory results have been reported.

ACHONDROPLASIA

It is a congenital malformation characterized by a smallness of stature and due to a deficient ossification of the cartilages of the long bones.

The first good description was given by P. Marie in 1900, although the condition had been known long before.

Symptoms.—Soon after birth the unusually small size and short limbs are noticed. The growth is retarded and the individual reaches adult life, but is a dwarf.

The following three peculiarities characterize achondroplasia: **dwarfishness, micromyelia** and **macrocephalia.**

The trunk and head are of normal size, but the limbs and particularly the lower are unusually short (micromyelia). The contrast is striking and

typical. The proximal ends of the limbs (arms and thighs) are more affected than the distal ends. Normally when the individual is in a standing position, the medius reaches the lower third of the thigh. In achondroplasia the same finger hardly reaches the trochanter.

Marie called attention to a special position of the fingers. When an attempt is made to bring them together, only the first phalanges are approached, the last two are separated.

The head may be of normal size, but not infrequently is enlarged (macrocephalia). Sometimes it resembles a hydrocephalic head.

Other Symptoms.—The scapulæ are short. The pelvis is small. The musculature on the contrary is well developed. The genitalia are normal in size and function. The mentality in the majority of cases is normal. In Marie's cases, however, it was deficient.

Pathogenesis and Etiology.—Various views have been advanced as to the nature of achondroplasia.

Some believe in a hereditary degenerative state of the cartilages. According to others a toxic material influences in some manner the nervous system, which in its turn produces trophic disturbances of the cartilages. Perhaps the ductless glands are the source of the toxic material.

Treatment.—Not much reliance can be placed upon the administration of thyroid extract, which has been advised.

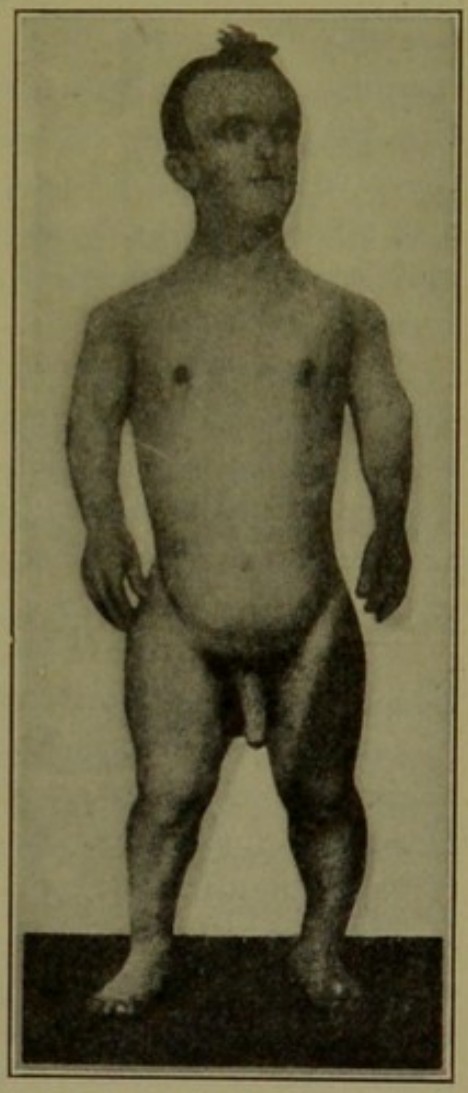


FIG. 166.—ACHONDROPLASIA. (*Apert.*)

ADIPOSIS DOLOROSA

It is characterized by deposits of fat in various portions of the body. These deposits are painful upon pressure.

The disease has been observed and described by clinicians under various names, but Dercum was the first to give this clinical entity the name of adiposis dolorosa.

Symptoms.—Four main symptoms characterize the disease, viz. **fat**

formation, pain, asthenia and **psychic symptoms.** The striking feature is the **nodes of fat** distributed all over the body. The deposition of fat may be not only nodular, but also circumscribed diffuse or generalized diffuse. The nodular is the most common form. The extremities are particularly involved. At first the bunches of fat are noticed in the lower and upper extremities; the trunk is affected later, but the face, hands and feet have been spared in all observed cases. When these nodes are compressed, the patient suffers considerable **pain.** In the nodular form the size of the nodules may be as small as a pea but more frequently they are of the size of an orange. They are usually mobile, but they may be adherent to the skin. In the circumscribed diffuse form the mass may involve a segment of a limb or an entire limb. The mass may be smooth or lobulated.

The generalized diffuse form usually begins on the abdomen and then spreads gradually to the trunk and extremities. The hands and feet which usually remain unaffected in the two other forms, here are not infrequently involved, but the fatty deposits are seen only on the palmar surface of the hand (thenar and hypothenar regions) and on the soles of the feet.

The skin over the involved area is usually white and soft. Pigmentation of the skin with atrophy and diminished sweating have been observed.

Other Symptoms.—The patient often complains of spontaneous dull aching, also of numbness, coldness or else burning. **Pain** is never absent. It may be spontaneous or elicited by pressure. In well-advanced cases it may be unusually severe. It does not follow the course of nerve trunks. Besides being continuous, the pain may present paroxysms of unusual severity. At each of such paroxysms the size of the fatty mass increases, but recedes at the disappearance of pain. There is usually a marked diminution of objective sensibility or complete anæsthesia in the affected areas. **Muscular weakness** is a frequent symptom. Sometimes it is so marked that the patient is compelled to remain in bed. Headache, hemorrhages in the nasal cavities occasionally occur. One of my patients presented retinal hemorrhages appearing without a strain, effort or any traceable cause. Contraction of the visual fields, scotomata, amaurosis have been observed. **Psychic symptoms** are quite frequent. Change of disposition, marked irritability are common. In some cases there is also mental exhaustion. Delusions of persecution, dementia have also been observed.

Vaso-motor disturbances are not rare. Cyanosis of the limbs, œdema, ecchymosis upon the least trauma or without any apparent cause have been also observed.

Among the rare manifestations arthropathies have been recorded.

Course, Duration, Prognosis.—The disease is essentially progressive. Its duration is indefinite. Death usually occurs from some intercurrent disease, pulmonary especially.

Diagnosis.—The disease cannot be confounded with myxœdema, first because of its localization and next because of the pain upon compression of the enlarged areas of the skin.

Etiology.—A neuropathic tendency was traced in almost every case so far reported. Alcohol and syphilis may have some etiological importance. Disturbances of sexual functions (menopause, menorrhagia, abortion) have been reported as immediately preceding the onset. Trauma, emotion, exposure to cold have been traced in some cases as immediately preceding the onset. Women are more frequently affected than men.

Pathogenesis.—Autopsies have so far failed to reveal the true nature of the disease. Cases have been reported with morbid changes in the thyroid gland and pituitary body. In one of Dercum's patients there was an interstitial neuritis with a moderate sclerosis of Goll's columns. The general opinion is that the affection is due to a connective tissue dystrophy (fatty changes) with an involvement of the nerve-fibers (neuritis).

Treatment.—The foregoing remarks suggest a trial of thyroid therapy. In my case mentioned above I obtained amelioration of the paræsthetic disturbances from internal administration of extract of parathyroid glandules. Avoidance of fatigue and general hygienic measures are not to be neglected. Pain may be relieved by coal-tar products. Prolonged rest in bed may be beneficial.

SCLERODERMA

It is chacarterized by an induration of the skin and its adherence to the subcutaneous tissue.

Symptoms.—Before the formation of the characteristic skin the patient complains of distressing subjective sensations, as pain, itching, tingling, etc. Soon œdematous spots appear on the skin; they may remain **circumscribed** (**morphea**) or merge one into another and become **diffuse.** Gradually atrophy of the skin develops, so that in an advanced stage the skin is thin, tense, hard and glossy. Pigmentation, vitiligo are noticeable. The skin cannot be wrinkled.

In still more advanced period the atrophy progresses and involves the underlying tissues. The subcutaneous, muscular and osseous tissues become invaded.

The disease may be **localized** (morphea) or **diffuse, bilateral** or **unilateral.** The most frequent seat of the affection is on the face, neck, upper part of the thorax and upper extremities, especially the hands. When the face is involved, the features are drawn, immobile, mask-like, the lips are thin and the display of the muscles in emotions is of course disturbed. Mastication and speech are difficult.

When the hands are affected, the fingers are rigid, thin and retracted (**sclerodactyly**). Constriction and subsequently spontaneous **amputation** of phalanges may occur. Raynaud's disease may sometimes complicate sclerodactyly.

When the neck and thorax are affected, the respiration may be interfered with. Sclerodema has been observed in association with trophic disturbances in other parts of the body, viz. atrophy of one-half of the body, facial hemiatrophy.

Course, Duration, Prognosis.—In the large majority of cases the disease is essentially progressive. Cachexia and death are the ultimate terminations. Frequently the patient dies from some intercurrent disease.

Scleroderma is sometimes seen complicated by facial hemiatrophy and erythromelalgia.

Raymond and Guillain have observed a case of generalized scleroderma with bilateral ocular palsies and nystagmus. The latter symptoms were due to sclerosis of the ocular muscles, but not to a genuine paralysis.

Etiology.—Scleroderma has been observed in the course of lepra, syringomyelia, paresis and tabes. A neuropathic and arthritic predisposition play some part. Traumatism, emotion, exposure to cold, pregnancy have been considered as etiological factors. The disease may occur at any age, even in infants, but particularly between twenty and forty years of age. Women are more frequently affected than men.

Pathogenesis.—Pathologically the affection is characterized by a **sclerosis** affecting the skin and subcutaneous tissue. Atrophy and retraction follow. The blood vessels are also involved: a peri- and endarterities are always present.

These facts, however, do not explain the nature of the disease. Some authors believe that the vascular lesion, which is so constant, is the only cause of the disease. Others consider it as a trophoneurosis or angiotrophoneurosis. Others believe in a sympathetic origin and still others in infection. Finally a disturbance of the function of the thyroid gland may have something to do with the scleroderma.

Treatment.—Thyroid extract, iodides, local massage, electricity, also hypodermic use of thiosinamin (15 per cent. of alcoholic solution) every

other day are all the remedies advised. Not much reliance can be placed on any of them.

PROGRESSIVE FACIAL HEMIATROPHY

Symptoms.—In the majority of cases one or two brownish spots appear first on one side of the face. The underlying skin begins to appear glossy and to undergo atrophic changes. The subcutaneous cellular

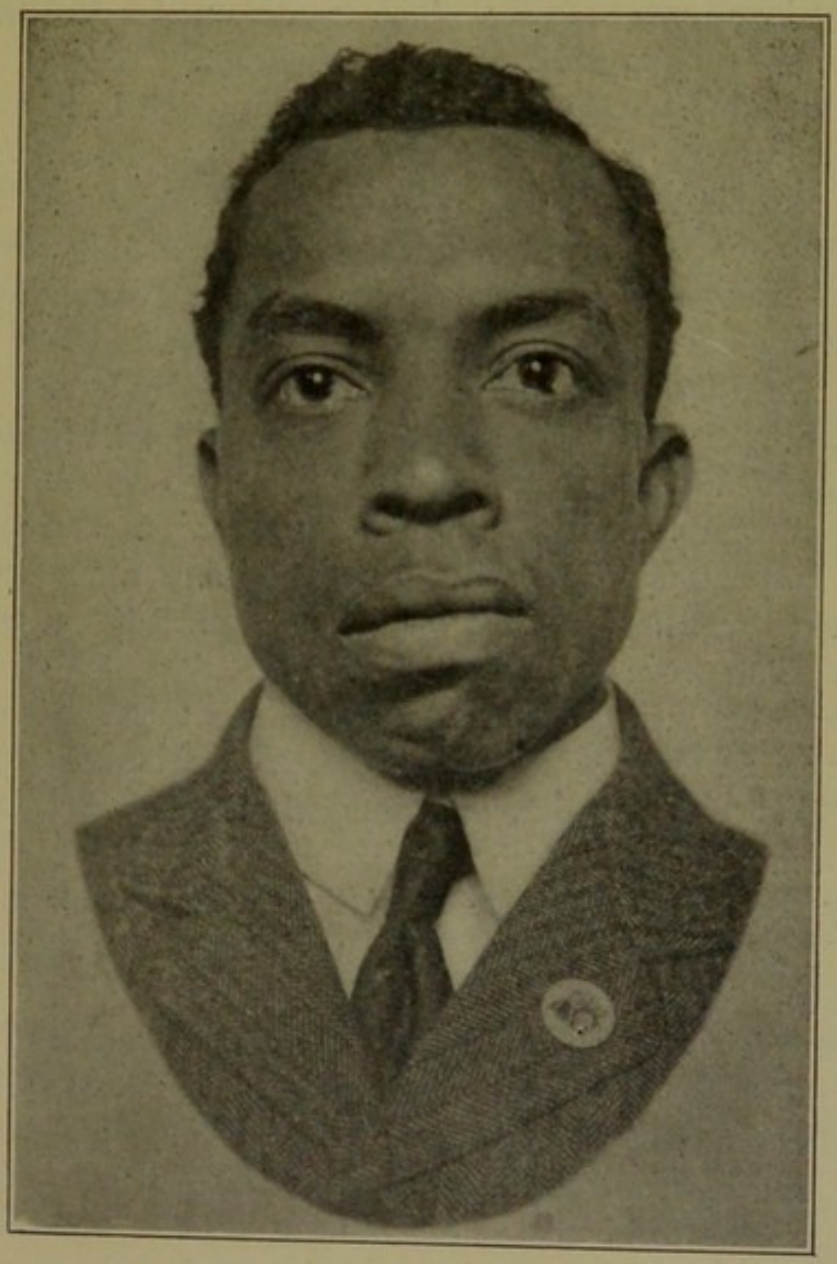


FIG. 167.—FACIAL HEMIATROPHY.

tissue follows. A depression develops because of the disappearance of fat.

The skin of the nose, orbit, cheek and lips becomes thin and wrinkled.

Other trophic disturbances occur. The teeth, the eyebrows fall out, the secretion of the sebaceous and sweat glands is diminished.

The face is asymmetrical and drawn to the normal side. The contrast between both sides is striking. The function of the muscles of the affected side is as a rule but slightly disturbed, as there is no paralysis. Their electrical reactions show no reactions of degeneration. The faradic contractibility is increased. However, an associated hemiatrophy of the masticators, of the tongue and of the palate which is occasionally observed, may interfere with mastication. The bones of the face participate in the atrophy in advanced cases. The frontal, molar and the maxillary bones, the nasal cartilages all atrophy and shrink.

Sensations are generally not disturbed, although in my case (*N. Y. Med. Jour.*, January, 1907) they were markedly altered. The patient frequently complains of paræsthesias (chilliness, numbness, etc.). Pain of a neuralgic character is sometimes present. In my case the disease began with severe pain around the right infraorbital foramen, which lasted two days; a few days later the hemiatrophy commenced.

Course, Duration, Prognosis.—The hemiatrophy is not always preceded by appearance of brownish spots. In my case a trigeminal neuralgia of two days' duration was almost immediately followed by the beginning of the atrophy. The disease is progressive. Sometimes the other side of the face becomes involved. Sometimes only a few symptoms are present (incomplete form). Sometimes other parts of the body may be implicated. The prognosis as to life is good.

The disease has been observed in association with tabes, syringomyelia, multiple sclerosis, epilepsy, chorea, facial tic. Dilatation, contraction and immobility of the pupil, also congenital palsy of ocular muscles, have also been observed.

Etiology and Pathogenesis.—Facial neuralgia, trauma, infectious diseases or localized infectious processes, organic nervous diseases (syringomyelia) are all possible causes.

There are at present two views concerning the nature of the affection. According to some observers, a **primary** atrophy of the subcutaneous cellular tissue is the essential feature. Others believe that hemiatrophy is of a **nervous** origin (trophoneurosis) and any of the following nerves is supposed to be the immediate cause: the sympathetic, trigeminus or facial. That the Gasserian ganglion and a cerebral lesion may also produce the disease, there are pathological proofs.

Treatment.—Local massage and galvanism may be tried. Gersuny's method of subcutaneous injections of paraffin should be undertaken for remedying the asymmetry of the face. The latter, however, is only an esthetic measure.

FACIAL HEMIHYPERTROPHY

It is a congenital condition. It is due to an anomaly in the development of the face. It is sometimes associated with a **congenital hemihypertrophy** of an entire half of the body. It consists of an increased development of the bony as well as of all the covering tissues of one side of the face. The skin is thick and coarse. Not only the superficial bones are involved, but also the palate, basilar process and sphenoid bone.

ACROPARÆSTHESIA

Symptoms.—The disease is characterized chiefly by **paræsthesias** in the distal ends of the extremities, viz. fingers and occasionally in the toes. The patient complains of a burning, tingling, pins and needles' sensation, of pain in the tips of the fingers or toes. The hands are most frequently involved. The disturbance is continuous, but in some cases it becomes aggravated at night or early in the morning. It is also worse when the fingers come in contact with cold objects, when exposed to cold or when placed in cold water. The affection is usually bilateral. There are **no objective** disturbances. The color of the skin is normal. The power of the hands as well as their function is also intact.

Course, Duration, Prognosis.—The onset is slow in the majority of cases. The disease lasts many years. Recovery is possible, but not frequent. The prognosis is good as to life. The disease incapacitates to a certain extent for physical work, as the latter increases the paræsthesias.

Etiology.—Undue exertion, exposure to cold are the exciting causes. Anæmia and pregnancy are predisposing factors. Women are more frequently affected than men. Washerwomen are particularly apt to develop the disease. The affection rarely occurs before the age of thirty.

Pathogenesis.—The disease is considered as a **vaso-motor neurosis.** Through some influence the arteries are in a state of spasm and thus the sensory nerve-endings not sufficiently nourished are being irritated. In advanced cases it is not impossible that a **neuritis** develops.

Treatment.—Avoidance of fatigue and of exposure to cold is the first indication. Arsenic, phosphorus, iron and quinine have been recommended. I have obtained favorable results from **nitroglycerine.** Very recently I studied a series of cases in which I have systematically applied Bier's method of induced hyperæmia. The results were very encouraging. A bandage was applied for an hour twice daily at the middle of the forearm (see, for details, *Therapeutic Gazette*, 1908). Galvanism and static electricity have also been advised.

ANGIONEUROTIC ŒDEMA (QUINCKE)

Symptoms.—The disease is characterized chiefly by **paroxysmal swellings** of the skin which are circumscribed and not inflammatory in nature, The **mucous membranes** are equally apt to be involved. The favorite seat is on the **face** and **lips,** but may also occur on the scalp. forehead, palate, pharynx, larynx and viscera (stomach and intestines), in rare cases in the brain (Osler). The articulations may also be involved by a sudden effusion. In one case, a girl of eighteen, the swellings appeared in the upper eyelids.

The circumscribed swelling resembles urticaria; it is round, of about 2 cm. in diameter; it is usually white, but it may be also somewhat reddish. It **does not pit on pressure.**

The swellings may last a few hours or days. They frequently disappear from one part of the body to appear in another. The patient does not suffer any special discomfort except some tension and stiffness, but **no pain.** When the mucous membranes are involved, the disturbance may interefere with the function of the pharynx, larynx, etc. Difficulty of swallowing and of breathing will ensue, and in some cases tracheotomy may become urgent. In exceptional cases albuminuria and hemoglobinuria have been observed. When the gastric and intestinal mucous membranes are affected, vomiting and diarrhœa will follow.

Course, Duration, Prognosis.—The disease may disappear spontaneously. In some cases it may last indefinitely. Recurrences are frequent. The prognosis, while not unfavorable, is, however, uncertain, as the disease is a very stubborn one. It is serious when the larynx is involved.

Diagnosis.—The paroxysmal character of the swellings, their circumscribed appearance, the absence of pain and the fact that they do not pit on pressure are all sufficiently typical signs for the diagnosis and cannot be confounded with other affections.

Etiology.—Heredity and neuropathic tendency play a prominent rôle. It is sometimes observed in several members of the same family. It is not infrequently associated with hysteria, epilepsy, exophthalmic goiter.

Physical and mental exhaustion are also predisposing factors. As exciting causes may be mentioned: cold, trauma, emotion, also toxic substances, such as alcohol and tobacco. Malarial poison may also produce the disease.

Males are more frequently affected than females. It usually occurs in young age.

Pathogenesis.—The disease is probably a vaso-motor neurosis, resulting either in a paralysis of the vaso-constrictors or stimulation of the

vaso-dilators. The consequence is a serious exudation. The latter is in the subdermal tissue.

Treatment.—In view of the neuropathic taint present in the majority of patients, a well-regulated life with plenty of rest and moderate exercises, also nutritious food, hydrotherapy, massage and other hygienic measures are the first indication in the treatment.

As drugs the following can be recommended: strychnia, atropin, quinine, nitroglycerine.

The condition of the pharynx and larynx should be watched, as operative measures may be necessary.

Alcohol, tobacco, or other stimulants are forbidden.

HEREDITARY ŒDEMA OF THE LEGS (MILROY'S DISEASE)

It was first described in 1892 by Milroy.

Symptoms.—Œdema may appear soon after birth or develop at the age of puberty. It is ordinarily confined to the legs up to the knees, but it may be confined only to the ankles or even reach the thighs. The skin of the swollen parts is usually pale. Acute attacks have been described. In such cases the swelling increases, the skin becomes red and some constitutional symptoms are present, viz. chills, fever, vomiting and pain in the abdomen. The chief features of this disease are: (1) absence of all local or general causes of œdema, (2) duration—mostly from birth, (3) the familial character and the presence of a neuropathic tendency in the family, viz. cases of epilepsy, imbecility, insanity. In Milroy's case he traced twenty-two individuals among ninety-seven persons in six generations.

Treatment.—Continuous bandaging is the only measure that is capable to give relief.

ERYTHROMELALGIA

It is characterized by reddening of the skin and paroxysmal pain in the feet and sometimes in the hands. It was first described and named by Weir-Mitchell in 1878.

Symptoms.—The feet are more frequently affected than the hands. **Pain** is the first symptom to appear. The great toe is particularly painful at the beginning. The pain occurs in paroxysms and later the intervals become shorter and shorter. The pain is excruciating and is relieved by recumbent position and cold. Station, gait, pressure or heat and exercise increase it. When the hands are affected, the patient gets relief by

crossing the arms over the breast or by raising them above the head. Such an attitude removes the blood from the periphery.

Soon **redness** of the skin develops. At first it is rosy-red, but later purplish-red. It is especially marked on the last phalanges. The veins are distended and a swelling is noticeable. The local temperature is increased and a hyperhidrosis takes place. The sensations may be diminished or increased.

While in the majority of cases the distal ends of the limbs are involved, nevertheless the pain and redness may sometimes extend to the entire limb. The symptoms may be observed also on other parts of the body, viz. face, ears, testicles, chest, back, also in rare cases in the mucous membranes of the mouth and throat (Seeligmüller.)

Other Symptoms.—During the paroxysms headache, vertigo, tinnitus aurium and even syncope may occur. Hardening of subcutaneous tissue, thickening of the nails, also œdema occasionally occur. Atrophy of the muscles of the extremities may develop in advanced cases. A diminution or increase of electrical contractility has been observed, but no reactions of degeneration.

Erythromelalgia is sometimes observed in the course of organic or functional nervous diseases, also in diseases of peripheral nerves.

Course, Duration, Prognosis.—Amelioration and aggravation in the course of the disease are observed. It lasts an indefinite time. The prognosis is unfavorable, as the disease is rebellious to treatment.

Diagnosis.—The cyanosis, local elevation of temperature, swelling, are sufficiently characteristic symptoms for diagnosis.

Erythromelalgia should be differentiated from angioneurotic œdema, acroparæsthesia and initial stages of Raynaud's disease (see these chapters). Mention should be made of so-called incomplete or **allied forms** of erythromelalgia. I observed a patient whose affection began with redness of the hands and the pain appeared only later; cold would increase the pain; exercise had no effect upon the pain. It is therefore a case which apparently belongs to the group of erythromelalgia and yet not typical.[1]

Etiology.—Cold, physical fatigue, are predisposing factors. Infections, rheumatism, syphilis and accompanying nervous diseases, such as hemiplegia, cerebral tumors, multiple neuritis, neuralgia, hysteria, neurasthenia, etc., are the main causes.

Pathogenesis.—The majority of observers consider the disease as a **vaso-motor neurosis.** Whether it is due to a paresis of the vaso-constrictors or to an irritation of the vaso-dilators, it is difficult to say. According

[1] *American Medicine*, August, 1907.

to some the spinal gray matter is the source of the disease. Others believe in a disease of the peripheral arteries. Weir-Mitchell thinks that there is a neuritis of the sensory nerves.

Treatment.—Electricity, application of cold and sedatives for relief of pain are the only measures employed. Perhaps Bier's method of artificial hyperæmia may render some service. If syphilis is suspected, and even without evidences of syphilis, mercury and iodides, also salvarsan may be tried. Removal of small portions of nerves or stretching of nerves, also amputations of toes or fingers, are to be attempted as a last resort.

RAYNAUD'S DISEASE

It is characterized by an ischemia or a hyperemia of the extremities which may terminate in a symmetrical gangrene. It was described by Raynaud in 1862.

Symptoms.—The disease presents three phases. The first is characterized by **local ischemia,** the second by a **passive hyperemia,** the third by **gangrene.**

First Phase.—Suddenly the fingers or toes or in exceptional cases the entire hand or foot become **pale,** wax-like and cold. This is accompanied or preceded by a numbness, tingling or severe pain. The condition may disappear rapidly or be followed by a **cyanosis** (**second phase**). In the latter case the skin becomes blue. Then the pain increases. The part is swollen but not œdematous. The anemia produced by pressure with a finger is slowly obliterated. The temperature is lowered. The pallor alternating with cyanosis (**local asphyxia**) or cyanosis without the preceding pallor, when repeated may become very frequent and then the patient enters into the **third phase** of the disease, characterized either by **active hyperemia** or **gangrene.** The blue color of the skin turns red. The part gets hot and throbs. This period lasts a variable time. It may be entirely missing. The asphyxia may persist and the circulation not be reëstablished. In such a case the final period of **gangrene** is to be feared. Gangrene may follow even the first stage of syncope: in such a case the parts never recover the circulation. Dark spots and vesicles appear; the serum of the latter dries up and a crust forms. When the latter falls off, an ulcerated surface is noticed which is slow in healing.

The gangrene may extend and involve an entire phalanx. The skin is parchment-like, the phalanx mummifies, a line of demarcation is formed and gradually the gangrenous part becomes detached. Cicatrization of the stump may be rapid or slow. During the entire process fever is absent, but the pain is excruciating. The suffering disturbs the patient's sleep, digestion and the general nutrition.

The phase of "pallor" and that of "cyanosis" correspond to "Raynaud's "local syncope" and "local asphyxia," respectively. They are, properly speaking, two successive stages of the disease and they eventually terminate in the final phase of symmetrical gangrene. However, the first two periods may exist a long time conjointly or separately without entering the final stage. Also the local syncope may exist a long time before the second period makes its appearance.

Local syncope occurs in attacks lasting but a few minutes or several hours. The patient complains of **"dead fingers."** The attacks recur sometimes with great regularity. They may occur either daily for some time or every two or three months. They are usually precipitated by exposure to cold.

The place of local asphyxia (cyanosis) is mostly preceded by attacks of local syncope and it may last days, weeks or months. In mild cases it may disappear, but in severer cases it is followed by the phase of symmetrical gangrene.

Other Symptoms.—Objective sensations are usually decreased. Anæsthesia is common in the period of syncope. Paræsthesia is present in the period of asphyxia. Hyperæsthesia is usually present after a paroxysm. Atrophy of the neighboring muscles with impairment of motion is not infrequent. Pupillary contraction, tinnitus aurium, albuminuria, may occur. Mental depression is a frequent accompaniment.

Not only the fingers and toes, but also the heels, coccyx, the ears, nose and the prominent bones of the cheeks may be affected.

The disease is usually **symmetrical and bilateral,** although unilaterality has been observed.

Complications.—Panas reports a case in which there was a relation between the cyanotic attacks and the condition of the arteries in the fundus oculi: when cyanosis occurred, the arteries were contracted; when the former subsided, the latter became normal.

In some cases Raynaud's disease was preceded or followed by attacks of transient hemiplegia, monoplegia, aphasia or amblyopia. Epileptic convulsions have been observed in association with attacks of local cyanosis. Hemoglobinuria is a rare complication.

Purpura and urticaria have been exceptionally observed.

Course, Duration, Prognosis.—The course may consist only of the first or of the first and second phases. More frequently the entire cycle is observed. The duration of the disease is from one to three months. After an interval of months or years another attack may occur and affect another part of the body. Life is usually not endangered, except when suppuration with septic infection is present.

Diagnosis.—The disease is easily recognized from its typical symptoms. Local gangrenous patches may also be observed in **hysteria,** but the necrosis is superficial and no changes of the blood vessels are found.

In **lepra** phalanges fall off without pain. Gangrene caused by intoxication with **ergot** presents special features characteristic of ergotism.

I saw a case of local gangrene caused by subcutaneous injections of **adrenalin** above the lesion.

There are also allied cases in which the picture of Raynaud's disease is not complete, cases that present intermediary forms, which may resemble

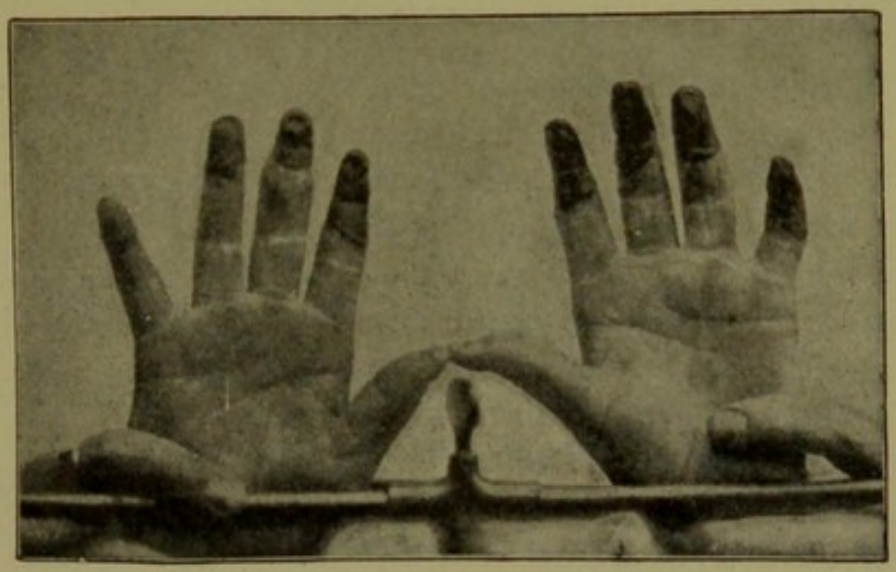


FIG. 168.—SYMMETRICAL GANGRENE. (*Dehio*).

one or another of the localized neuroses. In the case referred to in the chapter on Erythromelalgia the symptoms were those of Raynaud's disease and of erythromelalgia.

Etiology.—The disease occurs not infrequently in the course of organic nervous diseases and psychoses, also in functional nervous disorders. Infectious febrile diseases, syphilis, tuberculosis, lepra, diabetes, may be accompanied or followed by Raynaud's disease. It has been observed in the course of scleroderma. Anæmia and a congenitally small aorta play an important predisposing part.

Emotion, trauma, menses, cold, are exciting causes. Zenner observed a case due to caffein poisoning. Young persons, and especially women, are more frequently affected than old individuals and men.

Pathogenesis.—Pathological investigations show a localized involvement of the blood vessels (arteritis) and of the nerves (neuritis), but these changes are secondary. The majority of observers consider Raynaud's disease a vasomotor neurosis (see also erythromelalgia). Raynaud believed that the vaso-motor centers are in a state of irritability; the

irritant comes from the periphery (cold), the paroxsysmal contraction of the blood vessels comes from the center.

Treatment.—Rest and local applications of heat are important. Good hygienic and dietetic measures are necessary.

Pain can be combated by usual means, such as coal-tar products, etc. Amylnitrite in inhalations during the attacks may give relief from pain, but it is not always effective. Nitroglycerine and ergot have also been recommended. Bier's method, consisting of an application of bandage above the affected parts for an hour twice daily, will induce an artificial hyperæmia and give relief. It may be of benefit in the first two phases of the affection. Cushing's method may be very useful. He bandages lightly the limb, then applies a tourniquet for a minute or two. When the latter is removed, a large amount of blood rushes to the parts which were made anæmic. It may be tried several times a day.

Barlow recommends the following procedures. "Immerse the limb into a basin containing salt and tepid water; one pole of a constant current battery is placed in contact with the upper part of the limb above the level of the water, the other pole in the basin. The current is then made and broken at frequent intervals so as to obtain moderate contractions of the limb. The patient is also told to move the digits while electricity is being applied." He claims that considerable relief is obtained in cases of recurrent local asphyxia. High-frequency current has also been recommended. In case of gangrene antiseptic applications with rest of the limbs are the only measures besides operative considerations.

CHAPTER XXVIII

NERVOUS SYMPTOMS PRODUCED BY INTOXICATIONS

A. METALLIC POISONS

I. Lead Intoxication

LEAD may affect the central and the peripheral nervous system, also produce psychoses.

Pathology.—A striking and at the same time a common feature is **arteriosclerosis.** It is not, however, conclusively proven that lead is the direct cause of the thickening of the vessel walls. In the **brain** the meninges are altered: pachymeningitis, leptomeningitis with œdema and atrophy of the cerebral tissue are frequently observed. Changes of the cortical cells, proliferation of the nuclei in the vessel walls and of the glia-cells about the vessels have been found. In the **cord** chronic anterior poliomyelitis may occur. In a case under my observation I found microscopically degeneration of the posterior columns (*American Medicine*, 1905). The **peripheral nerves** present parenchymatous degeneration. For details see the chapter on multiple neuritis.

Symptoms. (*a*) **Cerebral Encephalopathy.**—In **acute poisoning** headache, vertigo, epileptiform convulsions, delirium and hallucinations, also coma, are observed. The **chronic form** may be accompanied by persistent headache, also convulsions, attacks of hemiplegia, attacks of aphasia, finally by psychoses (see below).

Functional nervous diseases may be induced by chronic lead poisoning. Choreiform movements, hysterical hemianæsthesia have been observed. Generalized or focal epilepsy are not infrequent.

The psychoses due to lead may present themselves in the form of a delirium or more frequently by stupor. Delirium usually lasts weeks. Symptoms of **paresis** are not rare, but they do not present the typical picture of this psychosis. The condition is therefore called **"pseudo-paresis"** (see Paresis). Delusions of persecution with terrifying hallucinations occur. The latter are particularly present in cases which are at the same time alcoholic.

Eye Symptoms.—Palsies of the ocular muscles, also nystagmus, may occur. Hemianopsia, usually homonymous, amaurosis are usually

transitory. In some cases profound changes in the optic nerves take place.

(*b*) **Cord Symptoms.**—Muscular atrophy due to poliomyelitis is not rare. Aran-Duchenne's type of atrophy with the claw-like hand is occasionally seen. Erb's type has also been observed (see Progressive Muscular Atrophy).

(*c*) **Peripheral Nerve Symptoms.**—See chapter on Multiple Neuritis.

Course, Duration, Prognosis.—Cerebral symptoms present a serious outlook. When optic neuritis is established, the prognosis is doubtful. Blindness may be the outcome. When cardiac or renal complications occur, the prognosis is grave. In most favorable cases the prognosis should be guarded, as recurrences are not infrequent. Finally the patient's future will depend upon whether he is removed from the influence of lead or not.

Etiology.—The occupations of painters, compositors, occupations requiring a free handling of lead and lead colors, especially lead carbonate in a dry state, the use of certain cosmetics, the use of water kept in lead vessels, are the usual sources of lead intoxication.

Treatment.—Elimination of lead is the first indication. Purgatives and diuretics are necessary. In view of the obstinate constipation characteristic of lead poisoning, moderate doses of atropine should be given with the purgatives in order to control the intestinal spasm. To enhance diuresis large quantities of water are to be given. Baths of potassium sulphuret will help elimination of lead: to six inches depth of water six ounces of potassium sulphuret are added. Potassium or sodium iodide internally is excellent. Exclusive milk diet at first and later milk and vegetable diet, hydrotherapy, avoidance of stimulants, including tea and coffee, are the main elements of the treatment. Insomnia, headache, gastric disturbances, convulsions, are treated accordingly. Venesection and lumbar puncture may be useful in cases of cerebral congestion.

Paralysis is to be treated with massage and electricity.

II. Arsenical Intoxication

Pathology.—No special lesion has been found in the brain. In the spinal cord there may be involvement of gray and white matter (myelitis). Changes in the peripheral nerves are the most important. **See Multiple Neuritis.**

Symptoms.—In chronic cases there may be present **cerebral symptoms,** viz. impairment of memory, of intellectual faculties. In acute cases delirium and confusion are the usual manifestations.

Eye symptoms may be amblyopia or amaurosis, ocular palsies. Puffiness of the eyelids, congestion of the conjunctivæ, are quite common.

When the **cord** is involved, symptoms of myelitis are evident.

For the symptoms of **arsenical neuritis** see chapter on Multiple Neuritis.

Course, Duration, and **Prognosis** are usually favorable and similarly to all toxic conditions may be serious, if the intoxication is severe and protracted.

Etiology.—Wall paper, colored dress goods, arsenical paint, rugs, carpets, crayons, beer (glucose in England), medicinal arsenic and finally intentional absorption are all sources for intoxication.

Treatment.—See preceding chapter.

III. Mercurial Intoxication

Pathology.—Cerebral œdema, degenerative changes in the tracts of the cord and in its gray matter, atrophy of the nerve fibers, sclerotic changes of the vessel walls, are the usual findings.

Symptoms—Cerebral excitement or else depression, vertigo, tremor, choreiform movements, convulsions, muscular weakness, disturbance of speech (caused by tremor of lips), diminution or loss of cutaneous sensibility, amblyopia and sometimes optic neuritis are the chief symptoms. In advanced cases impairment of memory, confusion and mild dementia may develop. **Tremor** is the most conspicuous symptom. At first it is very slight. Soon it becomes marked upon the least emotion. The range of the tremor may be small or gross. While at first it is present only on voluntary acts, later it appears even when the patient is at rest. The hands and lips are affected first, but later all extremities and face become involved. The twitchings may sometimes be very violent.

In the chapter on Multiple Neuritis details are given concerning the manifestations of the peripheral nerve involvement.

Course, Duration, Prognosis.—See the previous chapter.

Etiology.—Manufacture of mirrors, of rubber, the use in paints, in artificial flowers, in aniline colors, in fireworks, in mining, in smelting, in manufacture of thermometers, finally the use for medicinal purposes are all the usual sources of mercurial poisoning.

Treatment.—See preceding chapter.

IV. Carbon Monoxide Intoxication

Acute poisoning may be followed immediately or after a considerable time by persistent disturbances in the nervous system.

Pathology.—Diffuse encephalomyelitis is the characteristic feature of the changes in the central nervous system. Hyperæmia, hemorrhages in the cortex, in the basal nuclei, in the internal capsule, **foci of softening** particularly in the **lenticular nuclei** (see Diseases of Basal ganglia), poliomyelitic foci in the cord, peripheral neuritis, are all conditions met with.

Symptoms.—Immediately after the poisoning the following nervous disturbances are observed: headache, vertigo, nausea, vomiting, muscular weakness, relaxation of sphincters and loss of consciousness. Muscular twitching and even convulsions may occur.

If consciousness is regained, there is a marked mental hebetude.

The **permanent** nervous symptoms are: neuritis, neuralgias, anæsthesias, choreiform movements, intention tremor and scanning speech. For details on neuritis consult the chapter on Multiple Neuritis. Ocular palsies, nystagmus, partial or complete blindness may occur.

Paralysis, hemiplegia or monoplegia and mental disturbances are not rare. They may develop weeks after the poisoning. Amnesia is quite common. In my case (*New York Medical Jour.*, 1906) there was persistent antero-retrograde amnesia. Confusion or stupor with or without hallucinations may last from weeks to two or three months. The paralytic symptoms may be permanent. Cases with total dementia have been recorded. All these sequelæ may appear immediately or weeks after the poisoning. It is important to know that sometimes very serious sequelæ develop in cases of mild poisoning.

Course, Duration, Prognosis.—Recovery may follow in a very short time. It depends frequently upon the promptness with which treatment is instituted. The prognosis should be guarded in view of the sequelæ that may follow some time after the poisoning. Cerebral manifestations are usually of grave omen.

Etiology.—Illuminating gas, gas works, furnaces, heating apparatuses with poor draughts, tar distilleries, chemical factories, laboratories, are the sources of carbon monoxide poisoning.

Treatment.—Removal from the poisoned atmosphere, inhalation of oxygen, venesection followed by injection of normal salt solution, administration of stimulants, are the immediate measures. Prophylaxis is the most important feature.

V. Manganese Intoxication.

L. Casamajor has very recently (*J. Am. Med. Assn.*, 1913) called attention to a symptom-group produced by poisoning with manganese. The cases that came under his observation occurred among workers in the

separating-mill connected with a mine from which zinc, iron and manganese are the principal products. The handling of the ground ore is done in a dry state; the air which the workmen breathe is laden with a very fine gray dust.

As zinc and iron have never produced a poisoning similar to the one under discussion, the author concludes that manganese is very probably the etiologic factor.

Symptoms.—A man working in this dust for a period of six months or more begins to notice a difficulty of walking down an incline. If he does, he must run fast until he meets an object (propulsion). Walking up a hill or on a level ground is normal. Walking backward is impossible and if an attempt is made, the patient falls (retropulsion). In addition to the disturbance of gait, the patient may have pain and stiffness in the legs. The picture resembles that of paralysis agitans.

Other Symptoms.—Defective hearing, slurring speech, mask-like facies, tremor of the tongue and hands, unsteady station. These symptoms were not found simultaneously in all the patients. Sensations, reflexes, sphincters, are all normal.

Course, Prognosis.—The course is chronic. When removed from the dust the patient may improve, but this is not in every case. The outlook is poor.

Pathology.—In one case that came to autopsy the following condition was found. Perivascular spaces of the lenticular nuclei and of the outer part of optic thalami are larger than normal. No other gross changes have been observed.

B. ORGANIC POISONS

I. Alcoholic Intoxication. Alcoholism

Alcohol has a special predilection for the nervous system. Its effect upon the nervous system differs in **acute** and **chronic** intoxications.

Acute Alcoholism.—Mental symptoms are predominating. Delirium, confusion and stupor with or without hallucinations and illusions are the characteristic features. They are fugacious, transitory. Recovery usually follows. The promptness of the recovery depends upon the effect of alcohol on different individuals. When the alcoholic abuse is frequently repeated, a state of chronic alcoholism develops.

Chronic Alcoholism.—When the absorption of alcohol is slow and prolonged, the changes it produces in the individual's physical and mental spheres of life are enormous.

Physical Symptoms.—**Gastro-intestinal** disturbances are marked by anorexia, vomiting, constipation or else diarrhœa with bloody discharge.

Tremor is common. It is passive and intentional. It affects the extremities as well as the tongue and lips.

Epileptic convulsions are not rare. They may be unilateral or generalized.

Attacks of **apoplexy** followed by paralysis occur.

Neuritis is very common. For details see chapter on **Multiple Neuritis.**

Vaso-motor disturbances, increased mechanical irritability of muscles and nerves, various subjective disturbances, as pains, coldness, numbness, palpitation, fears, general weakness, are all accompanying symptoms.

Ocular disturbances are not infrequent. They are: contraction of the visual field, scotomata, optic atrophy and optic neuritis.

Mental Symptoms.—In my series of 277 cases of chronic alcoholism (*J. of Am. Med. Assn.*, 1907) the mental disturbances followed repeated subacute attacks or repeated attacks of delirium tremens, but also insidiously and progressively without preliminary acute symptoms. The mental state consists chiefly of a gradually developing intellectual feebleness, viz. **dementia.** Before the latter becomes conspicuous, the patient begins to show undue irritability which at first is noticeable only to the immediate surroundings. At the same time appears a weakness of the will power and of energy. The patient soon becomes depressed, his memory clouded, the power of application for work, physical or cerebral, decidedly impaired. The sadness, the realization of his physical and mental impotence, lead him directly to delusive ideas which become intensified by hallucinatory images, and criminal tendencies are not infrequently observed. Gradually the moral sense, the sense of propriety, becomes deteriorated, conventional laws are totally ignored. The patient becomes apathetic, brutal. His cerebral functions are totally disorganized, the judgment infantile and dementia is permanently established.

An **acute episode** in the course of chronic alcoholism is seen in

Delirium Tremens.—As the name implies, it is characterized by a **delirious state** and **tremor.** The condition usually develops gradually. At first there is a state of restlessness, insomnia with hallucinations; tremor appears in the hands and tongue. These few symptoms continue to increase in intensity. The patient becomes very restless, very talkative, attempts to get up, if he is in bed, runs from place to place; talks aloud and appears to converse with imaginary persons. His actions are prompted by various hallucinatory images. Through the visual apparatus he sees

terrifying animals, assassins, robbers, executions, fires, etc. Through the auditory apparatus he hears threats, oaths, bad names, etc. Through olfactory apparatus he perceives the most repugnant odors. Through the gustatory apparatus he tastes nauseating substances. Through the sense of touch he believes himself undergoing torture. Being under the influence of these hallucinations the patient is excited, defends himself, threatens, strikes or else is terrified and lays immobile. Orientation in time and space is frequently affected. A patient, for example, may remember the objects of his room, but does not recognize that he is in his own room. A feeling of great anxiety is ever present because of the hallucinations, but when the patient reaches the climax of his delirium, the anxiety is considerably diminished and may even disappear. He then becomes indifferent to the hallucinations.

These mental phenomena are accompanied by a **tremor** affecting the hands or the upper and lower extremities or the latter and the face. In some cases the tremor continues even in sleep. The speech shows an incoördination: stumbling over syllables and words, difficulty in pronunciation. The **loss of appetite** is striking during the delirious state. **The pulse** runs from 80 to 115 and is dicrotic. The temperature is usually normal, but it may rise as high as 103° and even 105°. Constipation is the rule. Albumen is present in the majority of cases. The polynuclear leucocytes of the blood are increased and the mononuclear are decreased (Elsholz).

The **duration** of an attack is usually from two to eight days, according to its intensity. It usually ends in a deep sleep lasting from twelve to thirty hours. When the patient awakens, the delirium and hallucinations are gone, orientation is good, but some mental hebetude remains, the memory is somewhat cloudy and a slight tremor persists.

Alcoholic **serous meningitis** (**wet brain**) is quite frequent in chronic alcoholism. It usually follows an attack of delirium tremens. In such a case the patient from the onset enters into semi-comatous state. Delirium is present but not of a violent nature. The patient though affected by hallucinations lies motionless and mutters in a low voice. He can hardly be roused for food. The pulse is rapid, temperature normal. The pupils are small. Hyperæsthesia is marked. Recovery is possible especially in those cases in which the neck is not rigid (Dana). In cases with rigid neck Dana believes the patients die. In such cases the semi-comatose state gets deeper and deeper, general rigidity develops, the abdomen gets retracted. If recovery occurs, the condition normally lasts about four weeks.

The **prognosis** depends upon whether complicating conditions are

present or not. It is grave in cases of trauma, infectious diseases, nephritis, cardiac weakness, pneumonia.

Recurrences are not infrequent. An attack may come on from some insignificant cause, as slight trauma or an ingestion of a small amount of alcohol after a period of abstinence, or else from an intercurrent febrile disease.

Pathology of Alcoholism.—Little or no special changes are found in acute alcoholism.

In chronic alcoholism the most constant alterations are observed in the **blood vessels. Atheromatous changes** of the minute blood vessels are conspicuous. They are enlarged, tortuous. In their vicinity neuroglia cells are abundant. The perivascular spaces are filled with lymphoid cells. **Pachymeningitis** and **leptomeningitis,** œdema of the brain, pachymeningitis hemorrhagica are all quite frequent. Degeneration of the cortical cells, especially in the motor areas; also of the descending tracts in the internal capsule and in the spinal cord; finally in the cells of the anterior cornua in the cord have been repeatedly found. In delirium tremens there is a great tendency to minute hemorrhages especially in frontal and central convolutions of the brain, frequently also in the gray matter of the aqueduct of Sylvius and around the third ventricle. As to the **peripheral nerves,** they particularly are affected by alcohol (see chapter on Multiple Neuritis). Korsakoff's psychosis is described in connection with Alcoholic Multiple Neurit's.

Treatment.—Withdrawal of the poison is the first indication. Some believe in gradual reduction of the beverage, others in sudden removal. This can be well accomplished by isolation. Sleep must be induced by all means. Bromides (twenty grains), paraldehyde (one dram), chloral hydrate (30-60 grains), sulphonal (gr. x), trional (gr. x), veronal (gr. x) are good drugs for insomnia. Morphia (gr. 1/8) associated with chloral (gr. xx) is especially advantageous. A tepid bath of half an hour's duration is also useful in insomnia. As withdrawal of alcohol may cause collapse or exhaustion, a hypodermic injection of ergot may be very efficient. In case the patient is exceedingly restless, or violent, a hypodermic injection of apomorphine (gr. 1/10) is advisable.

After sleep is secured, the regular treatment will consist of a complete or partial rest, moderate outdoor exercises, regular meals, hydrotherapy and internal administration of strychnia. Milk should be the main article of food. Meat is to be avoided. Purgatives (preferably saline) are useful when given two or three times a week. Coffee and tea are to be avoided.

Bromides administered regularly and for a prolonged period gave me

very satisfactory results. Hyoscine hydrobromate is also useful. **Capsicum** associated with strychnia is commendable.

For the treatment of alcoholic neuritis see chapter on Multiple Neuritis. In the majority of cases of chronic alcoholism institutional treatment is the most efficacious.

II. Morphine Intoxication. Morphinism

There are two phases to be considered in the history of morphinism. One is due to chronic intoxication, the other to abstention from the poison after a long period of intoxication.

Symptoms of Chronic Intoxication.—They usually appear after a few months of use of the poison. The exact time of their development varies with the individual. The symptoms are **physical** and **mental.**

1. The **physical** manifestations concern the motor, sensory and vaso-motor apparatuses.

Motor.—They are: muscular weakness, tremor, incoördination, cardiac weakness, vesical weakness and disturbance of ocular accommodation. Epileptiform convulsions, attacks of angina pectoris are sometimes observed.

Sensory.—They are: superficial or deep anæsthesias, hyperæsthesias or paræsthesias.

Vaso-motor.—Diminution of secretions. When the salivary and sebaceous glands secrete insufficiently, there is a dryness of the mouth and throat, a dryness of the skin. In the latter case the skin is easily irritated and furuncles easily develop. The gastric and pancreatic juices are also diminished and gastro-intestinal disturbances are frequent. The menstrual flow is suppressed. The amount of urine is decreased and renal congestion with albuminuria are sometimes observed. Impotence is frequent.

Perspiration on the contrary is increased. A morphinomaniac has a pale face, cold extremities, his temperature is below normal.

Cachexia gradually develops and may become alarming, if prompt treatment does not interfere.

2. **Psychic** manifestations are constant. The patient notices a gradual loss of mental energy. His will power is diminished. His thoughts and acts are characterized by an indifference, by apathy. His moral conceptions weaken equally and the tendency to deceive is marked. At the same time he is irritable, has outbreaks of anger upon the least provocation so that at times he may become dangerous. Gradually the memory becomes more and more impaired, the mental faculties low (dementia). Depression is marked and may lead to suicide. (See my article in the

Jour. of Am. Med. Ass., July, 1908.) Insomnia is very marked. Nocturnal hallucinations of a terrifying nature torture the patient, but they usually disappear during the day. Hallucinations of sight are more frequent than any other form.

Symptoms of Abstinence.—They depend upon whether the suppression of morphine is **gradual** or **sudden.**

In case of gradual removal of the drug there is a certain general malaise with loss of appetite, nausea, vomiting and diarrhœa. Noises in the ear, peculiar visions are present. Neuralgic pains, a general sensation of cold distress the patient. He is also in a state of anxiety and depressed.

When the suppression of morphine is complete and sudden, there is, first of all, a sense of great muscular fatigue. The perspiration is abundant, the tremor is generalized. Violent abdominal pains with vomiting and diarrhœa are sometimes extreme. A delirium with hallucinations develops rapidly. In some cases there may be a cardiac and respiratory failure and death may be imminent. A hypodermic injection of morphine promptly administered may save the patient.

The outlook in morphinomania is unfavorable, as recurrences are frequent.

Treatment.—Withdrawal of the poison is the main indication. It can be accomplished very **gradually,** or **rapidly** or else **abruptly.** The **gradual** method is applicable to patients that are very weak and more or less cachectic; syncope is to be feared in them. In such cases hypodermic administration of strychnia (gr. 1/60) several times a day is to be recommended.

In robust individuals the **rapid** suppression is advisable.

With the **abrupt** method one runs a great risk. When cardio-vascular disturbance is present, a sudden withdrawal may bring on a collapse or even death.

Whatever method is adapted, during the withdrawal particular attention should be given to the patient's general strength. Alcohol can be used freely. In extreme cases when collapse is threatened, a hypodermic of morphia will improve the condition.

The gastro-intestinal disturbances, which are so conspicuous, are relieved by sodium bicarbonate.

Good hygienic measures, hydrotherapy (cold douches), massage, nutritious food are not to be neglected.

Finally the question of **prophylaxy** is the most important. Morphine should be prescribed only in the most urgent cases and a patient should never be trusted with a syringe and the drug. Institutional treatment is always adorable.

Cocaine Intoxication. Cocainism

The symptoms of **acute** cocaine intoxication are: general excitement, restlessness, talkativeness, paræsthesia of the extremities and tinnitus aurium. Sometimes instead of excitation there is on the contrary cerebral depression, nausea and vomiting, abundant sweating, cold extremities, tachycardia. Epileptiform convulsions may occur. Recovery may follow, but death may also take place either in syncope or in convulsions.

In **chronic** cocainism there is at first a tendency to muscular and mental agitation. Gradually the latter becomes permanent. A feeling of exhilaration in the mental and sensory spheres is quite marked. The patient exhibits great activity. Insomnia develops. Hallucinations make then their appearance and persecutory delusions may accompany the hallucinations. In advanced stages of the disease the exaltation period is replaced by a period of depression and even stupor.

Sensory phenomena are quite conspicuous. Various paræsthesias are present. Not infrequently the patient complains of a sensation of insects crawling under his skin. Very often there are sharp pains in the extremities. Anæsthesias may be present. The acuity of vision is diminished. Tachycardia, rapid respiration, sweating, anorexia, diarrhœa and emaciation are the usual symptoms.

In advanced cases cachexia and mental enfeeblement (**dementia**) are very marked. Syncope may occur at any time. (See my article in the *Jour. of Am. Med. Assn.*, July, 1908.)

Treatment.—See the preceding chapter. It may be added, however, that sudden withdrawal of cocaine is preferable to the slow method.

CHAPTER XXIX

NERVOUS SYMPTOMS CAUSED BY SOME SPECIAL INFECTIONS

TETANUS (LOCKJAW)

The disease is due to a special bacillus discovered by Nicolaier in 1885.

Pathology.—There are no characteristic changes in the brain and spinal cord, but the toxins elaborated by the tetanus bacillus reach the central nervous system through the nerve trunks. Hyperæmia, œdema and minute hemorrhages in the brain and meninges have been observed. Congestion and softening, also changes in the cells of the cord and medulla have been found.

Symptoms.—A few days after the infection the muscles of the **jaw** and the **neck** become rigid, producing the symptom **trismus,** or "lockjaw." The patient is unable to open the mouth freely. The masseters are very hard to touch. The facies is immobile, forehead wrinkled, corners of the mouth retracted, lips protruded, eyes partly closed. This expression of the face is given the name of **"risus sardonicus."** Gradually the bilateral tonic spasm increases and spreads. The muscles of the trunk and abdomen become involved. The patient's body is arched, resting on his heels and the back of his head (**opisthotonos**). The spasm of the **diaphragm** interferes with the breathing. The involvement of the **pharyngeal** muscles produces difficulty in swallowing. Gradually the muscles of the extremities become involved, although the hands are usually spared. The patient is confined to bed, his entire musculature is rigid, the breathing is difficult, the jaws are tightly pressed against each other, the head is drawn backward. The last touch or handling of the patient causes a **convulsive contraction.** Sometimes the latter occurs spontaneously. It causes violent **pain.** Voluntary urination and defecation are impossible. Constipation and retention of urine are present. Sweating is a constant symptom. The temperature rises only before death. The pulse is accelerated. A moderate degree of leucocytosis has been observed by Brown and others.

Sensations are normal. The mentality is preserved.

Course, Duration, Prognosis.—In the acute case death may occur in a few days. Life is in danger either from asphyxia (laryngeal spasm),

cardiac paralysis, profound exhaustion or excessive rise of temperature. In subacute cases the outlook is favorable. In the acute forms the mortality is high in cases occurring early after severe trauma. The most favorable outlook is in cases which develop late after trauma.

Complications may develop or follow tetanus. Among them should be mentioned: nephritis, bronchitis or bronchopneumonia from difficulty of expectoration, rupture of muscles, fracture of the spinous processes from violent spasms. Sometimes for months after recovery a stiffness of muscles persists. Mental disturbances, suppuration of submaxillary and parotid glands have also been observed after recovery from tetanus.

Diagnosis.—**Strychnine poisoning and hysteria** should be always thought of in making a diagnosis. In the first the onset is rapid; gastric disturbances or tetanic contractions of the extremities appear first, while in tetanus trismus is first. Besides, the convulsions are violent and present periods of complete relaxation; the retina is hyperesthetic and objects look green; duration is brief—either recovery or death. Besides, a history of poisoning is present.

In hysteria there is no trismus. The presence of hysterical stigmata will decide the diagnosis.

Hydrophobia is recognized by absence of trismus and opisthotonus by violence of repiratory spasms and by psychic disturbances.

Etiology.—The usual mode of infection with Nicolaier's bacillus is through a wound. The epidemic or sporadic character of tetanus is well known. It is more prevalent in the hot than in the cold months of the year.

Infection may also take place in new-born children through the umbilicus. This is **tetanus neonatorum.** Trismus is here the most conspicuous symptom, which prevents the infant from taking nourishment. The prognosis is very grave.

Treatment.—Prophylaxis, consisting of rigorous antiseptic measures, is urgent in every case of infection.

As soon as the wound occurs, thorough disinfection and thorough cauterization of it must be done as promptly as possible. Immediately or very shortly after, **antitoxin** as a preventive measure should be used. About 500 units should be injected for this purpose. The early administration of antitoxic serum is of great importance and Behring urges its administration within the first twenty-four hours. Unless it is injected before trismus makes its appearance, its value is very small. Vaillard (*Bull. de l'Acad. de. Méd.*, 1908) injected the serum as a prophylactic measure in 13,124 horses and none contracted tetanus. His observations also show that when tetanus developed notwithstanding the preventive

injections, its course was milder. The high mortality depends largely upon the late use of the serum. As to the amount to be injected, it is advisable to inject at first 2.25 grm. and 0.5 grm. for each of the following doses. In extreme cases instead of subcutaneous injections the serum may be injected into the spinal canal after a small amount of cerebro-spinal fluid is allowed to escape.

Although satisfactory results have been reported, nevertheless a great many failures have been observed.

General measures should not be neglected. Absolute quiet, avoidance of manipulation of the body are necessary. Feeding should be done through the nasal cavities, if trismus exists. Bromides, chloral, morphia, warm baths will be utilized for relief of the tonic spasms. In spite of the spasms, cardiac stimulants are sometimes necessary. Alcohol, strychnine, digitalis, camphor are the most useful drugs. The latter three are to be given hypodermically. Care must be taken of the functions of the viscera.

In 1905 Meltzer proved experimentally that magnesium possesses an inhibiting power over the processes of the body, viz. inhibits respiration and produces paralysis when given by intravenous injections; when injected subcutaneously, it produces narcosis with muscular relaxation; when injected into the spinal canal it produces anesthesia. Since then it has been shown that an **intraspinal injection of magnesium sulphate** in cases of tetanus controls the convulsions and leads to recovery. The quantity to be injected is 1 c.c. of 25 per cent. solution to each 25 pounds of body weight. The injections may be repeated several times in case the results of the first injections are not entirely satisfactory. Kocher (*Corresp.-Blatt f. Schweiz. Aerzte.*, 1912) believes that 15 per cent. or even 10 per cent. solution of magnesium sulphate is sufficient. The greater susceptibility of children to toxic action must be borne in mind: 2 c.c. should be the maximum dose for a child. In view of the cumulative action of the drug, repetition of the dose should be made not oftener than at an interval of twenty-four hours. Kocher warns that if no cerebro-spinal fluid flows upon lumbar puncture, injection of magnesium sulphate is dangerous. The injections of magnesium may be associated with administration of antitetanic serum. This latter combined method has given highly favorable results.

Baccelli treats tetanus with **injections of carbolic acid** in 2 per cent. solution. He commences with a daily dose of 0.30 to 0.50 watching carefully the condition of the urine and the patient's tolerance. Gradually he increases the dose to 1 grm. in several injections in twenty-four hours. In very grave cases higher doses may be given. Baccelli claims (*Berl.*

klin. Wchn., 1911) that with his method the mortality in his ordinary cases has been only 2.12 per cent. and in the very grave cases 18.5 per cent.

Atkey (*Lancet*, 1913) basing himself on the effects produced by Souttar by injecting paraldehyde and ether intravenously, used these drugs in conjunction with copious injections of normal saline solution in treatment of tetanus. In a case of a boy of 19, he made twelve intravenous injections of a solution of paraldehyde and ether, 5 c.c. each in 150 c.c. of normal solution and the result was excellent. The boy recovered. The drugs apparently act as an anæsthetic and hypnotic.

Under the name of **Cephalic Tetanus** is described a variety of tetanus which is characterized by spasms confined mainly to the head and face and which results from injuries of the head. Spasm of the facial muscles, then trismus, spasms of pharyngeal, laryngeal and respiratory muscles are the symptoms observed. The rigidity may spread to other muscles.

This form is also called hydrophobic tetanus, because of the spasmodic contractions of the œsophagus. The majority of cases ends fatally.

Hydrophobia. Rabies. Lyssa

The infectious element of this disease is not yet known. It attacks animals and from them is communicated to man by inoculation through a bite. The exact nature of the virus is unknown.

Pathology.—In 1900 Van Gehuchten and Nélis discovered what they believed to be of diagnostic value, changes in the **intervertebral** ganglia, consisting of proliferation of cells, probably from the capsules of the ganglia. The ganglionic cells themselves degenerate, inasmuch as the protoplasma with the nucleus in some cases totally disappears and is replaced by the newly formed small cells. These changes were found not only in the spinal, but also in the pneumogastric and Gasserian ganglions. Babès also described a so-called "rabic tubercle," consisting of an accumulation of embryonal cells around the vessels and nerve cells of the anterior cornua of the cord and in the medulla. The cells of the latter undergo degeneration, viz. chromatolysis with disappearance of the nuclei; the pericellular spaces are dilated and filled with the characteristic embryonal cells. I have seen the same "rabid tubercles" around the pyramidal cells of the cortex in several cases of rabies. In 1903 **Négri's bodies** became a new element in the diagnosis of rabies. They are found in the nerve cells and in their largest dendritic prolongations especially of the cornu ammonis but also of the cortex, pons, medulla and spinal ganglia.

These bodies are of 1 to 27 μ in size, round or oval; they are surrounded by a hyaline membrane. They are very resistant and may be pre-

served for many days when the tissue is preserved in glycerine. The method for bringing them out to the best advantage is as follows:

Impregnate the tissue in a solution with a sublimate base, especially a sublimate with acetic alcohol of Gilson or sublimate of von Lenhossék. Next step is to cover the piece with a slight layer of thick celloidine, dry in the air for a few minutes, place tissue in: alcohol absolute 50 c.c.+ chloroform 5 c.c. Avoid xylol for clearing, but use toluidin or cedar oil. It is admitted now that Négri's bodies are pathognomonic of rabies. As to their nature, Négri believes them to be parasites undergoing degeneration.

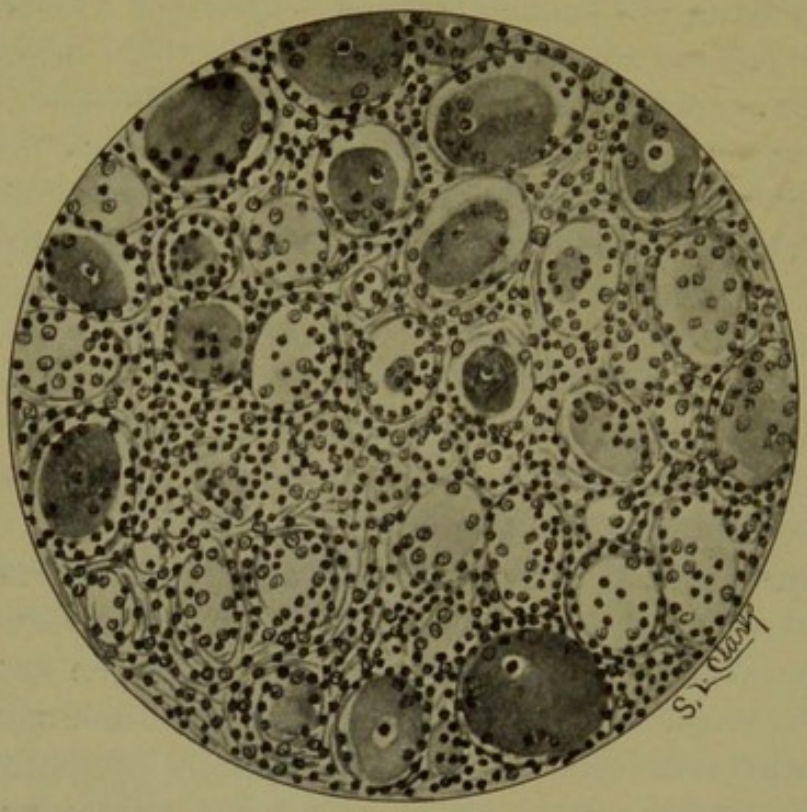


FIG. 169.—RABIES. SPINAL GANGLION. (*Original.*)

Symptoms.—After a period of incubation, which may last from a few days to two months (in children shorter than in adults), the infected part produced by the bite of the animal begins to itch and burn; sometimes there is pain, lancinating in character. Headache, insomnia and anorexia develop next. The patient becomes depressed and irritable. A state of **anxiety** appears and this is increased by a rigidity of the muscles of the throat and difficulty in swallowing. At the same time the temperature rises slightly, the pulse is accelerated.

Soon another phase of the disease makes its appearance. It is characterized by **restlessness,** cerebral **excitement** and **hyperæsthesia.** The least noise, a bright light, a slight breeze causes a **spasm.** The latter affects especially the muscles of the larynx and pharynx. As there is intense thirst, the patient makes great efforts to drink, but an attempt to

take **water** or the sight of water or even the suggestion of it produces a painful spasm of the pharynx and larynx (**hydrophobic spasm**). Dyspnœa is then pronounced. The **fear of water** (hydrophobia) is characteristic of the disease. The difficulty of swallowing makes the saliva dribble from the mouth. The temperature goes up to 101°–103°. At the same time a delirium with hallucinations and delusions develop. The convulsive attacks which were at first confined to the muscles of deglutition and respiration, now become generalized and may be so frequent that asphyxia occurs. They are brought on by the same excitants which produce the spasm of pharynx and larynx. During the convulsions patients make noises resembling the barking of a dog. The delirium is usually marked during the paroxysms; the patient may become violent, inflict injuries on himself or bite others and spit at them. This phase of the disease usually lasts one or three days. This may be followed by a **paralytic stage,** during which all the previous symptoms gradually abate and the patient becomes quiet. Gradually he sinks into unconsciousness and dies.

Paralytic Rabies.—In exceptional cases the convulsive phase is absent and there is only the paralytic phase. It is a very grave condition. It usually follows very extensive bites. Paralysis rapidly develops and assumes the type of acute ascending paralysis. Death follows from respiratory or cardiac paralysis.

Course, Duration, Prognosis.—The rapidity of development of the symptoms depends upon the part of the body bitten. Wounds on the face present the greatest danger. The prognosis is grave, except when antitoxic treatment is early instituted. Death usually occurs within two or five days.

Diagnosis.—In **tetanus** there is also a spasm of deglutition and of respiratory muscles, but the presence of trismus and absence of fear of water will decide the diagnosis.

Hysterical paroxysms may sometimes simulate rabies (pseudo-hydrophobia), but the special symptoms of hysteria will soon determine the disease.

Treatment.—As soon as the bite occurs, a ligature is placed above the wound and kept in place several hours. The latter should be enlarged and a thorough cauterization, followed by an antiseptic dressing, be done. It should be left open and free drainage given for several weeks.

As soon as possible the **antirabic serum** should be employed. Since **Pasteur** introduced this treatment, the mortality has greatly decreased.

The other symptoms of the disease are treated accordingly. Perfect quiet, sedatives (bromides, chloral, chloroform, morphia), rectal feeding, if not possible by the mouth, are practically all that can be done.

NERVOUS MANIFESTATIONS IN PELLAGRA

The nature of the disease is still unsettled. The old view with relation of the disease to ingestion of maïze is not held by all and considered as doubtful. Sambon's idea in regard to an infectious origin of the disease is viewed with favor. The endemic mode of propagation, its appearance at certain seasons, the failure of all prophylactic measures based on the theory of maïze, all speak in favor of infection. It is supposed that pellagra is an insect-borne disease.

The manifestations of pellagra concern the **gastro-intestinal tract, skin** and **the nervous system.**

The affection begins usually with gastro-intestinal and psychic disturbances. Gastritis and diarrhœa are frequent in the early stages. The patient is depressed, indifferent to surroundings and to his own appearance, or else may have maniacal outbursts. He is forgetful, commits errors of serious nature. Sudden lapses of memory resembling Petit Mal, vertigo are not infrequent. Exceptionally delusions and hallucinations are observed. Suicidal tendencies are not rare. Paræsthesias, headache, pain in back or in the extremities of tabetic nature, hypæsthesias or anæsthesias, tremor of the hands and tongue resembling that of multiple sclerosis, muscular spasm, Romberg's sign, increased patellar tendon reflexes, occasionally Babinski's sign—are symptoms which may all or only some of them be encountered in Pellagra.

Eye Symptoms.—Early forming cataracts are frequent. Retinitis and optic neuritis are not uncommon. The choroid is quite frequently involved.

The **skin** manifestations consist of symmetrical in shape and position erythemata resembling sunburn; they are seen on the exposed parts of the body, especially on the hands. Desquamation is the ultimate result. Changes in the postero-lateral tracts of the spinal cord have been found in some cases.

Treatment.—Mild cases require no treatment. In cases with severe gastro-intestinal disorders and asthenia, arsenic is indicated. Nutritious diet with rest are the chief measures to be relied upon. The mortality is quite high.

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