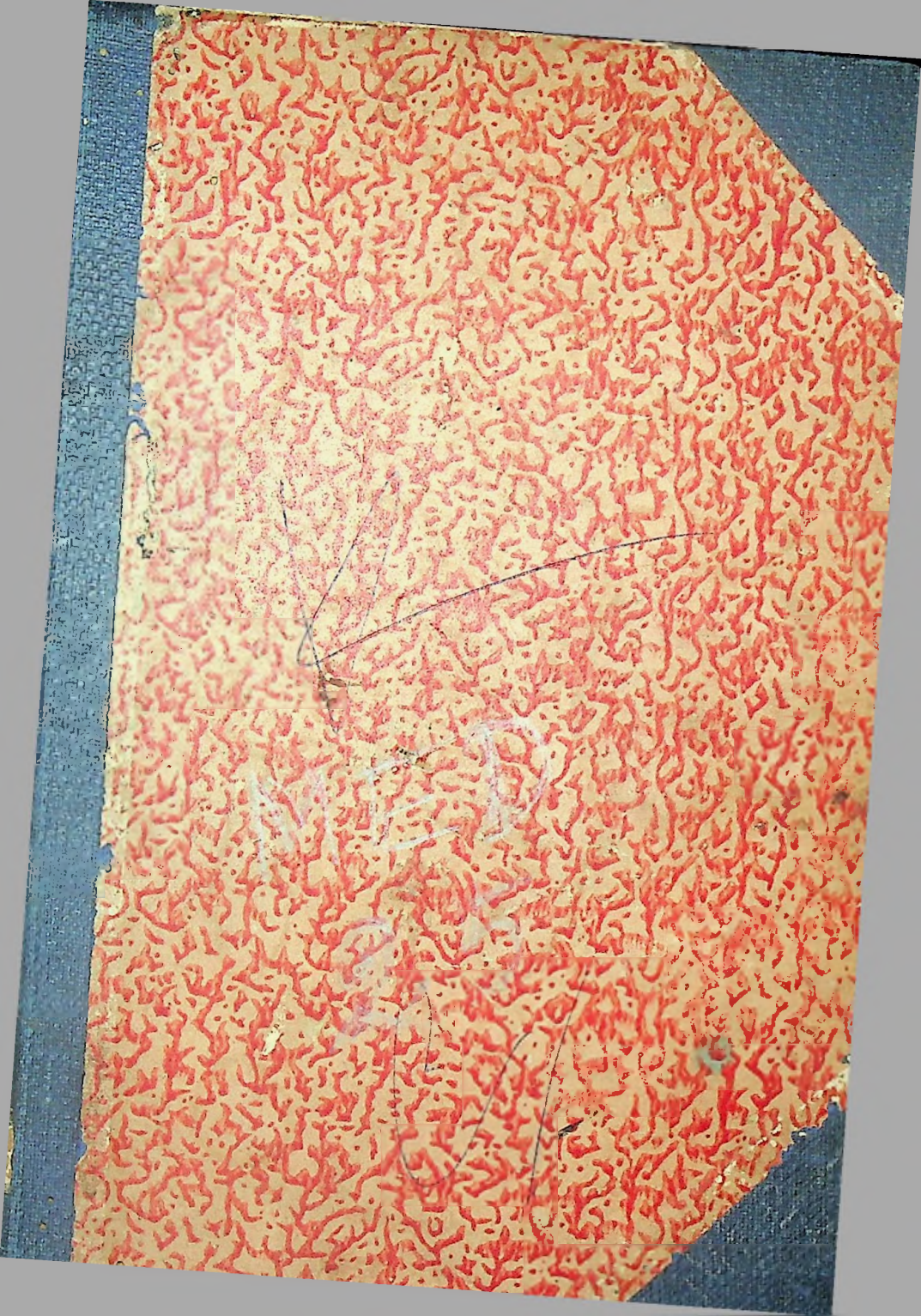

A MANUAL

OF

NERVOUS DISEASES

AND THEIR

Homeopathic Treatment

A COMPEND FOR

STUDENTS, COLLEGES AND PHYSICIANS

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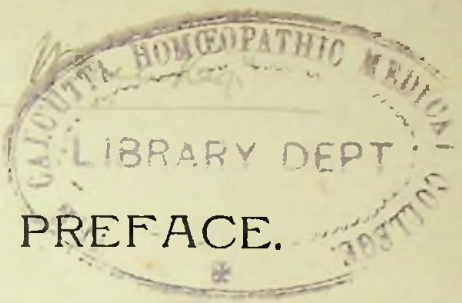

PREFACE.

In offering this little work to the profession it is the object of the author to furnish a treatise upon nervous diseases which will be concise and at the same time practical, both for the student and the busy practitioner. It is not intended as a substitute for the larger text-books on the subject, but to be merely an outline of the diseases described. To the student it will take the place of the quiz-compend, as it has been arranged according to the methods used in such books. To the practitioner it will be of use as a ready manual in which he can quickly determine the character of the case that he is looking up, while it at the same time outlines a method of treatment.

Though the time allotted to me by the publishers in which to prepare my manuscript has been brief, nevertheless I have endeavored to make the book as complete and accurate as possible. I have entirely avoided giving any theories in regard to the causation or treatment of the diseases under consideration, but have given merely such facts as are known to be thoroughly established. In the homeopathic treatment of the diseases I have mentioned only a few of the most prominent remedies, with their indications

for the special disease, and have not continued the list beyond the point of utility in a manual of this size.

In the preparation of this work I have drawn freely from many authors, mainly Gowers, Dana, Ranney, Bartlett, Hirt, Ross and Starr. For the homeopathic treatment I have consulted the works of Lilienthal, Hering, Farrington, Hughes and others. While thus referring to these authors I have yet followed my own general plan, which I have used during my years of teaching, both as to the arrangement and treatment of the subject.

It gives me pleasure to acknowledge the invaluable services of my wife, Dr. Eleanor F. Martin, who has most carefully arranged and corrected my manuscript, and has frequently suggested points which might possibly have been omitted by myself.

In conclusion I beg the indulgence of the readers to any errors which may be discovered, and trust that their criticisms may be as forbearing as the author's purpose in writing the book has been sincere.

March, 1896. The Author.

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A Manual of Nervous Diseases

PART I.

ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM.

DIVISIONS OF THE NERVOUS SYSTEM.

WHAT ARE THE TWO GREAT DIVISIONS OF THE NERVOUS SYSTEM?

The cerebro-spinal or the nervous system of animal life, and the sympathetic or nervous system of organic life.

OF WHAT DOES THE CEREBRO-SPINAL SYSTEM CONSIST?

(1). Those parts of the central nervous system contained in the cavities of the cranium and spinal column, viz., the brain and the spinal cord.

(2). The motor or efferent nerves, which convey nerve impulses from the centre to the muscles.

(3). The afferent or sensory nerves, which convey sensory impressions from the periphery of the body to the brain and spinal cord.

WHAT DO THE CEREBRO-SPINAL NERVES COMPRISE?

The cranial nerves, those which escape from the foramina of the cranium, and the spinal nerves or those which are given off from the spinal cord.

WHAT CONSTITUTES THE SYMPATHETIC NERVOUS SYSTEM?

(1). A continuous chain of nerve fibres and ganglionic enlargements extending from the head to the coccyx on both sides of the spinal column, which are in constant communication with the cerebro-spinal nerves.

(2). Three large gangliated plexuses situated in the thoracic (cardiac), abdominal (solar), and pelvic (hypogastric) cavities.

(3). Smaller ganglia situated in the visceræ.

(4). Numerous nerve fibres, some of which help to form plexuses which supply the coats of the principal bloodvessels and regulate their blood supply.

The intimate connection between the cerebro-spinal and sympathetic nerves enables the two systems to act in perfect harmony.

WHAT ARE VASO-MOTOR CENTRES?

Within the substance of the brain and spinal cord, along the course of the motor and sensory nerves, are special centres connected with the sympathetic nerve fibres. These are the so-called vaso-motor centres, which have to do with the contraction and dilatation of the bloodvessels — vaso-constrictors and vaso-dilators.

WHAT IS THE STRUCTURE OF A NERVE FIBRE?

There are two kinds of nerve fibres, grey and white.

A white fibre is made up of

(a.) A functional element, or central axis-cylinder which is surrounded by a medullary sheath or the white substance of Schwann, composed of myelin, a liquid, fatty material, supported by a fine network of horny substance called neurokeratin.

(b). A delicate membrane called the primitive sheath, or neurilemma, or sheath of Schwann, surrounding the white substance.

(c). Nuclei lying at intervals beneath the sheath, between it and the myelin.

The white substance is interrupted at regular distances by what are termed nodes of Ranvier, named from the discoverer. The end of each portion, or internode, is enclosed in a sheath through which the axis-cylinder runs. Each

internode may be conceived as a fat cell, consisting of membrane, nucleus, protoplasm and fatty matter, the cells being arranged end to end, and the axis-cylinder passing through them like a string through a series of tubular beads.

These nerve fibres are united into fasciculi by a delicate nucleated connective tissue, and these in turn are connected into large bundles and the whole is surrounded by a dense connective-tissue sheath forming the nerve proper.

The grey fibres, or non-medullated fibres, consist of an axis-cylinder, sheath and nuclei, but contain no myelin. The sympathetic nerves are made up of these fibres.

HOW DO NERVES TERMINATE?

Sensory nerves end peripherally in the so-called peripheral end-organs in the tissues, there being three varieties, viz.:

(a). The end-bulbs of Krause, found in the conjunctiva, the mucous membrane of the mouth and the cutis.

(b). The tactile corpuscles of Wagner, occurring in the papillæ of the skin of the fingers and toes: and

(c). The Pacinian corpuscles, which are found in the tissues of the fingers and toes.

Motor nerves are to be traced either into unstriped or striped muscular fibres. In the unstriped or involuntary muscles the nerves are derived from the sympathetic. Near their termination they divide into a number of branches which communicate and form an intimate plexus. From these plexuses are given off minute branches which divide and break up into ultimate fibrillæ, of which the nerve is composed.

Nerves supplying striped or voluntary muscles are derived from the cerebro-spinal nerves and are composed mainly of medullated nerve fibres. After entering the sheath of the muscle the nerve breaks up into fibres or bundles of fibres which form plexuses and gradually divide, a single nerve fibre, as a rule, entering a single muscular fibre. Within the muscular fibre the nerve terminates in a special expansion called motorial end plates.

THE BRAIN.

WHAT IS THE BRAIN?

The brain is that portion of the central nervous system contained within the cranial cavity. It is composed of gray and white matter, the gray being external, forming the cortex or rind, and the white matter forming the internal part.

WHAT IS THE WEIGHT OF THE BRAIN?

The average weight of the brain in an adult male is about forty-nine ounces, or a little over three pounds, while in a female it is about forty-four ounces. Its weight increases very rapidly from the time of birth up to the seventh

Figure 1.

A diagram designed to elucidate the chief component parts of the human brain. (Ranney.)

year, and then more slowly to between the sixteenth and twentieth years, and very slowly indeed between thirty and forty, when it usually ceases to grow. There have been instances where the brain, as in the case of Curvier, weighed a a little more than sixty-four ounces, and of Abercrombie, sixty-three ounces, and of a mulatto whose brain weighed sixty-eight and three-eighth ounces.

WHAT ARE THE PRINCIPAL DIVISIONS OF THE BRAIN?

(a). The cerebrum.
(b). The cerebellum.
(c). The medulla oblongata, and
(d). The pons Varolii.

THE MEMBRANES OF THE BRAIN.

NAME THE MEMBRANES OF THE BRAIN.

(a). The dura mater, or protecting membrane.
(b). The arachnoid, or lubricating membrane, and
(c). The pia mater, or nourishing membrane.

DESCRIBE THE DURA MATER.

It is a dense, white fibrous membrane. Its outer surface is rough and adheres closely to the inner surface of the skull, forming the internal periosteum. The inner surface is smooth. At the base of the skull it is very closely adherent, sends prolongations through the foramina, and becomes blended with the fibrous sheaths of the nerves and vessels which pass out of and into the cranial cavity. Its attachments to the periosteal ridges and the crista galli are particularly firm.

WHAT PROCESSES ARE FORMED BY THE DURA MATER?

(a). The falx cerebri.
(b). The falx cerbelli, and
(c). The tentorium cerebelli.

The two former processes prevent lateral oscillation of the cerebral and cerebellar hemispheres, while the tentorium forms the supporting structure of the posterior portions of the cerebrum and prevents it from pressing down upon the cerebellum.

WHAT ARE THE ATTACHMENTS OF THE FALX CEREBRI?

The falx cerebri is so called from its scythe-like form. Its upper margin is convex and is attached to the antero-posterior median line of the skull as far back as the internal occipital protuberance. It dips down between the cerebral hemispheres, being attached to the crista galli in front and to the tentorium cerebelli behind. Along its upper and

lower borders two re-duplicated layers of the dura assist in forming the superior and inferior longitudinal sinuses. At its attachment with the tentorium the straight sinus is formed.

WHAT ARE THE ATTACHMENTS OF THE FALX CEREBELLI?

The falx cerebelli separates the two lateral lobes of the cerebellum. Its base is attached above to the under part of the tentorium, its posterior margin to the lower division of the vertical crest on the inner surface of the occipital bone.

The occipital sinuses run along its sides.

WHAT ARE THE ATTACHMENTS OF THE TENTORIUM CEREBELLI?

The tentorium cerebelli is an arched process of the dura mater, elevated in the middle and inclining downward toward the circumference. It is attached behind by its convex border to the transverse ridges upon the inner surface of the occipital bone and encloses the lateral sinuses; in front it is attached to the superior margin of the petrous portion of the temporal bone, enclosing the superior petrosal sinus.

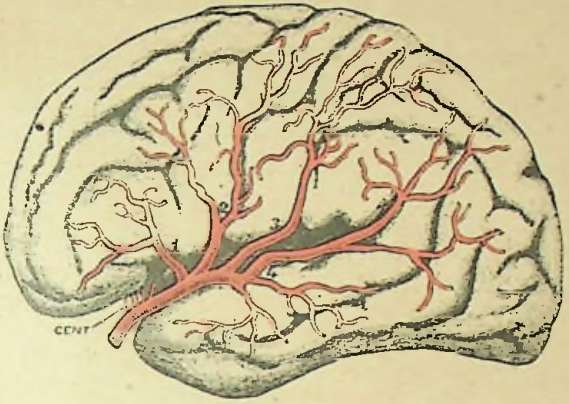
FROM WHENCE DOES THE DURA MATER RECEIVE ITS BLOOD SUPPLY?

The arteries of the dura mater are many, but there is one of special importance, the great or middle meningeal artery, a branch of the inferior maxillary which enters the skull through the foramen spinosum.

It passes upward over the outer surface of the dura mater in close connection with its two veins and divides into anterior and posterior branches, which are received by grooves in the inner table of the parietal bone.

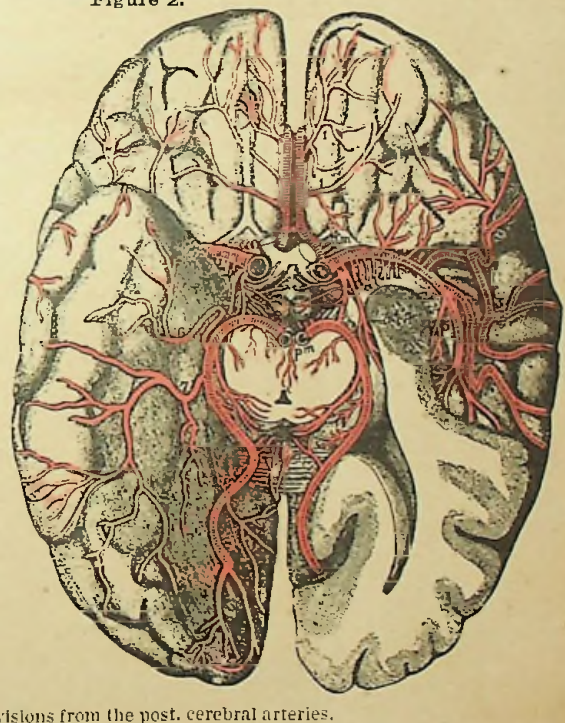
In almost every case of fracture of the vault of the skull attended with extravasation of blood it is one or the other of the branches of the middle meningeal artery that gives way.

Other arteries which supply the dura are the lesser meningeal and a small twig from the ascending pharyngeal. Anteriorly there are arteries from the ethmoid and internal



Cortical Distribution of the Middle Cerebral Artery. (Thane and Charcot.) The remainder of the convexity is supplied by the anterior cerebral (frontal and mesial) and the posterior cerebral. CENT. antero-lateral group of central arteries: 1, inf. ext. frontal; 2, ascending frontal; 3, ascending parietal; 4, parieto-temporal artery.

Figure 2.



The Arteries at the Base of the Brain. (After Thane and Duret, from Schafer.) The posterior cerebral are cut at their origin from the basilar. Central arteries (to the basal ganglia): *am*, antero-mesial group arising from the anterior cerebral; *al*, antero-lateral group (middle cerebral); *pm*, *pl* (on the optic thalamus), from the posterior cerebral; *ach*, *pch*, anterior and posterior choroidal arteries. Peripheral arteries: 1, 1, inferior internal frontal (ant. cerebr. art.); 2, inf. ext. frontal; 3, ascending frontal; 4, ascending parietal; 5, temporo-parietal (middle cerebr.); 6, 7, 8, ant. post. occipital divisions from the post. cerebral arteries.

Figure 3.

PLATE I.

carotid, and posteriorly branches from the occipital, ascending pharyngeal and vertebral arteries.

WHAT NERVES SUPPLY THE DURA MATER?

The recurrent branch of the fourth, some filaments from the Gasserian ganglion, the ophthalmic, the hypoglossal and the sympathetic.

DESCRIBE THE ARACHNOID MEMBRANE.

A thin, delicate membrane investing the brain beneath the smooth, inner surface of the dura mater. On account of its extreme thinness it has been named the arachnoid, from the Greek word denoting a spider's web. It is called the lubricating membrane because it throws out a serous fluid upon its surface, which allows of slight movement of the brain against the surface of the dura mater with very little friction and consequent irritation.

WHAT SPACES ARE FOUND BETWEEN THE MEMBRANES?

The sub-dural space, which contains a small quantity of fluid; the arachnoid covers the pia mater very closely, but it does not dip down into any of the sulci of the brain; upon the convex surface the arachnoid and pia mater can not be readily separated. But there are places, especially at the base of the brain, called the sub-arachnoid spaces, where the two membranes are separated to a greater or less degree.

WITH WHAT ARE THE SUB-ARACHNOID SPACES FILLED?

These spaces are filled with a fluid called cerebro-spinal, which acts as a cushion for the brain, thus preventing any serious effects from ordinary concussion.

The amount of fluid in these spaces is much more than in the sub-dural spaces, but the whole amount outside of the brain is about two ounces, it communicating with the fluid within the ventricles of the brain through the foramen of Magendie.

The brain is, therefore, supported by this fluid, which insinuates itself into all the inequalities of the surface and most effectually protects it.

In cases of fracture of the base of the skull involving

the petrous portion of the temporal bone the escape of this fluid through the ear is a diagnostic feature.

WHAT NERVES SUPPLY THE ARACHNOID?

The nerves of the arachnoid are filaments from the motor root of the fifth nerve, the seventh and the eleventh nerves.

DESCRIBE THE PIA MATER.

The true nourishing membrane of the brain, immediately investing it. It is extremely vascular, being composed of a minute network of bloodvessels held together by a very delicate areolar tissue. It covers the entire brain, dips down between the convolutions and sends prolongations into the interior, forming the velum interpositum and choroid plexus of the fourth ventricle.

On the surface of the cerebrum it is very vascular and gives off from its under surface a large number of minute vessels which extend perpendicularly into the cerebral substance. Upon the surface of the cerebellum it is thinner and not so vascular, and on the pons Varolii and medulla it is more fibrous and less vascular than elsewhere.

WHAT VESSELS SUPPLY THE PIA MATER?

Its derives its blood from the internal carotid and vertebral arteries.

WHAT NERVES SUPPLY THE PIA MATER?

The third, fifth, sixth, seventh, ninth, tenth, eleventh and sympathetic nerves, which chiefly accompany the bloodvessels. It is important that special note be taken of the nerve-supply of the membranes of the brain, for many headaches are due to meningeal irritation, and the location and character of the pain will often give a clew to the diagnosis.

THE CEREBRUM.

DESCRIBE THE CEREBRUM.

It is the larger part of the brain, filling the whole upper portion of the cranial cavity and overlying all other por-

tions of the brain. It rests in the anterior and middle fossæ of the base of the skull, and is separated posteriorly from the cerebellum by the tentorium cerebelli. It is divided into two lateral hemispheres by the great longitudinal fissure, which extends throughout its entire length. The hemispheres are unequal in size, the left being usually the larger. This has been attributed to the more direct blood supply to the brain on that side, the left vertebral and carotid arteries having independent origins from the arch of the aorta. The hemispheres are joined together by a broad transverse commissure of white matter called the corpus callosum. Each hemisphere presents a convex outer surface which corresponds with the inner surface of the cranium, an inner surface which is flat and in contact with the opposite hemisphere, and an under surface which is irregular, as it rests upon the bones at the base of the skull.

WHAT IS TO BE SEEN UPON THE CONVEX SURFACE OF THE HEMISPHERES?

The surface of each hemisphere presents a large number of convoluted eminences, the convolutions or gyri, which are separated from each other by depressions, called fissures or sulci, of various depths. The surfaces of these convulutions are composed of gray matter, the interior being of white matter. They are formed to increase the amount of gray matter within the cranial cavity without occupying additional space, so that its actual surface is nearly six times what it would be if it were merely a smooth envelope. Usually the larger the number of convolutions, and, consequently, the larger the extent of gray matter, the greater the intellectual capacity; but this is not always so, as in some cases the quality of the gray matter is to be considered more than the quantity. The sulci are generally about an inch in depth. They vary in different brains and in different parts of the same brain. The hemispheres are divided into lobes and lobules by deep fissures.

DESCRIBE THE FISSURES OF THE CEREBRUM.

(a). Fissure of Sylvius.—This fissure begins at the base of the brain, at the posterior boundary of the anterior

fossa of the skull, and divides into two branches, one passing upward toward the longitudinal fissure, called the ascending limb, and the other, the longer one, called the horizontal limb, running horizontally backward, usually terminating in the parietal lobe in a bifid extremity. The branches of the fissure of Sylvius originate from the development of the hemisphere around the central lobe, or insula, which is usually covered in. The portion of the hemisphere overlapping the central lobe is called the operculum, from its lid-like character. The fissure of Sylvius is the most conspicuous of the cerebral fissures and is easily recognizable. It is of great surgical and medical importance, because it contains the middle cerebral artery, which is particularly liable to obstruction from an embolus.

(b). Fissure of Rolando.—The fissure of Rolando begins near the longitudinal fissure on the upper surface of the brain, a little posterior to the middle, and runs obliquely over the convex surface of the hemisphere at an angle of about sixty-seven and one-half degrees, downward and forward almost to the junction of the two limbs of the fissure of Sylvius. This fissure is of special importance because it is situated in the middle of the motor area of the brain and constitutes, with the Sylvian fissure, the principal landmark used in cerebral localization.

(c). Parieto-Occipital Fissure.—The parieto-occipital fissure is situated partly upon the outer hemisphere, where it is called the external parieto-occipital fissure to distinguish it from that portion which is seen upon the inner surface and called the internal parieto-occipital fissure. It begins at the calcarine fissure, near the corpus callosum, and ascends vertically, ending on the external surface about an inch below the longitudinal fissure.

(d). Calloso-Marginal Fissure.—This fissure is above the gyrus fornicatus on the inner surface of the hemisphere.

(e). Calcarine Fissure.—The calcarine fissure begins near the posterior border of the hemisphere, passes forward and ends under the corpus callosum, penetrating into the posterior horn of the lateral ventricle. It joins the parieto-occipital fissure midway.

(f). Hippocampal Fissure.—The hippocampal fissure is seen upon the inner surface of the cerebral hemisphere and

'indicates the seat of a convolution known as the "hippocampus major."

(g). Transverse Fissure.—The transverse fissure separates the cerebrum from the cerebellum. It is continuous with the lateral and third ventricles and admits the pia mater into the interior of the brain to form the velum interpositum.

(h). Parietal Fissure.—The parietal fissure is sometimes connected with the parieto-occipital, and sometimes with the horizontal portion of the fissure of Sylvius; but

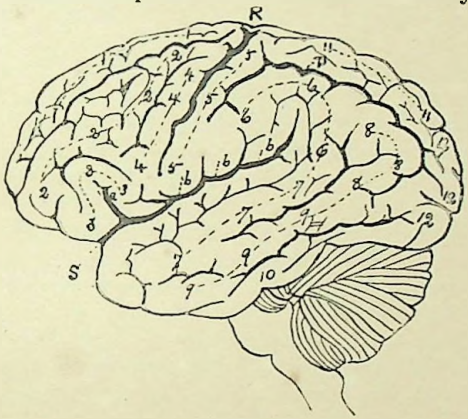


Figure 4.

A diagram showing the convex surface of the cerebrum. (Dalton.)

S, fissure of Sylvius, with its two branches, a and b, b, b. R, fissure of Rolando. P, Parieto-occipital fissure. 1, 1, 1, the first or superior frontal convolution. 2, 2, 2, 2, the second or middle frontal convolution. 3, 3, 3, the third frontal convolution, curving around the ascending limb of the fissure of Sylvius (*motor center of speech*). 4, 4, 4, ascending frontal (anterior central) convolution. 5, 5, 5, ascending parietal (posterior central) convolution. 6, 6, 6, supra-marginal convolution (parietal lobule), which is continuous with 7, 7, 7, the first or superior temporal convolution. 8, 8, 8, the angular convolution (or gyrus), which becomes continuous with 9, 9, 9, the middle temporal convolution. 10, the third or inferior temporal convolution. 11, 11, the superior parietal convolution. 12, 12, 12, the superior, middle and inferior occipital convolutions.

generally it begins between the latter and the fissure of Rolando and curves backwards parallel with the superior border of the cerebrum. (Fig. 4.)

NAME AND LOCATE THE LOBES AND PRINCIPAL CONVOLUTIONS OF THE CEREBRUM.

Frontal Lobe.—The frontal lobe is that portion of the cerebrum situated in front of the fissure of Rolando, and

above the fissure of Sylvius. It is divided into four convolutions or gyri.

The ascending frontal convolution or gyrus is situated just anterior to the fissure of Rolando. It is sometimes called the precentral gyrus.

The superior frontal convolution joins the ascending frontal and passes forward across the frontal lobe, horizontal to the longitudinal fissure. It borders on the anterior part of the corpus callosum internally and extends to the under surface, where it forms the olfactory lobe.

The middle frontal convolution passes parallel to the superior.

The inferior frontal convolution is below the preceding and in relation to the fissure of Sylvius. The left inferior frontal convolution is often called Broca's convolution or the speech centre, from the localization of the movements of the lips and tongue in articulation, in its posterior portion.

Parietal Lobe.—The parietal lobe is situated posterior to the fissure of Rolando and extends back to the parieto-occipital fissure. It is bounded below by the horizontal limb of the fissure of Sylvius which separates it from the temporo-sphenoidal lobe. It has also four convolutions.

The ascending parietal convolution or post-central is posterior to the fissure of Rolando and usually extends under it joining the ascending frontal. The two convolutions thus surround the fissure of Rolando and also form what is called the opercular lobe.

The superior and inferior parietal convolutions are separated by the parietal fissure and are usually continuous with the occipital lobes by bridges of gray matter called annectant convolutions.

The superior marginal convolution blends with the lower part of the posterior central gyrus and arches over the end of the horizontal branch of the fissure of Sylvius to join the superior temporo-sphenoidal convolution.

The angular convolution is behind the supramarginal and parallel to the fissure of Sylvius. It usually joins the temporo-sphenoidal and occipital lobes by annectant bands. Upon the median surface the superior parietal convolution joins with the upper extremity of the posterior central convolution to form the precuneus, or quadrate lobe.

Occipital Lobe.—The occipital lobe presents three principal convolutions, which are usually badly defined, the superior, middle and inferior. These are subdivided by the occipital fissure and are continuous with the convolutions of the parietal and temporal lobes. Upon the median surface the superior occipital convolution, somewhat triangular in shape, forms the cuneus lobule, placed between the parieto-occipital and calcarine fissures.

Temporo-Sphenoidal Lobe.—The temporo-sphenoidal lobe presents three well-marked convolutions which run in an antero-posterior direction.

The superior lies below the horizontal limb of the Sylvian fissure and is continuous behind with the parietal lobe.

The middle becomes continuous with the angular gyrus and is connected with the third occipital convolution.

The inferior is seen on the under surface of the cerebrum and is also connected with the third occipital convolution.

Limbic Lobe.—The limbic lobe when described separately includes the gyrus fornicatus, or convolution of the corpus callosum, which begins just in front of the anterior perforated space at the base of the brain, ascends in front of the genu of the corpus callosum, and runs backward along the upper surface of this body to its posterior extremity, where it passes downward and forward under the name of the gyrus hippocampi to terminate in the uncinati gyrus, nearly opposite to where it began.

NAME AND LOCATE THE LOBULES OF THE CEREBRUM.

(a). The island of Reil or lobulus centralis lies deeply situated in the commencement of the fissure of Sylvius. It can only be seen by the separation of the lips of that fissure. It is a triangular eminence and consists of from four to six small convolutions, gyri operti, arranged side by side, and appearing when exposed very much like the finger of the hand when closed upon the palm. It covers the lenticular nucleus of the corpus striatum. Behind the central lobe there are usually several small convolutions, known as the temporo-parietal or retro-insular convolutions.

(b). The para-central lobule is seen on the inner surface of the cerebrum, anterior to the calloso-marginal fissure. (Fig. 5.)

(c). The lobulus quadratus lies just posterior to the para-central lobule and extends back to the parieto-occipital fissure.

(d). The lobule-cuneus is a wedge-shaped body which lies between the parieto-occipital and calcarine fissures, on the inner surface of the cerebrum.

WHAT IS THE MICROSCOPICAL ANATOMY OF THE CORTEX?

The cortex of the cerebrum is composed of nerve cells, a net-work of nerve fibres, processes and neuroglia tissue,

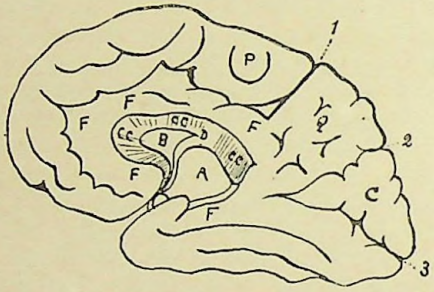


Figure 5.

A diagram showing a longitudinal median section of the cerebrum. (Dalton). 1, calloso-marginal fissure. 2, parieto-occipital fissure. 3, calcarine fissure. A, third ventricle. B, fifth ventricle. D, anterior crura of fornix. C, cuneus (occipital lobule). Q, precuneus (lobulus quadratus). P, para-central lobe. C. C, corpus callosum. F, gyrus fornicatus.

the latter being an intercellular substance which serves to cement the cells and to maintain a fixed position for them, as well as to furnish passage for the vessels of nutrition of the cells. These elements are formed into layers which are never strictly defined, as the elements of one layer interpenetrate a little to the regions of other layers.

Allowing for this we recognize the following cortical layers in the Rolandic area:

(a). The first or molecular layer, containing but few cells. Its outermost layer is formed of neuroglia cells.

(b). The second layer of cells, sometimes fusiform, sometimes pyriform, and again triangular or polygonal, interspersed with a large number of small pyramidal cells.

(c). The third layer of pyramidal cells, with long and short apical processes. The pyramidal cells with short

apical processes are scattered about throughout all the cortex below the molecular layer, and show no special gradation in size. None of their apical processes pass into the molecular layer, their terminations being always deeper. They are a different system of cells from the pyramidal cells with long apical processes, for the latter always reach up to and end in the molecular layer. They also increase in size of cell body from above downward.

(d). The fourth layer of polymorphic elements, in which the following are the main cell types: (1). Pyramidal cells with short apical processes. (2). Granule cells. Both of these may interpenetrate a little distance into the superadjacent layer. (3). Fusiform cells proper, with ascending axis-cylinder processes. (4). Fusiform cells with descending axis-cylinder processes passing into the white substance. (5). Asymmetrical or oblique pyramidal cells intermediate between the strictly fusiform and the pyramidal. (6). Inverted pyramidal cells. (7). Cells with short branching axis-cylinders, and cell body of polygonal shape.

Various types of cortex are described, depending upon the different degree of development of the cell layers and upon the fibre arrangements. The common or motor type has four layers. The large pyramidal cells are numerous and are arranged in clusters. The sensory type has at least five layers, the fifth a subdivision of the fourth; and here the large pyramidal cells are few and isolated. The basil processes of the pyramidal cells are continued as axis-cylinders. Some pass down into the white matter, and others turn up and enter the fibre system of the cortex.

To sum up, then, there are, according to Andriezen, four layers fundamental to the cortex in the Rolandic area: (1). Molecular. (2). Ambiguous. (3). Long pyramidal. (4). Polymorphic. Of the various cells entering into the constitution of these there are eight types: (1). The pyramidal cell with long apical process reaching to the molecular layer. (2). The pyramidal cell with short apical process not reaching to the molecular layer. (3). The ambiguous cell, whose sub-types may be asymmetrical, bicornate, globose, pisiform, etc. (4). Granule cell. (5). The fusiform or triangular cell, with ascending axis-cylinder process. (6). The fusiform cell, with descending axis-cylinder process.

(7). The oblique and inverted pyramidal cell, with descending axis-cylinder. (8). The polygonal cell, with short branching axis-cylinder, the sensitive cell of Golgi.

These cells are not only distinguished by their morphology but by their anatomico-physiological connections, as well. Some of them receive nervous impulses from other cells and from terminal nerve fibres coming in from the

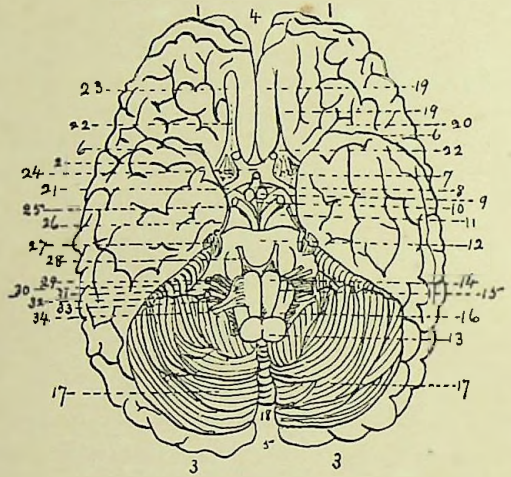


Figure 6.

The under surface of the brain. (Hirschfeld).

1, 1, anterior lobe of the cerebrum. 2, sphenoidal portion of the posterior lobe. 4, anterior extremity of the median fissure. 5, posterior extremity of the same. 6, 6, fissure of Sylvius. 7, anterior perforated space. 8, tuber cinerum and pituitary body. 9, corpora albicantia. 10, posterior perforated space. 11, crura cerebri. 12, pons Varolii. 14, medulla oblongata. 14, anterior pyramids. 15, ollvary body. 16, restiform body (only partially visible). 17, 17, hemispheres of the cerebellum. 18, fissure separating these hemispheres. 19, 19, first and second convolutions of the frontal lobe with the intervening sulcus. 20, external convolutions of the frontal lobe. 21, optic tract. 22, olfactory nerve. 22, section of olfactory nerve, showing its triangular prismatic shape; the trunk has been raised to show the sulcus in which it is lodged. 23, ganglion of the olfactory nerve. 24, optic chiasm. 25, motor oculi. 26, patheticus. 27, trigeminus. 28, abducens. 29, facial. 30, auditory nerve and nerve of Wrisberg. 31, glosso-pharyngeal. 32, pneumogastric. 33, spinal accessory. 34, hypo-glossal.

white matter. Others associate and co-ordinate these impulses, and still others discharge them. The processes of these cells are interwoven with each other and with white fibres, forming a close network over the whole brain. An enormous number of fine fibres are given off by the cells. Some connect neighboring parts, others distant parts, and

some pass to lower levels. There would seem to be three kinds of fibres—afferent, associative and efferent.

It will thus be seen that the cerebral cortex contains layers of nerve cells, into which nerve fibres penetrate.

The cortex has been likened to the cells of a battery, which generate and store electricity and discharge it under proper conditions.

Nerve-force is the energy which the cortical cell generates and gives off by means of the processes and nerve fibres connected with it.

The cortex is from two to four millimetres in thickness, and spreads over the whole surface of the cerebrum. It dips down into the convolutions and everywhere covers the white matter. The whole functional activity of the brain is centered in the cortex; so it can be readily seen of what great importance it is to the human economy.

WHAT POINTS ARE IN VIEW ON THE UNDER SURFACE OF THE CEREBRUM?

(a). From before backward, the anterior portion of the longitudinal fissure, which partially separates the two hemispheres. (Fig. 6).

(b). The corpus callosum, or the great transverse commissure, which is a thick stratum of transverse fibres exposed at the bottom of the longitudinal fissure. It connects the two hemispheres of the brain and forms the roof of a space in the interior of each hemisphere, called the lateral hemisphere. It is four inches in length and extends within an inch and a half of the anterior and within two inches and a half of the posterior part of the brain. Anteriorly it curves downward to reach the base of the brain, forming the "genu," and posteriorly it dips down to form the splenium. The fibres of the callosum may be traced to the white substance of the cerebral hemispheres, the gyrus fornicatus, the fornix, the occipital lobe and the temporo-sphenoidal lobe. The fibres are both longitudinal and transverse, and serve to unite component parts of the cerebral hemispheres. The transverse fibres probably unite homologous parts of each hemisphere.

(c). The lamina cinerea, a thin layer of gray tissue extending from the corpus callosum to the optic commissure.

(d). The fissure of Sylvius, between the frontal and middle lobes.

(e). The anterior perforated space or vallecula, through which the vessels that supply the corpus striatum pass.

(f). The olfactory bulbs and the olfactory tracts, which lie in a straight furrow on the orbital surface of the lobe and give origin to the olfactory nerves.

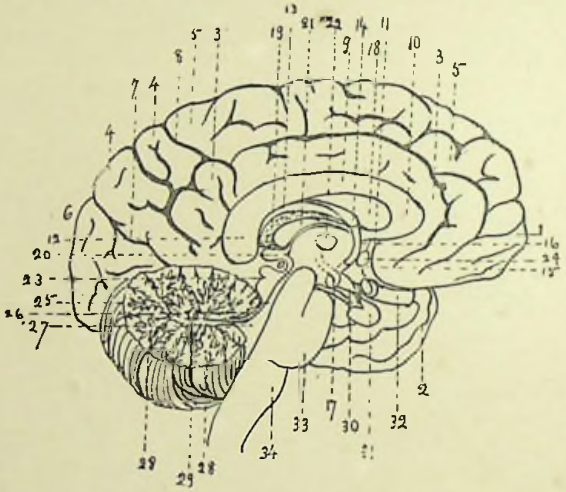


Figure 7.

1, frontal lobe. 2, sphenoidal lobe. 3, 3, convolution of the corpus callosum. 4, 4, convolutions of the parietal lobe of the internal surface. 5, 5, convolutions of the frontal lobe. 6, convolutions of the occipital lobe. 7, sulcus separating the parietal from the occipital lobe. 8, sulcus separating the frontal from the parietal lobe. 9, section of the corpus callosum. 10, genu of the corpus callosum. 11, rostrum of the corpus callosum. 12, posterior extremity of the corpus callosum. 13, fornix. 14, section of the fornix. 15, left anterior crus of the fornix, passing into the internal wall of the optic thalamus, to reach the corresponding corpus albicans—course indicated by a dotted line. 16, foramen of Monro. 17, corpus albicans, in which the anterior crus of the fornix bends upon itself, in the form of a figure of eight, to be lost in the substance of the optic thalamus. 18, septum lucidum. 19, section of the choroid plexus. 20, pineal gland. 21, left superior peduncle of the same. 22, section of the gray commissure of the third ventricle. 23, tuberula quadrigemina, above which are seen the pineal gland with its inferior peduncle and the posterior commissure. 24, section of the anterior commissure. 25, aqueduct of Sylvius. 26, section of the valve of Vieussens. 27, fourth ventricle. 28, 28, section of the middle lobe of the cerebellum. 29, arbor vitæ. 30, corpus cinereum. 31, pituitary body. 32, optic nerve. 33, pons Varolii. 34, medulla oblongata.

(g). The optic commissure, formed by the union of the optic tracts.

(h). The tuber cinereum, the gray prominence behind the optic commissure, which is the floor of the third ventricle,

from which projects a red-colored conical tube, the infundibulum, to which is attached the pituitary body (a small, reddish-gray vascular mass consisting of two lobes), which is lodged in the sella turcica of the sphenoid bone.

(i). The corpora albicantia, two white rounded bodies behind the tuber cinereum, formed by the bulbs of the fornix, a commissure situated beneath the corpus callosum, but continuous with it posteriorly. Each lateral half of the fornix presents an anterior pillar or crus, whose fibers pass downward and then after twisting in a figure of eight manner pass upward to end in the optic thalami, and a posterior pillar, which enters the middle horn of the lateral ventricle. (Fig. 7).

(j). The posterior perforated space, a gray depression posterior to the corpora albicantia, perforated by vessels which supply the optic thalami.

(k). The crura cerebri or cerebral peduncles, two thick cylindrical bundles of white matter which emerge from the anterior border of the pons and diverge as they pass forward and outward to enter the under part of each hemisphere. Each crus consists of a superficial and deep layer of longitudinal white fibres separated from each other by the substantia nigra or locus nigra, a mass of gray matter containing small multipolar ganglion cells. The superficial layer is called the crusta or basis cruris, and conducts motor fibres, the deep layer or tegmentum cruris conducting sensory fibres.

THE BASAL GANGLIA.

WHAT ARE THE BASAL GANGLIA?

Two nodal masses of gray matter situated within the substance of each cerebral hemisphere and resting nearly upon the floor of the cerebrum.

The anterior mass is called the corpus striatum, from the striated appearance of a section made through its substance.

The posterior mass is called the optic thalamus, from its supposed association with vision.

INTO WHAT IS THE CORPUS STRIATUM DIVIDED?

Each corpus striatum is divided by the fibres of the so-called internal capsule into two distinct portions, one of

which projects into the lateral ventricle while the other does not.

The first portion is known as the intra-ventricular portion, or the caudate nucleus, an ovoid-pyriform body, its base toward the frontal lobe, and a "tail-like" portion which passes up over the optic thalamus which lies just behind it, investing it like a surcingle.

The other portion is shaped somewhat like a lens and lies buried within the substance of the hemisphere. It is called the lenticular nucleus, or extra-ventricular portion.

WHAT FIBRES COMPOSE THE CORPUS STRIATUM?

(1). Fibres which pass to it from the cortex (afferent).

(2). Fibres which pass through it from the frontal and parietal cortex.

(3). Fibres which originate in it (efferent), and

(4). Fibres connecting its different parts.

The afferent fibres are in five groups: (1). Those which spring from the entire arch of the cerebral hemisphere, corona radiata. (2). Fibres springing from the temporal lobe to the most anterior part of the caudate nucleus, stria cornea. (3). Fibres which arise from the olfactory lobe. (4). Fibres from the septum lucidum; and (5). Fibres from the cerebellum.

The efferent set comprise those fasciculi which help to form the cerebral peduncles and which are dispersed after having passed through the pons Varolli, chiefly in the different segments of the spinal cord.

WHAT ARE THE FUNCTIONS OF THE CORPUS STRIATUM?

It is a ganglion in which cerebral, cerebellar and spinal activities are brought into intimate communication; it is a halting-place for voluntary motor impulses emitted from the cerebral cortex, and serves as a modifier of all motor acts.

DESCRIBE THE OPTIC THALAMI.

These ganglia are situated posterior and interior to the corpora striata. They are continuous with each other by means of the middle commissure. The upper surface forms part of the walls of the lateral ventricles; the mesial sur-

face forms the lateral wall of the third ventricle. They are oval-shaped masses of gray substance, covered superficially by a thin layer of white substance.

Upon the anterior part is a prominence called the anterior tubercle, and at the posterior and inner surface is the posterior tubercle, or pulvinar.

Below and external to the pulvinar and continuous with the gray matter of the optic thalamus is the outer geniculate body, and below that the inner geniculate body. The optic

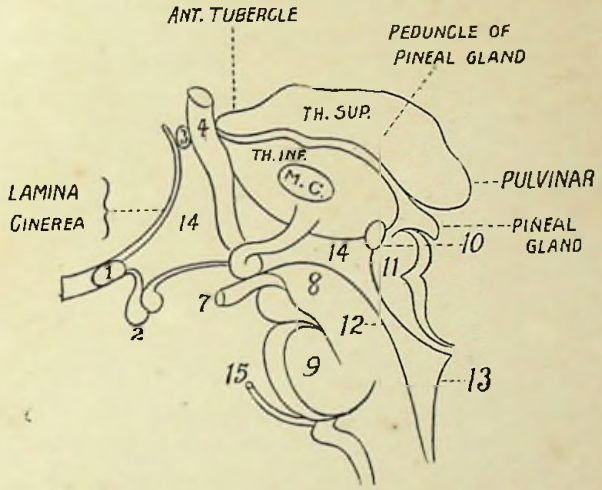


Figure 8.

The inner surface of the optic thalamus, the third ventricle, and neighboring parts. (Ranney).

Th. sup., superior part of thalamus. *Th. inf.*, inferior part of the same. *m. c.*, middle commissure. 1, section of optic commissure. 2, infundibulum and pituitary body. 3, anterior commissure of third ventricle. 4, anterior crus of fornix. 5, corpora albicantia candicans. 6, anterior crus of fornix. 7, the third nerve. 8, crus cerebri. 9, pons Varolii. 10, posterior commissure. 11, corpora quadrigemina. 12, aqueduct of Sylvius. 13, fourth ventricle. 14, third ventricle.

tracts wind around the posterior and outer edge of the thalamus.

The external surface lies in contact with the internal capsule of the cerebrum, and along this surface radiating fibres pass out and join the fibres of the internal capsule, to be distributed to the cerebral cortex. Fibres are also sent to the optic tract and to the tegmentum or cruris.

WHAT GANGLIA ARE FOUND IN THE OPTIC THALAMUS?

Four isolated ganglia are demonstrated in the thalamus.

The first, or anterior, has to do with the sense of smell and is called the olfactory centre.

The second, or middle, is the optic centre and receives fibres from the optic tracts.

The third, or median, ganglion is called the sensory centre, as it presides over sensation.

The fourth, or posterior, is the acoustic centre. (Fig. 8).

WHAT IS THE INTERNAL CAPSULE?

It is a tract of fibres which separates the lenticular nucleus and the thalamus, forming a capsule to the former.

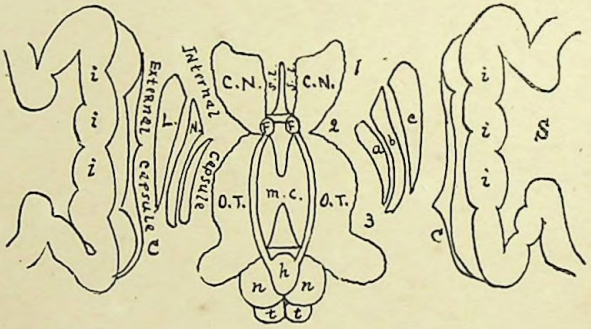


Figure 9.

Showing the relation of the internal capsule of the cerebrum to adjacent structure; viewed from above. (Ranney).

The section of the brain has been made horizontally in a plane to intersect the basal ganglia. C. N. caudate nucleus of the corpus striatum. L. N. lenticular nucleus of the same with its three parts (*a, b, c*). O. T. optic thalamus. S. fossa of Sylvius. c. claustrum. E. C. external capsule of cerebrum. *i, i, i*. convolutions of the Island of Reil. *a, b, c*, the inner, middle and external member of the lenticular nucleus. 1, anterior limit of the internal capsule. 2. "knee" or bend of the same. 3. posterior limit of the same. 1-2. "caudo-lenticular" portion of the same. F. crura of fornix, the fifth ventricle lying in front, and the third ventricle behind it. *s, l*. septum lucidum, showing its two layers with the fifth ventricle between them. *m. c.* middle commissure of the thalamus. *p*. pineal gland and its peduncles. *n*. nates cerebri. *t*. testes cerebri.

WHAT DOES IT CONTAIN?

(1). Pyramidal fibres which control voluntary movements of the limbs. (2). A sensory tract, which conveys sensations from the surface of the body to the cortical cells. (3). The speech tract. (4). Face fibres—motor. (5). Special sense

fibres — sight, smell, hearing, taste and touch. (6). The hypoglossal tract.

There is an angle in the formation of the internal capsule, at about its centre, called the knee. That portion of the internal capsule anterior to the knee, between the lenticular and caudate nuclei, is called the caudo-lenticular portion; that lying posterior to the knee is called the thalamo-lenticular portion, and it is this latter portion through which the bundles of fibres just mentioned passes. These fibres in the anterior portion are not yet thoroughly understood. A similar tract of fibres to the internal capsule, called the external capsule, separates the lenticular nucleus from the claustrum. The internal capsule seems to be a continuation upward into the cerebral hemispheres of both the motor and sensory portions of the crus, where its fibres diverge and form the greater part of the "corona radiata" which pass to the convolutions. It has no structural relation to the basal ganglia although it passes through them. (Fig. 9).

WHAT ARE THE CORPORA QUADRIGEMINA?

Four rounded bodies, mainly composed of gray matter, arranged in pairs, two in front and two behind, situated immediately behind the third ventricle and above the aqueduct of Sylvius. The anterior pair are the larger, are of gray color and are called nates: the posterior pair, lighter in color, are called the testes. They are connected on each side with the optic thalamus and optic tracts by two white bands termed the brachia, those connecting the nates with the thalamus being called the anterior brachia, while those connecting the testes with the thalamus are called the posterior brachia. They are also connected with the cerebellum by means of a large white cord on either side called the *processus e cerebelli ad testes*, or superior peduncles of the cerebellum.

WHAT ARE THE FUNCTIONS OF THE CORPORA QUADRIGEMINA?

It is believed there is a centre within the nates which controls the accommodation of vision for near objects, as well as the coördination of all ocular movements. The connection of these bodies with the optic thalami and the optic tracts would lead us to suppose that this might be true. It

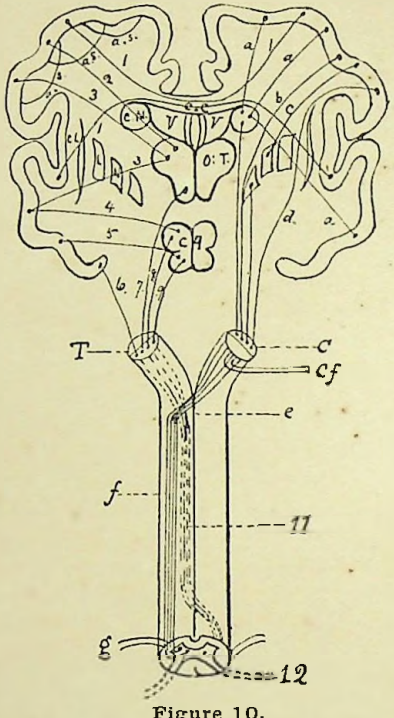


Figure 10.

A diagram showing the general arrangement of the fibres of the cerebro-spinal system. (Ramsey, modified from Landois).

The shaded portions represent the collections of gray matter. On the left side of the diagram the sensory fibres of the crus are traced upward from the spinal cord to different portions of the cerebrum; on the right side the motor fibres are similarly represented. Numerals are used in designating the sensory and commissural fibres; the motor fibres are lettered in small type. The cortical layer is shown at the periphery of the cerebral section, with commissural fibres (1) connecting homologous regions with the hemispheres, and associating fibres (a, a). connecting different convolutions of each hemisphere. C. N. caudate nucleus of the corpus striatum. L. N. lenticular nucleus of the same. O. T. optic thalamus of each hemisphere, united to its fellow in the median line. *c*, *q*. corpora quadrigemina. *c*, *l*. claustrum, lying to the right of the letters. *c*, *c*. corpus callosum, with its commissural fibres. S, fissure of Sylvius. *V*. lateral ventricle, the fifth ventricle being shown between the two layers of the septum lucidum. *C*. the motor tract of the crus cerebri (basis cruris..crusta). *T*. the sensatory tract of the crus cerebri (tegmentum cruris). *Cf*. the cerebellar fasciculus, which is turned to the right for perspicuity, but which in reality decussates. *e*. the point of decussation of the motor fibres of the spinal cord. *f*. the course of the motor fibres of the spinal cord below the medulla, showing their connection with the cells of the anterior horns of the gray matter, and their continuation into the anterior roots of the spinal nerve (*g*). *a*. fibres which radiate through the caudate nucleus. *b*. fibres of the "internal capsule." *c*. fibres which radiate through the lenticular nucleus. *d*. fibres of the "external capsule." 2, 3, 4, 5, 6, 7, 8, 9, sensatory fibres radiating from the tegmentum cruris to the cortex by means of various nodal masses of gray matter. 11, course of the sensatory fibres of the spinal cord (shown by dotted lines), intimately connected with the posterior root of the spinal nerve (12), and decussating at or near the point of entrance into the spinal cord.

also undoubtedly intensifies the inhibitory or controlling influence of the brain upon the reflex actions of the spinal cord.

WHAT AND WHERE IS THE PINEAL GLAND?

It is shaped like a fir-cone, *pinus*, hence its name. It is a small reddish-gray body, placed immediately behind the posterior commissure and between the nates, upon which it rests. It is joined to the cerebrum by two peduncles called the superior and inferior peduncles of the pineal gland. It is regarded as one of the ganglia of origin of the tegmentum cruris, since it is connected with the crus by means of the posterior commissure of the third ventricle.

THE CENTRUM OVALE.

OF WHAT DOES THE INTERIOR OF THE CEREBRUM CONSIST?

Of an oval-shaped centre of white substance surrounded on all sides by a narrow, convoluted margin of gray matter which presents an equal thickness in nearly every part. This white-central mass is called the centrum ovale, and is formed of nerve fibres which connect various parts of the brain with each other.

HOW MANY SETS OF THESE FIBRES ARE THERE?

Four. The first spring from the cortex, cross over to the opposite hemisphere by means of the corpus callosum, to connect homologus regions of the cortex of the two hemispheres. They are called commissural fibres, and constitute the transverse fibres of the corpus callosum. The second set arise from the cortex, accompany the commissural fibres for a short distance and then separate without passing to the opposite hemisphere, some passing into the basal ganglia, others going to form the internal and external capsules. They are called radiating fibres, or corona radiata. The third set connect different portions of the cortex of the same hemisphere, and are called associating fibres. The fourth set connect the cortex of the temporo-sphenoidal lobes with the optic thalami, and constitute the so-called fornix fibres. (Fig. 10).

THE CEREBELLUM.

WHAT IS THE CEREBELLUM AND WHERE IS IT SITUATED?

The cerebellum, or little brain, is that portion of the encephalon contained in the inferior occipital fossæ. It is situated beneath the posterior lobes of the cerebrum, from which it is separated by the tentorium. Its average weight is about five ounces. In form it is oblong and flattened from above downward, its greatest diameter being from side to side. It is composed of gray and white matter, the gray matter occupying the surface, as it does in the cerebrum. The surface of the cerebellum is not convoluted like the cerebrum, but has numerous curved furrows or sulci.

WHAT ARE THE DIVISIONS OF THE CEREBELLUM?

It is divided into two hemispheres and a central portion called the vermiform process.

Upon the upper surface of each hemisphere there are two lobes: the anterior or square lobe, and the posterior or semi-lunar lobe. Upon the surface there are five lobes; the flocculus or sub-peduncular, the amygdala or tonsil, the digastric, the slender, and the inferior posterior lobe.

WHAT KIND OF FIBRES ARE FOUND IN THE CEREBELLUM?

The peduncular fibres, which form the peduncles of the cerebellum, and the fibres proper of the cerebellum.

WHAT ARE THE PEDUNCLES OF THE CEREBELLUM?

They are bundles of fibres which connect the cerebellum with other portions of the brain. There are three of these bundles, called the superior, middle, and inferior peduncles.

The superior peduncles (processus e cerebelli ad testes) arise from the middle of the white matter of the cerebral hemispheres, pass beneath the testes of the corpora quadrigemina, and run outward and backward to the cerebellum.

The middle peduncles (processus ad pontem) connect the two hemispheres of the cerebellum, forming their great transverse commissure.

The inferior peduncles (processus ad medullam), connect the cerebellum with the medulla oblongata.

The fibres proper of the cerebellum are of two kinds: commissural fibres, which cross the middle line to connect the opposite halves of the hemispheres, and the arcuate or association fibres, which connect one lamina with another.

WHAT MASSES OF BRAIN MATTER ARE IN THE INTERIOR OF THE CEREBELLUM?

The nucleus of the roof, or the nucleus fastigii, the nucleus emboliformis, the nucleus globosus, and the corpus dentatum. The gray matter of the surface with the white matter of the interior is so arranged in a series of laminæ that it gives a foliated appearance which is called the arbor vitæ.

HOW IS THE CORTEX OF THE CEREBELLUM DIVIDED?

Into a granular layer, a layer of large cells, and a molecular layer.

The granular layer is composed of minute round cells and larger cells with processes called the cells of Purkinje.

The molecular layer contains large and small cells, some of which are multipolar and send processes to end in a plexus around the cells of Purkinje.

WHAT IS THE FUNCTION OF THE CEREBELLUM?

The precise function subserved by the cerebellum is not exactly known, but there is abundant evidence, experimental and pathological, to show that it has to do with coördination of movement, and particularly with those muscular actions which enable a person to maintain his equilibrium. It is the median portion or the vermiform process which is really the active portion of the cerebellum and controls coördinate movements. Diseases of the outer portions of the hemispheres seem to have no effect at all upon the function of the cerebellum.

THE MEDULLA OBLONGATA.

WHAT IS THE MEDULLA OBLONGATA?

It is the upper enlarged part of the spinal cord, and extends from the upper border of the atlas to the lower border of the pons Varolii. It is pyramidal in form, its broad ex-

tremity directed upward, its lower end being narrow at its point of connection with the cord. It measures about one and one-fourth inches in length and three-fourths of an inch in breadth at its widest part, and one-half an inch in depth. Its surface has an anterior and posterior-median fissure, continuous with the anterior and posterior-median fissure of the cord, which divides the medulla into two

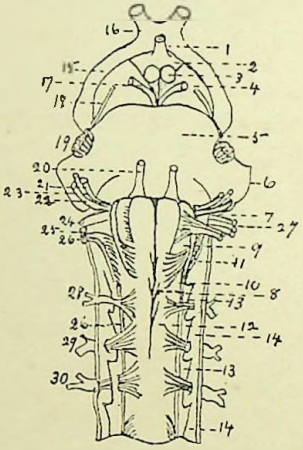


Figure 11.

Anterior view of the medulla oblongata. (Sappey).
1, infundibulum. 2, tuber cinereum. 3, corpora albicantia. 4, cerebral peduncle. 5, tuber annulare. 6, origin of the middle peduncle of the cerebellum. 7, anterior pyramids of the medulla oblongata. 8, decussation of the anterior pyramids. 9, olivary bodies. 10, restiform body. 11, arciform fibers. 12, upper extremity of the spinal cord. 13, ligamentum denticulatum. 14, 11, dura mater of the cord. 15, optic tracts. 16, chiasm of the optic nerves. 17, motor oculi. 18, patheticus. 19, fifth nerve. 20, motor oculi externus. 21, facial nerve. 22, auditory nerve. 23, nerve of Wrisberg. 24, glosso-pharyngeal nerve. 25, pneumogastric. 26, 26, spinal accessory. 27, hypo-glossal or sublingual nerve. 28, 29, 30, cervical nerves.

halves, each half being divided into five columns—the anterior pyramid, lateral tract, olivary body, restiform body and the posterior pyramid. There are also arcuate fibres, which separate portions of gray matter of the medulla. (Fig. 11).

The posterior surface of the medulla contains part of the floor of the fourth ventricle and is a most important part of the human anatomy, because it contains the nuclei of origin of several of the cranial nerves.

THE PONS VAROLII.

WHAT IS THE PONS VAROLII AND WHERE IS IT SITUATED?

The pons is the bond of union of the various segments of the brain, connecting the cerebrum above, the medulla oblongata below, and the cerebellum behind. It is situated above the medulla, below the crura cerebri, and between the hemispheres of the cerebellum. It is composed of longitudinal fibres, which ascend from the medulla to pass to the cerebrum, and transverse fibres, which connect the hemispheres of the cerebellum. These fibres are arranged in alternate layers and are intermixed with gray matter. The transverse fibres constitute the great transverse commissure, or tuber annulare. The longitudinal fibres are continued up from the anterior pyramids, from the olivary body and from the lateral and posterior columns of the cord.

THE VENTRICLES OF THE BRAIN.

WHAT ARE THE VENTRICLES OF THE BRAIN AND HOW MANY ARE THERE?

They are serous cavities formed in the interior of the brain, five in number—two lateral, and the third, fourth and fifth.

DESCRIBE THE LATERAL VENTRICLES.

The two lateral ventricles form the upper part of the ventricular space. Each consists of a central cavity and three smaller cavities or cornua. Each ventricle is lined by a thin membrane called the ependyma. They are separated from each other by a vertical septum, the septum lucidum.

The central cavity is bounded above by the corpus callosum, which forms the roof of the cavity. Its floor is formed by the corpus striatum, tenia semicircularis, optic thalamus, choroid plexus, corpus fimbriatum and fornix.

The anterior cornua is triangular in form and curves around the anterior extremity of the corpus striatum. It is bounded above by the corpus callosum and below and externally by the corpus striatum.

The posterior cornua curves backward into the substance of the posterior lobe.

WHAT IS THE CHOROID PLEXUS?

It is a little vascular, fringe-like membrane, occupying the margin of the fold of pia mater (velum interpositum) in the interior of the brain. It extends in a curved direction across the floor of the lateral ventricle.

WHAT IS THE CORPUS FIMBRIATUM OR TENIA HIPPOCAMPI?

It is a narrow, white, tape-like band situated immediately behind the choroid plexus.

WHERE IS THE FIFTH VENTRICLE?

It is situated between the two layers of the septum lucidum, a semi-transparent septum which is attached above to the corpus callosum, and below to the anterior part of the fornix.

DESCRIBE THE THIRD VENTRICLE?

It is a narrow, oblong fissure situated between the optic thalami. It is bounded above by the velum interpositum; its floor is formed by the posterior perforated space, corpora albicantia, tuber cinereum, infundibulum and the lamina cinerea. The cavity of the ventricle is crossed by three commissural bands; the posterior connects the optic thalami; it bounds the ventricle posteriorly and is placed in front of and beneath the pineal gland and above the aqueduct of Sylvius, a canal leading from the third to the fourth ventricle. The middle commissure is composed of gray matter and also connects the optic thalami. The anterior commissure is a round, white cord of fibres placed in the forepart of the cavity. Just behind the anterior commissure are the foramina of Monro, which afford communication between the third ventricle and the two lateral ventricles by a Y-shaped passage.

DESCRIBE THE FOURTH VENTRICLE?

It is the space between the cerebellum and the posterior surfaces of the medulla oblongata and pons. It is triangular in shape and is roofed over by the valve of Vieussens, a thin layer of gray matter, and bounded on either side by the superior peduncles of the cerebellum, and behind by the diverging posterior pyramids and restiform bodies.

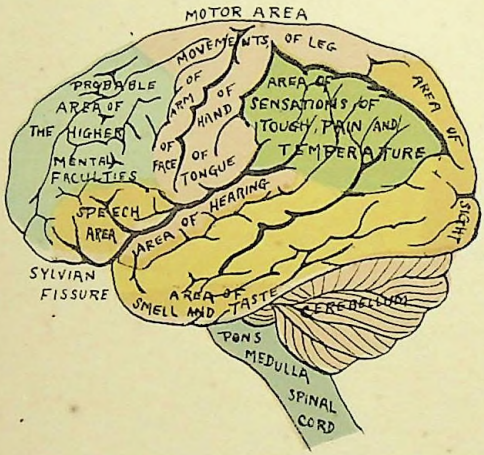


Figure 12.

Diagram illustrating the probable functions of different areas of the cerebral cortex. (Ranney).

PLATE II

CEREBRAL LOCALIZATION.

WHAT IS MEANT BY CEREBRAL LOCALIZATION?

The determining of the location of the various functional centres upon the surface of the cerebral cortex which control the different parts of the body. (See Plate II).

NAME CENTRES IN ANTERIOR PORTION OF FRONTAL LOBE.

Probably the higher mental faculties, such as volition, attention, emotion, self-control and thought.

NAME CENTRES IN POSTERIOR PORTION OF FRONTAL LOBE.

The head, shoulder, head and eye movements, and in the left third frontal convolution, the speech centre of Broca.

NAME CENTRES IN ASCENDING FRONTAL AND ASCENDING PARIETAL CONVOLUTIONS.

The trunk, hip, knee, leg, conjoint shoulder, arm, wrist, fingers and thumb, eyes, upper face, lower face, lips, larynx and pharynx.

WHAT CENTRES ARE LOCATED IN THE PARIETAL LOBE?

In the superior portion, the foot and toe centre: in the middle and inferior convolutions, the areas of sensation, touch, pain and temperature.

WHAT CENTRE IS LOCATED IN THE OCCIPITAL LOBE?

The centre of sight.

WHAT CENTRES ARE IN THE TEMPORO-SPHENOIDAL LOBE?

In the superior portion, the area of hearing; in the inferior portion, the areas of smell and taste.

FROM WHENCE DOES THE BRAIN RECEIVE ITS BLOOD SUPPLY?

From the two internal carotid arteries, which enter the skull through the carotid canals in the temporal bones; and from the two vertebral arteries, which after passing through the foramen magnum unite to form the basilar artery on the surface of the pons Varolii.

These two vessels give off branches which go to form the circle of Willis.

WHAT IS THE CIRCLE OF WILLIS?

It consists of two sets of vessels: the anterior or carotid set, from which arise the anterior and middle cerebral arteries; and the posterior or vertebral set, consisting of the basilar and posterior cerebral arteries.

The internal carotid and the posterior cerebral arteries are joined together, across the longitudinal fissure, by the anterior communicating arteries.

GIVE ORIGIN AND COURSE OF ANTERIOR CEREBRAL ARTERIES.

The anterior cerebral artery arises from the internal carotid, passes forward in the great longitudinal fissure, curves around the anterior border of the corpus callosum, running along its upper surface, and anastomoses with the posterior cerebral arteries.

DESCRIBE THE COURSE OF THE MIDDLE CEREBRAL ARTERY.

The middle cerebral artery is the largest branch of the internal carotid. It passes within the fissure of Sylvius and divides into three branches; the anterior supplying the anterior lobe, the posterior supplying the middle lobe, and the median supplying the small lobe of the outer extremity of the fissure of Sylvius. A particular branch called the lenticulo-striate artery is distributed to the lenticular and caudate nuclei, and is often the source of cerebral hemorrhage.

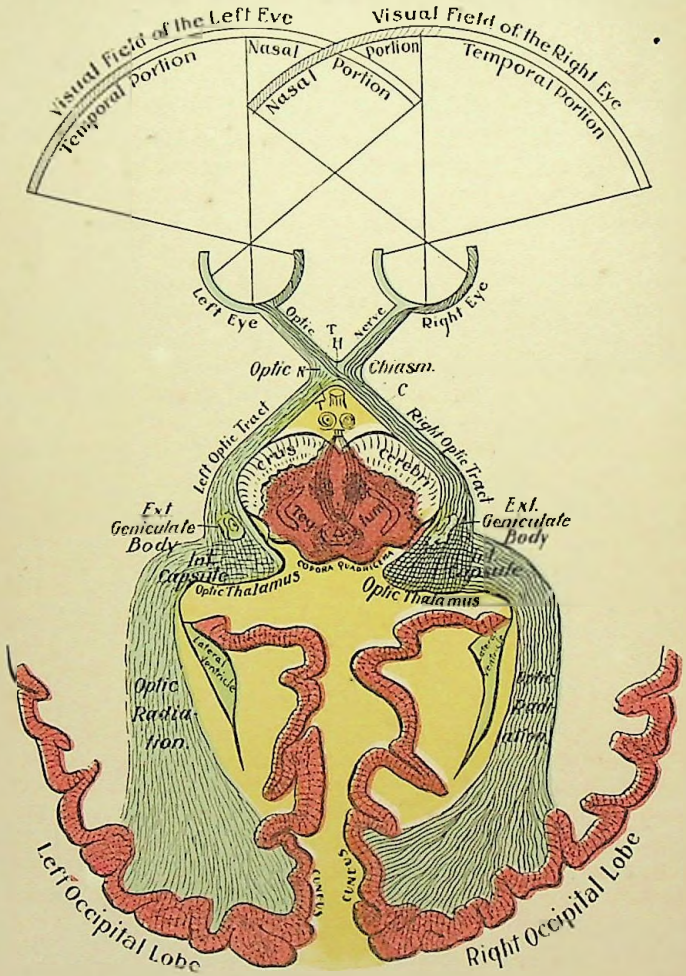
GIVE THE COURSE OF THE POSTERIOR CEREBRAL ARTERY.

The posterior cerebral artery passes along the under surface of the occipital lobes and gives off branches which pass into the posterior perforated space and are distributed to the uncinate gyrus, to the temporo-sphenoidal lobe, and the cuneus and occipital lobes.

CRANIAL NERVES.

GIVE DIVISIONS AND NAMES OF THE CRANIAL NERVES.

There are twelve pairs. 1st, or olfactory; 2nd, or optic; 3rd, or motor oculi; 4th, or patheticus or trochlear; 5th, or trigeminus or trifacial; 6th, or abducens; 7th, or fa-



Optic and Visual Tracts. N, lesion causing nasal hemianopsia; T, lesion causing temporal hemianopsia; H, lesion causing bi-temporal hemianopsia; C, lesion of optic tract, causing left-lateral hemianopsia.

PLATE III.

cial: 8th, or auditory: 9th, or glosso-pharyngeal; 10th, or pneumogastric; 11th, or spinal accessory; 12th, or hypoglossal.

GIVE THE ORIGIN, COURSE AND DISTRIBUTION OF THE CRANIAL NERVES.

Olfactory.—The first, or olfactory, arises by three roots. The outer white root originates in a nucleus of gray matter in the anterior part of the middle lobe of the hemisphere, and from the fissure of Sylvius passes along the outer side of the anterior perforated space. The middle or gray root arises from the anterior perforated space. The interior white root arises from the gyrus fornicatus. These roots join together to form the olfactory bulb. On their under surface they rest upon the cribriform plate of the ethmoid bone, and give off about twenty nerves which are distributed to the nasal mucous membrane.

Optic.—The second, or optic, arises from the optic commissure. This commissure is formed by fibres which pass across from one side of the brain to the other without any connection with the optic nerve, which are called intercerebral-commissural fibres, and connect the optic thalami of the opposite sides. There are also fibres which cross from one side to the other in the anterior portion of the commissure, connecting the optic nerves of the two sides, having no relation with the optic tracts, and called interretinal-commissural fibres. The fibres of the outer portion of each tract pass to the optic nerve of the same side, while the central fibres of each tract pass to the optic nerve of the opposite side, decussating in the commissure with similar fibres of the opposite tract. The optic tracts arise from the anterior lobes of the corpora quadrigemina, the corpora geniculata, and the posterior portions of the optic thalami, winding around the crura cerebri. The optic nerve passes forward through the optic foramen, enters the back part of the eye-ball and expands into the retina.

Motor Oculi.—The third, or motor oculi, appears in front of the pons Varolii, issuing from among the fibres on the under side of the crus cerebri. The nerve originates from a nucleus beneath the passage-way between the third and fourth ventricles, passing forward through the locus

niger and the tegmentum, and enters the orbit by the sphenoidal fissure to be distributed to the muscles of the eye-ball, with the exception of the external rectus and the superior oblique. It is the motor nerve of the eye. The fibres connected with the internal rectus nucleus decussate.

Patheticus.—The fourth, or patheticus, or trochlear, originates from a gray nucleus in the aqueduct of Sylvius, winds around the outer side of the crus cerebri, and enters the orbit by the sphenoidal fissure to supply the superior oblique muscle. Their fibres decussate in the roof of the aqueduct.

Trifacial.—The fifth, or trigeminus, or trifacial, appears at the back of the brain, issuing in two separate bundles of fibres from the sides of the pons Varolii near its anterior border. Each nerve has two distinct roots: the anterior root consisting of three or four bundles of fibres, and having motor function; the posterior composed of from seventy to one hundred bundles of fibres and having sensory function. The two roots commence in the upper portion of the medulla, the sensory root originating in the gray tubercle of Rolando, and the motor root from some large cells connected with the medulla. After the two roots issue from the pons they proceed forward to the apex of the petrous portion of the temporal bone where a ganglionic enlargement occurs upon the sensory root, called the Gasserian ganglion. The motor root passes beneath the ganglion and, not having connection with it, proceeds independently with the inferior maxillary branch of the sensory root to the foramen ovale, and after its exit from this foramen blends its fibres with those of the inferior maxillary. From the anterior border of the Gasserian ganglion three nerves are given off: the ophthalmic nerve, which passes through the sphenoidal fissure: the superior maxillary nerve, which passes through the foramen rotundum: and the inferior maxillary nerve, which passes through the foramen ovale. The ophthalmic branch, which is a sensory nerve, supplies the eye-ball, the lachrymal gland, the mucous lining of the eye and nasal fossæ, the integument and muscles of the eye-brow, forehead and nose. The superior maxillary branch, also sensory, subdivides into branches which spread out upon the side of the nose, the lower eye-lid and

the upper lip, joining with filaments of the facial nerve. The inferior maxillary branch distributes branches to the teeth and gums of the lower jaw, the integument, the tympanum and external ear, the lower part of the face and lower lip, and the muscles of mastication. It also supplies the tongue with a large branch which possibly serves as a nerve of the special sense of taste. The anterior root of the fifth nerve, which is motor, divides into two branches which supply the muscles of mastication.

ABDUCENS.—The sixth, or abducens, supplies the external rectus muscle. It arises from the gray substance in the floor of the fourth ventricle, and emerges from between the pons and the anterior pyramids of the medulla. It leaves the skull by the sphenoidal fissure.

FACIAL.—The seventh, or facial, has its deep origin in the floor of the fourth ventricle, and emerges between the pons and the restiform tract of the medulla. It enters the internal auditory opening in the temporal bone, and after passing through the aqueduct of Fallopius passes out by the stylo-mastoid foramen to be distributed to the facial muscles. It is motor in function.

AUDITORY.—The eighth, or auditory, also arises from the floor of the fourth ventricle, near the origin of the seventh nerve and beneath the acoustic tubercles. It enters the internal auditory opening of the temporal bone in company with the facial nerve. Within the auditory opening the nerve subdivides into cochlear and vestibular branches, which are distributed to the internal ear. It is the nerve of the special sense of hearing.

GLOSSO-PHARYNGEAL.—The ninth, or glosso-pharyngeal, arises from the floor of the fourth ventricle, below the nucleus of the auditory nerve, and appears on the surface of the restiform body. It leaves the skull from the middle part of the jugular foramen and is distributed to the mucous membrane of the pharynx and the back of the tongue. It is the special nerve of taste in all parts of the tongue to which it is distributed.

PNEUMOGASTRIC.—The tenth, or pneumogastric or par vagum, is composed of both motor and sensory fibres, and supplies the organs of voice and respiration with motor and sensory filaments, and the pharynx, esophagus, stomach

and heart with motor influence. It has its deep origin in the lower part of the floor of the fourth ventricle; and its superficial origin between the restiform and olivary bodies, below the glosso-pharyngeal, and passes through the jugular foramen.

Spinal Accessory.—The eleventh, or spinal accessory, consists of two parts: one, the upper or accessory part to the vagus; and the other, the lower or spinal portion. The accessory part arises from the medulla, below the pneumogastric; and the lower part arises from the spinal cord. The combined nerve passes out to the jugular foramen with the pneumogastric and glosso-pharyngeal nerves; the accessory portion blends with the pneumogastric, while the spinal portion supplies the sterno-mastoid and trapezius muscles.

Hypoglossal.—The twelfth, or hypoglossal, is the nerve of motion of the tongue. It may be seen to arise on the surface of the medulla, between the olivary body and the anterior pyramid. The nucleus from which it arises is found on the floor of the fourth ventricle. It passes out of the skull through the anterior condyloid foramen, and is distributed to the muscles of the tongue and the depressor muscles of the hyoid bone and the larynx.

THE SPINAL CORD.

WHAT IS THE SPINAL CORD?

It is that portion of the central nervous system contained within the canal of the vertebral column. In the fetus it extends the whole length of the vertebral canal, but it does not grow in proportion to the vertebral column and in the adult is about fifteen or eighteen inches in length. It ends in a mesh of nerve fibres called the cauda equina. It is about as large around as a lead-pencil, and is in the form of a flattened cylinder with the flat surfaces anterio-posterior. It has two enlargements, one called (a) the cervical because it is situated in the cervical region; and the other, called (b) the lumbar enlargement because it is situated in the lumbar region.

(a). The cervical enlargement extends from the third cervical to the first dorsal vertebra and its greatest diameter is across the cord.

(b). The lumbar enlargement extends from the last dorsal to the second lumbar vertebra, and its greatest diameter is antero-posterior.

The cord weighs about one and one-half ounces when denuded of its membranes.

WHAT ARE THE COVERINGS OF THE CORD?

(a) The dura mater, (b) the arachnoid, and (c) the pia mater, being of the same structure as and continuous with those of the brain.

(a). The dura mater forms a loose sheath around the cord, separated from the walls of the spinal column by areolar, adipose tissue. It is attached to the edges of the foramen magnum, extends below to the top of the sacrum, and then continues as a slender cord to the coccyx, where it becomes a part of the periosteum. It is the protecting membrane of the cord as it is in the brain.

(b). The arachnoid is a thin, delicate membrane investing the surface of the cord and connected to the pia mater by connective tissue filaments. The space between the dura mater and the arachnoid is known as the sub-dural space.

(c). The pia mater is beneath the arachnoid membrane and invests the cord like a glove. It is the true nourishing membrane of the cord, and sends prolongations into its substance which are abundantly supplied with bloodvessels. It sends out also little fibrous processes to the inner surface of the dura mater, called the ligamenta denticulatæ, situated on each side of the spinal cord throughout its entire length and separating the anterior and posterior roots of the spinal nerves. There are about twenty of these processes upon each side of the cord, and they serve as supports to the cord. The space between the arachnoid and the dura mater is called the sub-arachnoid space.

Both the sub-dural and the sub-arachnoid spaces are filled with cerebro-spinal fluid. This is more abundant, however, in the sub-arachnoid space which is directly continuous with the ventricular cavities in the interior of the brain. The fluid is the same in both and passes from the ventricles of the brain through the foramen of Magendie. The amount contained within the sub-arachnoid space is

probably about an ounce. It acts as a cushion to the spinal cord by subduing the effects of shock in the cord from external violence. The pia mater extends downward from where the cord terminates to the top of the sacral canal in a prolongation called the filum terminale.

WHAT DOES THE SURFACE OF THE SPINAL CORD PRESENT?

On its anterior surface, along the median line, there is a longitudinal fissure called the anterior median fissure; and on the posterior surface is the posterior median fissure. They divide the cord into two halves which are united in the middle throughout their entire length by a band of nervous substance called the commissure. The floor of the anterior median fissure is formed by white matter called the anterior white commissure, and the floor of the posterior median fissure is formed by gray matter called the posterior gray commissure.

On either side of the anterior median fissure are the antero-lateral fissures of the cord, and on either side of the posterior median fissure are the postero-lateral fissures. Between the postero-lateral fissure and the posterior-median fissure are the postero-intermediary fissures.

The anterior roots of the spinal nerves spring from the antero-lateral fissure; the posterior roots of the spinal nerves enter the spinal cord at the postero-lateral fissure.

NAME THE COLUMNS OF THE CORD.

On either side of the anterior median fissure are the columns of Turck, or the direct pyramidal tracts. On either side of the columns of Turck are the anterior root-zones. Outside of the anterior root-zones are the antero-lateral columns, or the unknown tracts. The direct cerebellar columns occupy the space on either side of the cord at its outer extremity; and just within these columns are the crossed pyramidal tracts. On either side of the posterior median fissure are the columns of Goll, or the posterior-median columns, and outside of these columns are the columns of Burdach. These latter columns, with the columns of Goll, are often called the posterior columns.

These columns are all composed of white matter. Within

the centre of the cord is the gray substance, and it is so arranged as to present on the surface of a section two crescentic masses, one in each lateral half of the cord, united by a transverse band of gray matter called the gray commissure.

Each of these masses has an anterior and posterior horn or cornua. The anterior horn is broader and thicker than

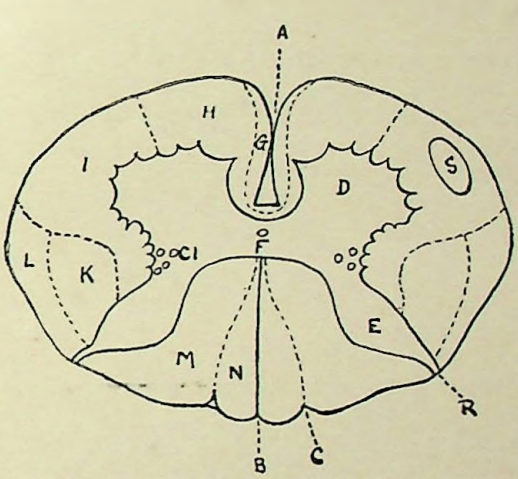


Figure 13.

A diagram showing the more important subdivisions of the spinal cord (Flechsig).

A, anterior median fissure. B, posterior median fissure. C, intermediate fissure. D, anterior gray cornu. E, posterior gray cornu. F, gray commissure with central canal. G, direct pyramidal tract or column of Turck. H, fundamental part of the anterior column (anterior root-zones). I, anterior part of lateral column. K, crossed pyramidal tract of lateral column. L, direct tract from lateral column to cerebellum. M, column of Burdach. N, column of Goll. Cl, vesicular column of Clarke. S, sensory tract of lateral column.

the posterior and does not come to the surface of the cord; while the posterior horn is narrow and projects to the postero-lateral fissure upon the surface of the cord. In the centre of the gray commissure, and extending throughout its whole length, is a small canal called the central canal.

On the under side of the neck of each posterior cornua, just behind the gray commissure, are the visceral columns (of Clarke). (Fig. 13).

GIVE THE MINUTE ANATOMY OF THE SPINAL CORD.

The gray matter consists of a structure called the substantia spongiosa, composed of nerve cells imbedded in a mesh of connective tissue, the neuroglia, and is traversed by bundles of medullated nerve fibres passing in different directions and interspersed with small bloodvessels. The cells of the anterior cornua are large and multipolar. The processes of these cells intermingle with each other. Many of the medullated nerve fibres are connected by their axis-cylinders with the nerve cells, then become associated into bundles, which go to form the anterior roots of the spinal nerves. Transverse nerve fibres passing through the anterior commissure connect some nerve cells of the opposite side with each other. The gray commissure consists chiefly of transverse medullated fibres upon a matrix of neuroglia. Some of these fibres pass from the posterior roots of the spinal nerves of one side across the gray commissure to the posterior cornua of the opposite side; some pass to the anterior cornua of the same side, and some to the anterior cornua of the opposite side.

The white matter is destitute of nerve cells, and is composed of longitudinal medullated nerve fibres, except in the white commissure, where the fibres are transverse, and in the anterior roots of the spinal nerves, where they are oblique. The white commissure is formed of fibres extending from the anterior cornua of gray matter on one side to the white substance of the anterior column of the opposite side. The nerve fibres of the white matter are supported by neuroglia which has small bloodvessels ramifying within it.

The nerve fibres within the spinal cord are destitute of the white sheath of Schwann. The coarser fibres are found in the white matter, the next in size are found in the anterior gray matter, while the finest nerve fibres are found in the posterior gray matter.

WHAT IS MEANT BY A SEGMENT OF THE SPINAL CORD?

A segment of the spinal cord is a transverse section about the width of one of the vertebræ, including a pair of spinal nerves. The spinal cord is composed of a series of superimposed segments.

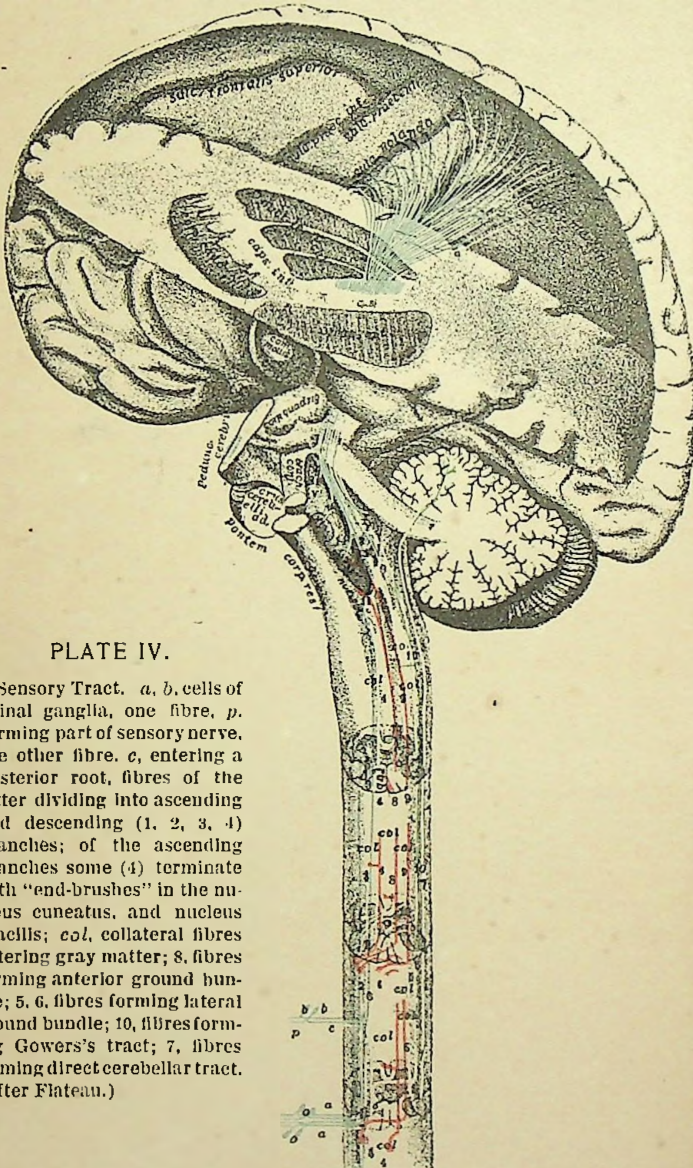


PLATE IV.

Sensory Tract. *a*, *b*, cells of spinal ganglia, one fibre, *p*, forming part of sensory nerve, the other fibre, *c*, entering a posterior root, fibres of the latter dividing into ascending and descending (1, 2, 3, 4) branches; of the ascending branches some (4) terminate with "end-brushes" in the nucleus cuneatus, and nucleus gracilis; *col*, collateral fibres entering gray matter; 8, fibres forming anterior ground bundle; 5, 6, fibres forming lateral ground bundle; 10, fibres forming Gowers's tract; 7, fibres forming direct cerebellar tract. (After Flateau.)

WHY ARE THE DIRECT PYRAMIDAL TRACTS SO CALLED?

Because they convey longitudinal fibres directly from the anterior pyramids within the medulla down to the spinal cord.

WHY ARE THE CROSSED PYRAMIDAL TRACTS SO CALLED?

Because their fibres decussate within the medulla. Nine fibres decussate to one that passes directly down.

WHAT ARE THE FUNCTIONS OF THE DIRECT AND CROSSED PYRAMIDAL TRACTS?

They convey motor impulses from the brain to the periphery.

WHAT ARE THE FUNCTIONS OF THE COLUMNS OF GOLL AND BURDACH?

The columns of Goll convey sensory impulses, probably tactile sense.

The columns of Burdach convey tactile sense: they also convey sensations from the muscles and articulations, and when they are diseased there is a loss of the so-called muscular sense.

The fibres of the columns of Goll decussate in the medulla, while those of the columns of Burdach cross over at once.

WHAT ARE THE FUNCTIONS OF THE ANTERO-LATERAL COLUMNS?

The antero-lateral columns convey sensations of pain and temperature from the opposite side of the body, coming across the anterior and posterior commissures.

WHAT ARE THE FUNCTIONS OF THE CELLS CONTAINED IN THE GRAY MATTER?

The cells in the anterior horn have motor and trophic functions. The larger cells are situated at the outer part of the horn and send fibres to the large muscles. The smaller cells are situated near the centre and send fibres to the small muscles, those having more delicate function. In the innermost cells are the trophic cells for muscles and groups of cells which preside over vaso-motor and secretory functions.

The visceral columns of Clarke receive fibres from the viscera and bloodvessels, and conduct impulses from the viscera.

The cells of the posterior horns are sensory in function. In the posterior horns there are also situated the trophic centres for the joints, bones and skin; their fibres pass out through the posterior roots.

WHAT IS MEANT BY THE AUTOMATIC ACTION OF THE SPINAL CORD?

That function of the spinal cord which enables it to perform voluntary actions independently of the brain, as when a person suddenly recovers himself after slipping upon some substance. There are groups of nerves and cells in the cord which are called spinal automatic centres. They are:

(a). The cilio-spinal centre, which extends from the seventh cervical to the second dorsal vertebra; and its stimulation causes the pupil to contract.

(b). The genital centres which extend from the first to the third sacral segments and preside over erection and ejaculation.

(c). The bladder and rectal centres, which are located in the fifth sacral segment.

(d). The vaso-motor centres, located in that portion of the cord extending from the second dorsal to the second lumbar segments.

GIVE THE BLOOD SUPPLY OF THE SPINAL CORD.

From the branches of the vertebral, ascending cervical and superior intercostal arteries above, and from the dorsal, intercostal, lumbar and sacral arteries below. They enter the spinal cord through the foramen magnum above and the intervertebral foramina on the sides. They are distributed on the pia mater and in the cord.

The arteries that thus supply the cord are the anterior spinal, posterior spinal and lateral spinal.

The anterior spinal arteries unite to form the anterior median artery which extends down the whole length of the cord, receiving branches from the lateral arteries.

The posterior spinal arteries are much smaller than the anterior, and unite with each other on the posterior surfaces

of the cord. They do not continue down, however, in the median artery as the anterior arteries do.

The lateral spinal arteries are derived from branches of the subclavian.

The substance of the cord is supplied by central arteries which are branches of the anterior median and peripheral arteries, coming from plexuses on the pia mater.

SPINAL NERVES.

WHAT ARE THE SPINAL NERVES?

Nerves which take their origin from the spinal cord and pass through the intervertebral foramina on each side of the spinal canal. They are arranged in groups corresponding with that portion of the spinal canal through which they pass. There are thirty-one pairs of them—the cervical, having eight pairs; dorsal, twelve pairs; lumbar, five pairs; sacral, five pairs and the coccygeal one pair. Each pair arises from two roots; an anterior or motor root and a posterior or sensory root. Upon the posterior root is the small ganglion which is common to all sensory nerves. The two roots join together to form a combined nerve which passes out of the spinal cord.

WHAT ARE THE DIVISIONS OF THE SPINAL NERVES?

After the spinal nerves pass out of the intervertebral foramina they divide into an anterior division, which supplies the anterior part of the body, and the posterior division, which supplies the posterior part. Each of these divisions contains fibres from both roots.

The anterior divisions are usually larger than the posterior, because they supply a larger extent of structure. Each division is connected with the sympathetic by a slender filament.

WHAT PLEXUSES DO THE ANTERIOR DIVISIONS OF THE SPINAL NERVES FORM?

The four upper cervical nerves form the cervical plexuses; the four lower cervical nerves and first dorsal form the brachial plexus; and the anterior divisions of the dorsal from the second to the eleventh constitute the intercostal

nerves; the four upper lumbar nerves form the lumbar plexuses; and the five lumbar nerves and four upper sacral nerves form the sacral plexus.

WHAT DO THE POSTERIOR DIVISIONS SUPPLY?

The erecto-spinæ muscles and the superficial muscles of the back; also the skin of the back of the head, neck and trunk.

INTO WHAT TWO GROUPS ARE THE BRANCHES OF THE CERVICAL PLEXUS DIVIDED?

Into the superficial and deep.

SUPERFICIAL GROUP.—Branches of the superficial group are the superficialis-colli, the auricularis magnus and the occipitalis minor, the sterno-clavicular and acromial.

DEEP GROUP.—The branches of the deep group are the communicating, muscular, communicans noni and the phreni.

WHAT ARE THE BRANCHES OF THE BRACHIAL PLEXUS?

They are divided into two groups—those above the clavicle and those below.

The branches above are the communicating, muscular, posterior thoracic and supra-scapular.

The branches below the clavicle are the anterior thoracic, distributed to the chest; the subscapular and circumflex, distributed to the shoulder; the musculo-cutaneous, internal cutaneous, lesser cutaneous, median, ulnar and musculo-spiral, distributed to the arm, forearm and hand.

HOW MANY INTERCOSTAL NERVES ARE THERE AND TO WHAT ARE THEY DISTRIBUTED?

Twelve on each side, distributed to the parietes of the chest and abdomen. They are not joined together in a plexus, in which respect they differ from the other spinal nerves.

The six upper nerves are distributed to the walls of the chest; and the six lower supply the walls of the chest and the abdomen.

TO WHAT ARE THE POSTERIOR DIVISIONS OF THE DORSAL NERVES DISTRIBUTED?

They are divided into external and internal branches.

The external branches are distributed to the longissimus dorsi, the sacro-lumbalis and the levatores costarum.

The internal branches supply the multifidus spinæ, the semi-spinalis dorsi, the rhomboidei and the trapezius.

WHAT DO THE POSTERIOR DIVISIONS OF THE LUMBAR NERVES SUPPLY?

The erector spinæ and inter-transverse muscles, the latissimus dorsi, the integument of the gluteal region, and some of its fibres pass as far as the trochanter major. Some of its branches supply also the multifidus spinæ and inter-spinalis muscles.

WHAT DO THE ANTERIOR DIVISIONS OF THE LUMBAR NERVES SUPPLY?

The lumbar plexus, which gives off the following branches: the ilio-hypogastric, ilio-inguinal, genito-crural, external cutaneous, obturator, accessory obturator, anterior crural: they also supply the integument of the front and under side of the leg and all the muscles of the front of the thigh except the tensor vaginæ femoris.

HOW ARE THE SACRAL NERVES DIVIDED AND WHAT ARE THEIR BRANCHES?

The first four are divided into anterior and posterior branches

The posterior branches pass through the posterior sacral foramina and supply the multifidus spinæ muscles and the integument the sacrum and coccyx.

WHAT CONSTITUTES THE SACRAL PLEXUS?

The first three anterior branches, with the lumbo-sacral, and the four sacral nerves which unite and form the sacral plexus. A large number of the fibres of this plexus go to form the sciatic nerve. The sacral and coccygeal nerves arise from the cauda equina.

The following are the branches of the sacral plexus: Superior gluteal, muscular, pudic, small sciatic, great sci-

HOW MANY KINDS OF INTERNAL PACHYMENINGITIS ARE THERE?

Purulent, which is usually associated with inflammation of the pia mater; and

Hemorrhagic, or hematoma of the dura mater, which consists of an effusion of blood between membranous layers which extend over both hemispheres, with a slight inflammation of the inner surface of the dura mater.

LEPTOMENINGITIS.

HOW MANY KINDS OF LEPTOMENINGITIS ARE THERE?

Simple, tubercular, syphilitic and epidemic cerebro-spinal meningitis, all of which may be acute or chronic.

WHAT ARE THE CAUSES OF ACUTE SIMPLE LEPTOMENINGITIS?

It is most common in children under ten years of age, more frequent in males than in females. Traumatic influences, such as laceration of the membranes and hemorrhage or concussion, are direct causes; as are also adjacent diseases, such as caries of the bone, abscess of the middle ear, which is extremely common in children, and disease of the mastoid cells, diseases of the upper nasal passages, inflammation of the eye, tumors or abscesses of the brain, cerebral hemorrhage, acute specific diseases, such as measles, scarlet fever, small pox, typhoid fever, acute pneumonia. Septicemia, from any cause, may also produce the trouble, this result being undoubtedly due to the presence of an organized virus within the blood which produces a toxic condition. Exposure to heat, such as sunstroke, excessive mental work and worry are occasional causes.

WHAT IS THE PATHOLOGICAL ANATOMY?

In the early stages diffuse reddening of the pia mater, which is soon after followed by an opacity of the membranes. This opacity is well seen over the convexity, and also at the base of the brain. There are also collections of yellowish-white, semi-purulent lymph around the nerve trunks which very closely resemble tubercular granulations. The nerve trunks are often involved, and small hemorrhages may be seen in the nerve itself. The dura mater may also

be involved, either in the reddening or its under surface may be covered with lymph. The fluid in the sub-arachnoid space is sometimes increased in quantity. There may be an ependymitis, an inflammation of the ependyma within the ventricles. The choroid plexus and velum interpositum may also be inflamed. Spots of softening occur over the surface of the brain near the inflamed portions.

WHAT ARE THE SYMPTOMS?

There is sometimes general indisposition, languor and malaise, mental irritability and vomiting without known cause, for some days before the onset of the disease. The pronounced symptoms may set in suddenly and are as follows:

Headache.—Characterized by its severity, persistency, and frequent exacerbations. Even during stupor it is apparent that the patient is suffering from intense pain in the head. There is also a rolling about of the head, and a sharp shriek called the "hydrocephalic cry."

Delirium.—Which may be either slight or very violent. It is sometimes seen in the early stages of the disease in those who are especially predisposed to delirium.

Vomiting.— Is simply a rejection of food without nausea, projectile in character. It is a common and early symptom. The tongue may be clean, bowels constipated and abdomen retracted.

General Convulsions.—More frequent in children, occurring at any time during the disease. There is rigidity of the muscles of the neck, with retraction of the head.

Stupor.—Stupor and unconsciousness are usually present.

Temperature.— This varies from 101° to 103°; it sometimes reaches 105°; before death it may reach 106° and 108°.

Pulse.—In some cases rapid, and in others slow—60, 50, and 40; before death it may be 160 or 180.

Respiration.—Just before death the Cheyne-Stokes respiration occurs. At other times it may be regular.

Eyes.—Pupils contracted and often unequal. In the later stages may be dilated.

Optic neuritis frequently occurs in meningitis at the base. It is due to an extension of inflammation to the membranes of the sheath and substance of the optic nerve.

Strabismus is a common and very important symptom, often transient at first, and never comes to stay. It is due to either paralysis or irritation of the ocular nerves.

Ptosis is often present: and also nystagmus.

Skin.—Hyperesthesia of the skin and special senses frequently occurs, so that the least touch, noise or light will cause extreme suffering and pain. It is due to the excitability of the brain. Drawing the finger nail across the skin causes a red line to appear, which is called "*tache cerebrale*" by Trousseau.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The symptoms above enumerated are usually clearly enough defined to diagnose this disease from others, and especially would the diagnosis be certain if some of the causes given, such as necrosis, abscess, or adjacent disease are also present. We have to distinguish mainly between simple meningitis and epidemic cerebro-spinal meningitis and tubercular meningitis. The presence of the purpuric spots, with the characteristic brain symptoms, will readily distinguish the cerebro-spinal form, while the history of tuberculosis or the apparent presence of tubercle will enable us to diagnose that form.

WHAT IS THE PROGNOSIS?

This depends upon the severity of the symptoms. If the fever is high, headache intense, delirium violent, followed by stupor, the prognosis is decidedly unfavorable. If the symptoms are mild, the prognosis would be more favorable. In any case where coma is present death is usually certain. Death may occur within forty-eight hours after the onset of the symptoms. In some cases, however, the onset is gradual, and when so the prognosis is more favorable.

EPIDEMIC CEREBRO-SPINAL MENINGITIS, OR SPOTTED FEVER.

WHAT IS EPIDEMIC CEREBRO-SPINAL MENINGITIS?

An inflammation of the membranes of the brain and spinal cord occurring in epidemic form.

WHAT ARE THE CAUSES OF THE DISEASE?

It occurs most frequently in persons under twenty, and males are attacked more frequently than females. Sometimes children are exclusively attacked. Poor sanitary conditions and overcrowding in the tenements in large cities are important causes. Undoubtedly some miasmatic or malarial influence induces the disease. While it is epidemic yet it is not contagious: but the miasmatic influences that produce it in one individual may also produce it in many others living in the same locality. It occurs most frequently during cold weather, when the people are crowded together more closely than at any other season of the year.

WHAT ARE THE SYMPTOMS?

General malaise and languor for two or three days preceding the active symptoms, which are very similar to those of the simple form of leptomeningitis, intense headache, delirium, vomiting, convulsions, fever, and extreme retraction and rigidity of the neck, in which there is intense exacerbation upon attempting to bring the head forward. The pain extends up into the head, down to the spine and into the extremities, and is increased by the slightest movement. There is also rigidity of the limbs and extreme hyperesthesia of the body.

The characteristic symptom of the disease is the presence of herpetic and purpuric spots upon different portions of the body, particularly upon the lower legs and forearms. They sometimes coalesce and form large patches of a purplish color.

WHAT IS THE PROGNOSIS?

It varies in different epidemics, but it is always serious. From thirty to eighty per cent. die. When coma occurs within a few hours of onset and the other symptoms are extreme death may take place within a few days, and sometimes within forty-eight hours.

TUBERCULAR MENINGITIS, OR ACUTE HYDROCEPHALUS.

WHAT IS TUBERCULAR MENINGITIS?

It is a form of meningitis due to the presence of the bacillus tuberculosis within the membranes of the brain.

WHAT ARE THE CAUSES OF TUBERCULAR MENINGITIS?

It occurs most frequently in children between two and ten years of age, and there is usually an hereditary history of tuberculosis. The presence of tubercle in other parts of the body predispose to it; also a scrofulous diathesis, unsanitary surroundings, trauma, and great mental excitement in tuberculous subjects.

WHAT ARE THE SYMPTOMS?

Loss of flesh, gradual wasting of strength, evening rise of temperature, restlessness, irritability and sleeplessness may exist for some time before the acute symptoms come on; these are severe headache, occasional convulsions, delirium, vomiting, fever, optic neuritis. There are also marked symptoms of compression of the brain, due to the increased amount of cerebro-spinal fluid within the ventricles and the sub-arachnoid spaces. The child soon passes into a comatose state and dies.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The presence of tuberculosis in other parts of the body, or a history of hereditary tuberculosis with the symptoms of meningitis, will enable us to diagnose the disease from any other without difficulty.

WHAT IS THE PROGNOSIS?

It is usually grave. Occasionally a patient dies of some other condition, and upon post mortem examination, evidences of tubercular meningitis have been found, showing that the disease had been arrested and the patient had apparently recovered from its effects. Death usually occurs within two or three weeks from the onset of the irritative stage.

CHRONIC HYDROCEPHALUS.

WHAT IS CHRONIC HYDROCEPHALUS?

An accumulation of fluid within the ventricles of the brain, when it is called internal, and in the sub-arachnoid spaces, when it is called external.

It is usually a congenital disease of infancy, but may be

acquired. The accumulation of fluid is due to defects of nutrition or to mechanical causes. When due to such defects or mechanical causes, such as obstruction of the veins or pressure of tumors, it is the internal form, the external form being usually inflammatory.

WHAT ARE THE SYMPTOMS?

Gradual enlargement of the head, with defective mentality and symptoms of irritation of the brain, due to the pressure of fluid within the ventricles. Sometimes the head is so large at birth that the fluid within the canal has to be expelled before the child can be born. There is bulging of the forehead over the face, which is preternaturally small, giving a senile expression; the occiput protrudes, the fon-

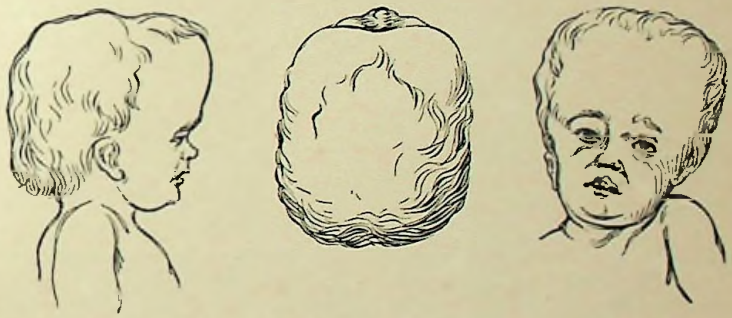


Figure 21.
Chronic Hydrocephalic Heads.

tanelles are wide open, and the head is sometimes so large that the child cannot hold it up on account of its weight. The body is usually small, due to lack of development, and the mental powers are much interfered with. Strabismus or optic atrophy are frequently present, convulsions, coma, and vomiting come on later, and the child may die of exhaustion.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The only condition which this is like is rickets: but in rickets the head is square and the enlargement is not so great: there are also evidences of disease of the bones in other parts of the body.

TREATMENT OF MENINGITIS.

WHAT IS THE PROGNOSIS?

Those cases which are congenital usually die early. Those developing within the first year or two of life may last for several years; while in mild cases the disease may become arrested and the patient develop mentally and physically and live a useful life.

WHAT IS THE TREATMENT OF MENINGITIS?

GENERAL.—Rest, both of mind and body, is the most important of all. The room should be darkened and kept absolutely quiet, on account of hyperesthesia of sight and hearing.

LOCAL.—If the disease be caused by trauma surgical methods may be required. Applications of cloths wet in cold water, not ice water, all over the head, with hot water-bottles at the feet, will help to equalize the circulation and relieve pain.

DIETETIC.—This is extremely important. In severe cases nourishment must be given frequently and in small quantities. Milk is the main article of diet when the patient cannot swallow easily; it may be given iced. If there is not much fever beef juice, every hour, in teaspoonful doses, may be given. When fever is high all meat extracts or broths should be avoided. Oatmeal or rice boiled three or four hours and strained through a cloth, and given either hot or cold, as the patient may desire, is good. Water may be given in as large quantities as the patient wishes. As convalescence becomes established milk toast, farina, blanc mange, scraped beef, pancreatised meat-broth, eggs, custard, and wine jelly are useful.

REMEDIAL.—*Aconite.*—When due to exposure to heat. Chill followed by fever; full, bounding pulse; restlessness; anxiety; intense burning pains through the head; face red and puffed; sensation as if the brain were in boiling water; temperature 103° or higher; skin hot and dry.

Apis.—When due to suppression of some of the exanthemata. Sopor interrupted by piercing shrieks; muttering delirium; congestion of head and face; squinting of the eyes; grinding of the teeth; boring head in pillow; one

side twitching, the other paralyzed: head wet from sweating: photophobia and diplopia: dry, burning skin which grows gradually cool in places; violent fever.

Belladonna.—In early stages. Intense, congestive, throbbing headache, with cold feet: excessive nervous excitability, the least noise or light aggravating intensely; head feels full of blood to bursting: stabbing as with a knife from one temple to the other: general convulsion: inclination to bite: violent delirium, alternating with coma; suppression of urine, with involuntary micturition: boring of head in pillow, and head drawn backward: distortion of eyes, and redness of conjunctivæ.

Bryonia.—When there is effusion. Intense headache causes the child to scream from the slightest motion: face dark and congested; partial loss of consciousness, with constant chewing motion: must lie perfectly quiet, and does not want to be touched.

Cuprum metallicum.—When due to metastasis of exanthemata. Violent epileptiform convulsions: vomiting of watery substance from the stomach: intense thirst; cold water temporarily prevents vomiting: great heat of head while child is in deep sopor: twitching and working of limbs: coldness of hands and blueness of fingers.

Gelsemium.—At the very onset of the disease. Severe chill, followed by congestion of the brain and spinal cord; dilated pupils; thirstlessness; great exhaustion: staggering gait; dullness of speech: icy cold hands and feet: weak, hardly perceptible, laborious respiration: involuntary closing of eyelids in spite of all he can do: sweating relieves: mental faculties retain their activity though power over muscles is impaired: sometimes coma.

Helleborus.—Total unconsciousness, cannot be aroused; neck rigid, and head drawn far back: dilated pupils, insensible to light: eyes staring and wide open: constant moving of one arm and one foot: grinding of teeth: constant picking of lips and clothes.

Hypericum.—When due to trauma, and from the effects of nervous shock, concussion of the brain and spine. Vertebræ sensitive to touch: headache as if the brain would be torn to pieces, after a fall upon the occiput: pressive pain in the occiput upon motion: great dread of the slightest motion.

WHAT REMEDIES ARE OF SPECIAL VALUE IN TUBERCULAR MENINGITIS?

Artemisa vulgaris.—Complete unconsciousness; piercing shriek: turns the eyes with violent clonic spasms; left side paralyzed, when the right is in a state of clonic spasm: drinks large quantities of water without being entirely aroused: head bent backwards and sideways.

Baryta carbonica.—The child has a large head, thin, scrawny neck, with an apparently scrofulous diathesis; great mental and bodily weakness; child does not want to play: pressure in brain under vertex, towards occiput: tendency toward glandular enlargements, with hacking cough.

Calcarea carbonica.—In children with distended abdomen, wasted limbs, glandular enlargements, headache and sweating of head during sleep; weakness of memory: mental anxiety.

ANEMIA OF THE BRAIN, OR CEREBRAL ANEMIA.

WHAT IS ANEMIA OF THE BRAIN?

A deficiency of the quantity of blood within the brain. It may affect part of the brain only, or the whole brain, and may be sudden or gradual in its production.

WHAT ARE THE CAUSES?

General cerebral anemia may be produced when there is a deficiency of the quantity of blood in the whole system, due to hemorrhage or exhausting discharges; weakened heart's action; exhaustion after protracted diseases; excess of cerebro-spinal fluid in the brain, and cerebral tumors.

Partial cerebral anemia may be due to obstruction of the bloodvessels by an embolus, thrombosis, or pressure of tumors; narrowing of the calibre of the vessels by syphilis and vaso-motor spasm.

WHAT IS THE PATHOLOGICAL ANATOMY OF THE ANEMIA?

There is pallor of the brain, which may be partial or general. The membranes are pale, and there is usually effusion of serum in the pia mater and between the convolutions; edema of the brain sometimes, and degeneration of the walls of the vessels.

WHAT ARE THE SYMPTOMS OF ANEMIA?

When sudden, the patient feels drowsy, faint, with a cold perspiration upon the surface of the body, dullness of the special senses, ringing in the ears, vertigo, muscular weakness, pallor, nausea, and sometimes loss of consciousness: pupils at first contracted, but afterwards dilated.

When the anemia comes on slowly there is a general weakness of the whole body, inability to concentrate the mind, sleeplessness, irritability, headache, tinnitus aurium, vertigo, all of which are made decidedly worse when the patient is in the erect position. The only comfort he can have is when he is lying down.

Partial anemia causes loss of function in the part affected, and death of tissue will result if it be present.

WHAT IS THE PROGNOSIS?

This depends upon the severity of the symptoms and their causes. If due to exhausting diseases the brain will recover as the patient improves in general. When due to embolus or thrombosis collateral circulation will sometimes relieve the anemia in a measure and the patient will recover.

WHAT IS THE TREATMENT?

GENERAL.—Lay the patient in the recumbent position with the head lower than the rest of the body. If any fatal termination seems imminent, inhalations of nitrite of amyl will be beneficial. It is far better than any alcoholic stimulant. Inhalations of camphor or ammonia may also be used. Dashing cold water in the face is often of use.

REMEDIAL.—*Aconite.*—Fainting as soon as the patient raises his head from the recumbent position: deathly paleness of the face; chilliness; violent palpitation of the heart.

Camphora.—Icy coldness of the surface of the body, with sudden and extreme prostration; redness of the face when lying down, deathly pale if the patient raises up; lassitude and depression of spirits, with frequent yawning and stretching.

Carbo vegetabilis.—Fainting after sleep while yet in bed in the morning, or after rising, caused by debilitating losses, such as blood, seminal fluid or excessive diarrhea.

Cinchona officinalis.—Faintness after loss of animal fluids; head feels weak, can hardly hold it erect; faintness on rising, as if he would fall backwards; head inclined to sink backwards; on waking at night he does not dare to rise lest he might faint; loss of sight; ringing in the ears; cold surface after hemorrhages.

Lachesis.—Tendency to faint, in women; apparent death, neither pulse nor breathing perceptible, after great fright or grief; headache over eyes and in occiput in the morning on rising, with faintness.

Laurocerasus.—Long-lasting faints; no reactive power; bluish tint of skin; rapid sinking of forces; restless sleep, gasping for breath.

HYPEREMIA OF THE BRAIN, OR CEREBRAL CONGESTION.

WHAT IS HYPEREMIA OF THE BRAIN?

An excess of blood within the brain. It may be either active or passive, according as it is due to the increase of arterial or venous blood.

WHAT ARE ITS CAUSES?

(a). Active congestion may be caused by over-action of the heart; contraction of the arterioles in other parts of the body, due to sudden exposure to cold or during a chill; dilatation of the vessels of the brain which is produced by various toxic agents, such as nitrite of amyl, nitro-glycerine and alcohol; great mental excitement; worry; over-action of the brain; vaso-motor paralysis of cerebral vessels from general nerve exhaustion; exposure to heat, as in sunstroke, which is really the result of over-heating of the body and not of the action of the sun upon the head.

(b). Passive congestion is due to a mechanical obstruction of the vessels which prevents the return of blood to the body; dilatation of the heart; hyperemia of the liver; obstruction to the flow of blood through the lungs, which produces coughing; playing upon wind instruments; severe muscular exertion, as in lifting or straining at stool; pressure of tumors upon cerebral vessels; suffocation and strangulation.

WHAT IS THE PATHOLOGICAL ANATOMY IN HYPEREMIA?

There are really no signs of any cerebral hyperemia observable after death. Large quantities of blood may have been in the brain before death but do not remain there after life is extinct.

WHAT ARE THE SYMPTOMS?

Sleeplessness; great mental excitement, which may go on to mania; flushed face; eyes injected; head hot; acuteness of the special senses; throbbing headache; delirium; screams and tears his clothes, but there are no actual delusions; temperature may be raised to 103° or higher. Such attacks last only a few hours. Some attacks come on similarly to those of apoplexy, in which the patient suddenly falls to the ground in unconsciousness. There is a transient hemiplegia which passes away in a few days; convulsions may also be present. This is called congestive apoplexy.

In mild cases of cerebral hyperemia there is simply a dull, full feeling in the head; drowsiness during the day when the patient is sitting up, but sleeplessness at night when he is lying down; vertigo on stooping over; head hot; burning face; cold feet; patient irritable and over-excitable; twitching of the muscles in different parts of the body; numbness and weakness of the limbs; veins of the neck and face prominent; carotids throbbing. Bleeding of the nose is of frequent occurrence and relieves some.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The principal disease which may be mistaken for cerebral hyperemia is cerebral hemorrhage or apoplexy. In the latter condition the hemiplegia persists, while in hyperemia it passes away in a few days. Consciousness is not impaired to such a degree in cerebral hyperemia as in cerebral hemorrhage. In mild cases of hyperemia the symptoms given above, when present, are sufficiently clear to enable us to make a diagnosis.

WHAT IS THE PROGNOSIS?

Life may be endangered by the intensity of the congestion; organic changes in the brain may result from com-

plete congestion. The majority of cases are transient, and are relieved when the causes which produced them are removed.

WHAT IS THE TREATMENT?

GENERAL.—Rest of mind and freedom from all harassing occupations; excitement of all kinds should be avoided; change of scene and an indolent, lazy life should be recommended upon the first appearance of the symptoms, with long hours of sleep and rest.

DIETETIC.—All stimulants, such as coffee, tea, alcohol and tobacco should be absolutely prohibited; all spiced foods, sweets and pastries should also be avoided; over-eating and late suppers should be forbidden: meat should not be eaten oftener than once a day, and the white meat of poultry and broiled fish are better than red meat; milk may cause indigestion, and hence should be taken with care; fresh, green vegetables are good, but corn, cabbage and tomatoes should be avoided.

REMEDIAL.—*Aconite.*—Active congestion; full, bounding pulse; burning in the interior of the head, with pale, cold face, covered with perspiration; great restlessness; thirst for large quantities of water; delirium; especially applicable in cases which have been produced by exposure to the sun.

Belladonna.—Excessive, nervous irritability, with exalted sensibilities of all the organs; the least noise or light is annoying; great irritability; headache as if the brain would be pressed out in the forehead, which prevents sleep; throbbing of carotids; intense congestion of the head, better when sitting up, worse when lying down; pain in the head and eye-balls; eyes feel as if starting from their sockets; starting on falling asleep; sleepy while sitting, wakeful when lying down; convulsions and congestive apoplexy.

Coffea.— Full of apprehension of terrible things happening; cannot get to sleep because of ideas perpetually forcing themselves upon the mind; general nervous excitement.

Glonoinum.—In congestive apoplexy, due to sunstroke; head feels full to bursting; sensation as if the head were

hanging downwards, and that there was a great rush of blood to the head in consequence: head feels large: nausea, then unconsciousness, with convulsive action of the facial muscles; face pale; breathing stertorous; feeble pulse.

Hyoscyamus.—Is indicated in the milder forms of cerebral congestion with symptoms similar to those of belladonna, but in a milder degree.

Nux vomica.—When due to excessive mental work or worry, with loss of sleep; abuse of stimulants, such as alcohol, tobacco; irritable, morose, sullen.

Opium.—Passive cerebral congestion, with somnolency after meals, in persons predisposed to apoplexy; fainting turns, with vertigo whenever attempting to rise from the bed; great heaviness of head, making thought and writing difficult; congestion of blood to the head, with pulsation in it; coma; incomplete insensibility; will be aroused for a moment when addressed in a loud tone of voice and then relapses into stupor; sterterous breathing; bluish or livid face.

APOPLEXY.

WHAT IS APOPLEXY?

It is a condition which is characterized by sudden shock, paralysis, loss of consciousness, usually due to rupture of a bloodvessel, or a stoppage of the circulation of blood through a bloodvessel in the brain. The term apoplexy comes from the Greek and means, "*to strike down.*" It has always been used synonymously with cerebral hemorrhage; but now the pathological condition which produces the apoplexy is used instead of that term. Cerebral hemorrhage, cerebral embolism, and thrombosis of a cerebral vessel are the pathological states which produce this condition.

CEREBRAL HEMORRHAGE.

WHAT IS CEREBRAL HEMORRHAGE?

A hemorrhage into the brain substance, due to rupture of a cerebral vessel.

WHAT ARE ITS CAUSES?

(1). The diseased condition of the vessel which predisposes to the rupture, such as a weakening of the walls of the

vessel, which produces miliary aneurisms; atheromatous degeneration of the vessel, which consists of the formation of yellowish patches composed of fat granules on the under surface of the elastic coat of an artery, and which subsequently assumes the consistency of gruel; syphilitic disease of the artery, producing weakness of the coats of the vessel; endarteritis.

(2). The causes producing these pathological conditions, namely, hereditary predisposition; sex, males suffering more frequently than females; age, rarely occurs under forty, usually in the degenerated period of life; hemorrhagic diathesis; more common in warm climates than cold; excessive indulgence in alcohol; Bright's disease of the kidneys.

(3). The immediate causes of the rupture. which may be great mental excitement, over-work, loss of sleep, lifting heavy weights, violent coughing, the sexual act, over-eating, straining at stool, great muscular exertion, such as running, or from recumbent position.

WHAT IS THE PATHOLOGICAL ANATOMY OF CEREBRAL HEMORRHAGE?

In most cases there is only one hemorrhage. Sometimes there are two or more, one of which is larger than the other. After the hemorrhage takes place a clot is formed which may vary in size from that of a nut to that of an egg. The greater part of one hemisphere may even be torn up, and all of the ventricles distended. The two hemispheres are about equally affected as regards frequency.

The parts of the brain which are the most frequent seats of hemorrhage are the corpus striatum, the centrum ovale, the cortex, pons, and the cerebellum. Hemorrhage into the medulla oblongata and the crus cerebri occasionally occurs.

In the cerebral substance we find a cavity varying in size, formed by the laceration of brain tissue. At first the extravasated blood is red, but as the clot forms it becomes reddish-black in color, with fragments of brain tissue mixed with it. The cavity is usually irregular in shape and the brain tissue around it softened. Sometimes the blood tears its way into the lateral ventricles and soon distends the third and fourth. About the twentieth day the clot begins to shrink; it becomes first a chocolate, then brownish, and

finally a reddish-yellow color, containing fat globules, pigment and other granules. The walls of the cavity also undergo a change. The mild degree of inflammation which is usually present leads to the formation of connective tissue which lines the walls, and a firm wall is thus developed. Connective tissue bands may extend across the cavity, uniting these walls and forming a cicatrix. The middle cerebral artery is the one most frequently ruptured.

WHAT ARE THE SYMPTOMS OF CEREBRAL HEMORRHAGE?

Premonitory symptoms may sometimes be present but they are rare. They consist of headache, slight vertigo, weakness or tingling in the limbs, slight mental changes, and sometimes a thickness of speech. These symptoms come on occasionally, lasting a few hours or days and then passing away.

The onset of the attack may be sudden or gradual, depending upon the rapidity with which the blood escapes from the vessel. The position of the clot will determine the character of the initial symptoms. Clots in the medulla, pons, or cerebellum will cause the patient to fall to the ground without warning as if struck by a blow. A hemorrhage into the ventricles will do the same, and may sometimes produce instant death. In the majority of cases there is present

Loss of Consciousness.—This may last from a few moments to several hours. In mild cases there may only be a slight mental confusion or dullness. The unconsciousness may go on to complete coma, with muscles relaxed and flaccid, urine and feces escaping, and reflex action abolished, not only in the limbs, but in the conjunctiva and iris as well. With this loss of consciousness there are occasionally

Clonic Convulsions.—These are likely to be accompanied by paralysis of the arm, leg and face, of the opposite side from the lesion. Whenever convulsion is present it is usually owing to the fact that the hemorrhage has passed into the cortex, although it may occur when the hemorrhage is into the corpus striatum.

Respiration.—The breathing is of a labored character, with puffing and blowing out of the cheeks, accompanied

by a snoring sound called stertor. In this state the patient may die in a few hours after the onset. Whenever Cheyne-Stokes respiration is present death is sure.

Pulse.—The pulse is generally slow and often small and incompressible.

Face.—The face may be flushed and turgid, or pale and pinched, the surface usually being covered with perspiration.

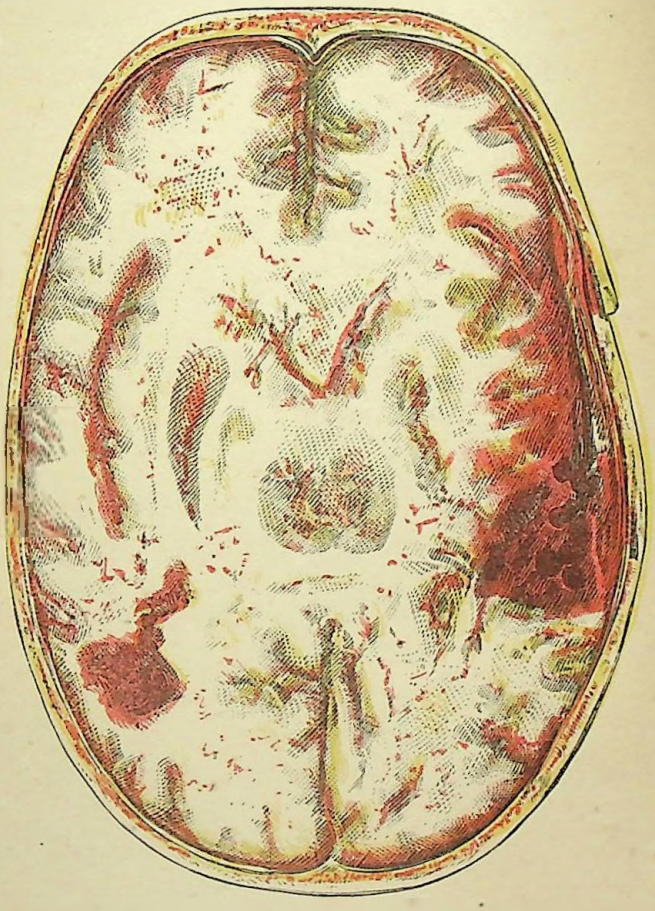
Vomiting. Emesis may occur but it is more frequent when the hemorrhage is in the cerebellum.

Temperature.—This generally falls within an hour or two after the onset from one to three degrees. In cases which are fatal within twelve hours the temperature may fall steadily until death. Whenever there is considerable rise of temperature within a few hours it is of evil omen. In mild cases there is usually only a slight rise of temperature.

These symptoms gradually pass away until only a paralysis of one side of the body is left, opposite to that of the lesion. After a time the paralysis, too, gradually passes away, the lower extremities improving faster than the upper extremities, until in some cases there is but little of it remaining. In the majority of cases there is a secondary sclerosis of the pyramidal tracts, which produces rigidity, contractures and exaggerated reflexes in the members affected. There is also a blunting of the mental sensibilities, and the patient is a wreck of his former self.

Sensory Symptoms.—There are sometimes certain sensory symptoms, usually hemianesthesia. Anesthesia is not profound, as a rule, and the sensory symptoms may be limited to a sense of numbness and formication. Sensibility to touch is more affected than that of pain.

Trophic Disturbances.—There may be persistent elevation of temperature in the paralyzed parts, with reddening and wasting of the muscles: the skin may become edematous and the limb appear swollen. Sweating of the paralyzed limb is observed in some cases: bed-sores are developed: the nails become yellowish and disfigured with ridges: the hair of the paralyzed parts grows thick and dark, and the joints become inflamed.



Horizontal section of cranium showing depressed fracture of skull, with extra-dural, sub-dural and interstitial hemorrhage.

Number 1, extra-dural clot; 2, laceration of brain substance, with large intra-cerebral clot; 3, same condition from contre coup. Punctate hemorrhages and minute lacerations at numerous points. Characteristic of contusion of brain. Modified after Anger.

PLATE V.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

This is often difficult. Coma may be due to so-called congestive apoplexy, the effects of alcohol, opium, uremia, cerebral embolism, cerebral tumors, and compression of the brain from compressed bone. In the three latter conditions there is usually paralysis with the coma.

Cerebral Embolism.—Embolism has no prodromal symptoms: the coma and the paralysis are not so profound, and usually begin to improve within a short time. Embolism may occur at any age: hemorrhage usually after forty. The slow pulse, stertorous breathing and irregularity of the pupils point to hemorrhage.

Cerebral Tumors.—The symptoms come on gradually and are less severe. There are also choked disk, spasmodic conditions, and neuralgias present during the development of the tumor.

Uremia.—There are albumen and casts in the urine, edema and general anasarca, and there is no paralysis.

Alcoholic Coma and *Coma of Opium.*—There will be an absence of paralysis.

WHAT IS THE PROGNOSIS?

It is most grave when the coma is prolonged, the temperature rises to an extreme degree after an attack, when symptoms of pulmonary edema are developed, when there are paralysis of the sphincters, prolonged and frequent convulsions and extreme dilatation of the pupils.

It is favorable when the coma is absent or mild and of short duration: when convulsions are only slight and when the paralysis is not complete.

WHAT IS THE TREATMENT?

GENERAL.—The patient should be placed in the recumbent position, with the body raised to an angle of 45°: hot-water bottles should be placed at the feet, and cloths wet in ice water applied to the head, if collapse is not present. Care should be taken to avoid any constriction of the clothing about the neck. If stertor be present the patient may be turned on the paralyzed side, thus stopping the stertor. Absolute rest and quiet should be enjoined, the

room darkened and friends kept out. If the bowels are loaded a high enema should be given to cleanse them as thoroughly as possible.

Dietetic.—No food of any kind should be given for from twelve to twenty-four hours after the attack. Cold water may be given if the patient wishes it, or the mouth may be frequently wet with cold water in case the patient is unconscious, as the mouth becomes dry. Milk, egg and milk, made palatable with sugar and nutmeg, is first to be given as soon as the patient cares for food; a little every two or three hours is better than larger quantities at greater intervals. The liquid food is best given in teaspoonful doses, care being taken that the patient swallows one spoonful before another is given, so that he does not choke. Nutriment enemata may also be given when there is paralysis of the muscles of deglutition. Thick broths, long-boiled rice, sago, barley with cream, custards and soft-cooked eggs may be given as the patient is convalescing, always being careful that the patient is not over-fed. *Never give alcoholic stimulants.*

Remedial.—*Belladonna.*—Flushed, hot, bloated face; dilated pupils; fixed look; nausea; stertorous breathing; grinding the teeth; mouth drawn to one side; difficulty in swallowing; unconsciousness; convulsive movements of the limbs and muscles of the face; paralysis of the extremities, especially of one side; thickness of speech; staggering gait; throbbing headache; tired feeling in the limbs; weak memory; stiffness of the tongue.

Gelsemium.—Threatened or actual apoplexy, with stupor, coma, and usually general paralysis; headache with nausea; lightness of the brain; giddiness; intense passive congestion to the head, with nervous exhaustion; weakness and trembling throughout the whole system; muscles will not obey the will, feel bruised; pricking, tingling and crawling; complete relaxation of the whole muscular system.

Glonoinum.—Intense cerebral congestion; vertigo, intoxication and heaviness of the head when bending the head forward; reeling; trembling; falling; headache; heat in the head; redness of face; photophobia; injection of conjunctivæ; flickering before eyes; buzzing in the ears;

pulsation of carotids: nausea, then unconsciousness, with convulsive action of the facial muscles; face pale: breathing stertorous: feeble pulse.

Hyoscyamus.—Sudden falling down with a shriek: soporus condition; face red: lower jaw dropped: twitching of muscles: stertorous breathing: inability to swallow: involuntary stool; pulse quick and full: bloodvessels swollen: numbness of hands after consciousness returns.

Lachesis.—Left-sided apoplexy, especially after mental emotions or abuse of alcohol; blowing expiration: stupefaction or loss of consciousness; blue face: convulsive movements: tremor of extremities.

Opium.—Coma: incomplete insensibility; extremities and face bluish or livid: loud, stertorous inspiration, coldness of skin: congestion to head, with great roaring in the ears; eyes open: pupils dilated; tongue drawn to one side: speech impeded: inability to swallow: limbs cold and paralyzed; tetanic stiffness of the whole body.

Veratrum viride.—Congestive apoplexy: feeling as if the head would burst open, with nausea and vomiting: ringing in the ears: blood-shot eyes: thick speech; hot head; pulse full, slow and hard as iron: convulsions.

Arnica.—To be used after the active symptoms have passed away, to promote absorption of the clot: forgetful; what he reads quickly escapes his memory; thinking tardy; excitable and timid: indifference to everything: morose; easily frightened: dizziness, with sickness of the stomach: vertigo when shutting the eyes.

Causticum.—For the paralytic states remaining after the apoplexy has been removed: paralysis and contracture of the lower extremities; paralysis and trembling of the hands; sensation of fullness in the hand when grasping anything: numbness of feet and toes: paralytic weakness of limbs: hands and feet go to sleep.

INFANTILE MENINGEAL HEMORRHAGE, OR CEREBRAL BIRTH-PALSY.

WHAT IS MEANT BY INFANTILE MENINGEAL HEMORRHAGE?

A hemorrhage occurring in the meninges of the brain, usually during birth, which causes destruction of brain tis-

sue and produces permanent symptoms remaining during life.

WHAT ARE ITS CAUSES?

Usually due to difficult birth, when the presentation is unnatural, the head being born last. The intense mechanical congestion produced by the compression of the neck in these cases induces the hemorrhage. In head presentations when there has been a tedious, difficult labor due to a large head in a small parturient canal, when the compression of the head is accordingly very great; also in cases where forceps have been used awkwardly in delivering the head, producing great compression. Violent extension of the head just after its birth sometimes produces it.

WHAT IS ITS PATHOLOGICAL ANATOMY?

Extravasation of blood, sometimes over the convexity of the brain and sometimes at the base. When on the convex surface, it is usually bilateral. It is sometimes found in the median surface of the brain. Atrophy of the convolutions, due to long-continued compression by the blood, is present in older children. The thickest part of the extravasation is at the motor region. The cerebral tissue in various parts is sometimes injured, being broken up and infiltrated with blood.

WHAT ARE THE SYMPTOMS OF INFANTILE MENINGEAL HEMORRHAGE?

In the majority of cases nothing is noticed the first few days of life, until—

(a). Convulsions, usually either general or bilateral, manifest themselves, after which

(b). Paralysis of one or more members is observed. There is also difficulty in holding the head up; but these paralytic symptoms may not be noticed to any degree until the patient is four or five months old, when they become more manifest. The paralysis may pass away in a measure, and only weakness of the member be left.

(c). Spasm of the weakened muscles, producing flexion of the elbow or of the wrist and fingers. Flexion of the leg

at the knee, and of the foot may be present, preventing the patient from walking.

(d). Inco-ordination of the affected members is usually present.

(e). Mental weakness is manifested as the child grows older, and also defective articulation and sometimes permanent inability to speak: idiocy, in which there is also difficulty of swallowing and drooling of saliva.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

In cases where paraplegia is present it may be attributed to a spinal cord lesion, but there will usually be noticed some defect in the movements of the arms and hands, and when it is, remember that primary chronic spinal cord disease is almost unknown in young children.

From disease of the brain before birth infantile meningeal hemorrhage can be diagnosed from the fact that there have been no noticeable initial symptoms after birth to produce the paralysis.

In tumor of the brain the symptoms come on gradually.

WHAT IS THE PROGNOSIS?

The damage to the brain tissue is permanent and so are its effects. If there is not much mental defect the child, as it grows older, may acquire considerable movement in the weakened members and to some extent overcome the influence of the spasm. The mental condition itself will also gradually improve, but the child will never become as strong and well as other children.

WHAT IS THE TREATMENT?

This can only be general, as drugs will have no effect in reconstructing destroyed tissue. Whatever symptoms, either special or general, may arise during the first years of life should be met by the properly indicated homeopathic remedy so as to alleviate as much as possible the sufferings of the little patient. When convulsions occur they should be treated as the convulsions of epilepsy, for which see remedies under that disease.

CEREBRAL EMBOLISM.

WHAT IS CEREBRAL EMBOLISM?

Occlusion of a bloodvessel in the brain by a plug called an embolus, coming from a distant part of the body and carried into the vessel by the blood. This plug, in most cases, comes from the heart and consists of fibrin or other foreign material detached from it.

WHAT ARE ITS CAUSES?

Endocarditis and endarteritis, which lead to the formation of fibrinous deposits upon the valves of the heart, are the primary causes of embolism. Great emotional excitement, which produces increased action of the heart, may be the exciting cause which precipitates the attack by detaching these fibrinous deposits and sending them into the circulation to be lodged in a cerebral artery. A history of acute rheumatism which produced the endocarditis is usually present. It occurs in about equal frequency in men and women and between the ages of twenty and fifty, though it may occur at any time of life.

WHAT IS THE PATHOLOGICAL ANATOMY?

The most common seats of embolism are the middle cerebral arteries and their branches. The next most common seat is the posterior cerebral and then the vertebral arteries. (Figs. 2 and 3). The first effect of occlusion of a vessel is to cut off the blood-supply to the part of the brain to which it is distributed. There is arterial anemia, but distension of the capillaries with blood from the veins, the capillaries giving way in places and producing fine hemorrhagic points. After twenty-four hours the consistency of the brain-tissue rapidly lessens, the nerve-cells and fibres soon break down and serum is effused between the fragments so that softening quickly takes place.

WHAT ARE THE SYMPTOMS?

There are no premonitory symptoms.

Aphasia.—This is the first symptom in mild cases, and may be complete for an hour or two and then gradually be-

gin to pass away, though it may not wholly disappear for some months.

Loss of Consciousness.—This may occur when a large artery is suddenly occluded, but it usually passes away in a few minutes or a few hours.

Convulsions.—These are not as severe as in cerebral hemorrhage, and may consist of only convulsive twitchings of different members of the body without a loss of consciousness.

Paralysis.—Paralysis is usually complete for a few hours or days in one or more members. It is generally either a monoplegia or a hemiplegia. The muscles of deglutition and of speech may also be affected.

Temperature.—The body heat may at first be slightly raised, but in a few days fever is liable to develop.

Vomiting.—Emesis may be present but is not a common symptom.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

Cerebral Hemorrhage.—The diagnosis from brain hemorrhage is often difficult. In embolism the coma is not usually as profound, there are no premonitory symptoms, and the paralysis begins to improve more rapidly than in hemorrhage. There is also frequently a co-existence of valvular lesion of the heart or aneurism of the aorta.

Cerebral Thrombosis.—The suddenness of the onset, the rapid development of aphasia, the absence of prodromal symptoms, the presence of heart trouble, the absence of atheromatous changes in the vessels will readily differentiate embolism from cerebral thrombus.

WHAT IS THE PROGNOSIS?

In favorable cases all symptoms begin to pass away within a few days, and the patient may go on to complete recovery on account of the establishment of collateral circulation in branches of the occluded artery, which supplies the part of the brain that had been deprived of its nourishment.

In severe cases death may take place in a few hours. If the paralysis does not begin to improve within forty-eight hours, the patient is not likely to wholly recover, as cerebral

softening has probably commenced. Abscess of the brain may also be a result of embolism.

The treatment is similar to that of cerebral hemorrhage.

CEREBRAL THROMBOSIS.

WHAT IS CEREBRAL THROMBOSIS?

It is an obliteration of the calibre of an artery in the brain by a fibrinous deposit formed at the seat of obstruction.

WHAT ARE ITS CAUSES?

It occurs most frequently between fifty and seventy years of age, or during the degenerated period of life. The main causes are a roughening of the walls of the artery, due to atheroma or syphilitic endarteritis; hyperinosis, an abnormal abundance of fibrin in the blood; pressure upon a bloodvessel so that the circulation within it is rendered extremely slow; chronic interstitial nephritis and pyemia.

WHAT IS THE PATHOLOGICAL ANATOMY?

The most common seats of thrombosis are the internal carotid, middle cerebral, basilar, vertebral, and posterior cerebral arteries. The walls of the artery gradually become thickened, until finally the calibre of the vessel is completely occluded. The condition of the brain from thrombosis is similar to that in embolism.

WHAT ARE THE SYMPTOMS?

Premonitory symptoms are headache, which is generally dull, with heaviness of the head; giddiness; tingling, numbness and paresis of one-half the body, sometimes confined to one limb; weakness of memory and irritability due to lack of nutrition of the brain.

When occlusion has taken place the symptoms are similar to those of embolism.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The time of life in which degeneration of arteries occurs, the evidences of syphilis, the premonitory symptoms, which

gradually increase, followed by weakness and paralysis of the limbs, would lead us to diagnose it as cerebral thrombosis.

Cerebral hemorrhage and cerebral embolism both occur suddenly; and coma is a symptom of hemorrhage but is not of thrombosis.

WHAT IS THE PROGNOSIS?

Usually unfavorable. Cases due to syphilis may sometimes recover.

The treatment is like that of cerebral hemorrhage.

CEREBRRAL SOFTENING, OR ENCEPHALOMALACIA.

WHAT IS CEREBRAL SOFTENING?

It is a necrosis of brain tissue.

WHAT ARE ITS CAUSES?

Cerebral embolism, cerebral thrombosis, cerebral hemorrhage, inflammation of the brain, cerebral anemia and senility.

WHAT IS THE PATHOLOGICAL ANATOMY?

There are three varieties of softening, red, yellow and white, depending upon the amount of blood effused into the softened area. The most frequent seats of softening are the cerebral cortex, corpus striatum, and the optic thalami. There is more or less edema into the brain substance. The softened tissue will be found to consist of fat-granules, altered blood-corpuscles, pus-cells, disintegrated nerve-tissue, and caseous matter. The red variety will exhibit a large admixture of blood-corpuscles and pigment-granules. The yellow and white varieties have an excess of fatty matter or caseous substance, and a small admixture of altered blood-pigments.

WHAT ARE THE SYMPTOMS?

When due to embolism or thrombosis we find the symptoms of these conditions, and they may be called symptoms of the acute stage of softening.

The chronic stage manifests itself by loss of mental power, headache, more or less continuous; an inability to articulate properly, with a tendency to clip off words during conversation; an inability to maintain continuous muscular contraction; excitability upon the least provocation; lack of personal cleanliness; groundless prejudices, and all the symptoms of mental deterioration. Paralysis of motion may develop gradually, commencing in fingers and toes first and steadily advancing toward the trunk. This is sometimes called creeping palsy. Difficult deglutition; paresis of ocular muscles; and disturbances of hearing, smell and taste: hemianopsia and word blindness may exist.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The gradual loss of mental and physical power after an attack of cerebral embolism, thrombosis or hemorrhage, will enable us to diagnose this disease from any other.

WHAT IS THE PROGNOSIS?

Always grave, the patient gradually deteriorating, both mentally and physically, day by day. By careful treatment, both hygienic and remedial, these patients may live for several years, but are unfortunately a burden to themselves and everyone around them.

WHAT IS THE TREATMENT?

GENERAL.—Hygienic.

DIETETIC.—Ordinary, plain, wholesome food.

REMEDIAL.—Homeopathic remedies, according to the symptoms as they arise. The mental symptoms being the most prominent will present indications for the remedies.

Abrotanum.—Feebleness and dullness of mind; no capacity for thinking, as if all bodily and mental power were gone; excited, loquacious, feels like shouting, exceedingly peevish; aversion to physical exercise; irritable; violent; easily fatigued by conversation or mental effort.

Anacardium.—Loss of memory after general paralysis; cannot remember anything about his previous state; imbecility; irresistible desire to curse and swear; when walking

he feels anxious as if someone were pursuing him; suspects everyone around him: anxiousness; despairs of getting well; despairs of being able to do that which is required of him; feels as though he has two wills, one commanding him to do what the other forbids; a slight offense makes him excessively angry.

Arsenicum.—Fear of being left alone; dread of death when alone on going to bed; anxiety and restlessness worse after night; great fear; restlessness; trembling; cold sweat; prostration; very cross and despondent; vexed about trifles; cannot rest anywhere, moves from place to place; head feels confused whenever it is moved; exhaustion from the slightest exertion, must lie down; very rapid sinking of strength.

Digitalis.—Thinking difficult; forgets everything immediately; lascivious fancies day and night; profound melancholy, worse by music, with frequent sighing and weeping which bring relief; gloomy; morose; ill humor; great fear of the future; insane obstinacy and disobedience; great anxiety as of a troubled conscience; lassitude, mental and bodily; faintness; exhaustion; extreme prostration; coldness of limbs; irregular, small pulse.

Phosphorus.—Apathy; indifference; answers no questions, or replies wrongly; takes no notice of things about him: answers slowly; moves sluggishly; weary of life: full of gloomy forebodings; dementia paralytica; weak, exhausted; silliness; idiocy.

Picric acid.—Great indifference; lack of will power; disinclination for mental and physical work; desire to sit still without taking any interest in surroundings; mental prostration after the least intellectual work; cannot collect thoughts; quickly prostrated from using the mind.

THROMBOSIS IN THE CEREBRAL SINUSES AND VEINS.

WHAT IS MEANT BY THROMBOSIS IN THE CEREBRAL SINUSES AND VEINS?

A coagulation of blood in the sinuses or veins of the brain, which may cause mechanical congestion, edema, capillary hemorrhage, and occasionally softening in the

parts of the brain from which the blood should be conveyed by the vessels occluded.

WHAT ARE THE CAUSES?

The state of the blood and circulation generally, in consequence of disease near the sinuses. The first is called primary, and the latter secondary thrombosis.

Primary thrombosis occurs as a complication of marasmus, diarrhea or other exhausting disease in children, and is hence often termed marantic thrombosis. In adults it occurs in the last stages of tuberculosis, cancer and general malnutrition. It occurs in children up to fourteen years of age, but most frequently during the first six months of life. Any prostrating disease, such as pneumonia, long-continued suppuration or acute specific diseases may produce it.

Secondary thrombosis occurs as the result of caries of the bone, especially in diseases of the internal ear; fracture of the skull, when attended by inflammation of the diploe; meningitis; compression of a sinus by tumor; erysipelas outside of the skull; carbuncle of the neck, and suppurating eczema of the scalp.

WHAT IS THE PATHOLOGICAL ANATOMY?

The sinus affected is usually filled with a clot, which adheres closely to its walls. The older it is the more firmly it becomes attached. A recent clot is dark red and soft, but in a few days it becomes paler, granular and friable. It may be limited to one part of the sinus, or may extend to its whole length. The veins from which the sinus receives its blood are always largely distended, on account of the mechanical obstruction to the return of the blood, and intense congestion and edema of the part from which the vessels come are present.

WHAT ARE THE SYMPTOMS?

The symptoms are not well defined, as they are usually covered up by the primary disease. Headache, apathy, coma, vomiting, general convulsions, rigidity of the neck, strabismus, trembling of the tongue or limbs and epistaxis are the common symptoms. Mental dullness for a day or

two, followed by repeated convulsions involving one side, are sometimes present. Edema about the eyelids and temples and protruding eye-balls occur when the cavernous sinus is affected.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

Whenever the above train of symptoms supervenes upon any of the diseases mentioned as the cause of this condition we may be confident that there is thrombosis of the veins or sinuses.

WHAT IS THE PROGNOSIS?

It is extremely grave, but occasionally children survive the condition. It runs its course from a few days to six or eight weeks.

WHAT IS ITS TREATMENT?

The treatment should be directed to the condition which has produced the thrombosis.

ACUTE CEREBRAL PALSY OF CHILDHOOD, OR INFANTILE HEMIPLEGIA.

WHAT IS MEANT BY ACUTE CEREBRAL PALSY OF CHILDHOOD?

A paralysis occurring suddenly in children, due to some organic disease of the brain.

WHAT ARE ITS CAUSES?

Hemorrhage into the brain or its membranes during birth or during childhood; embolism; thrombosis; anemia, or inflammation of the brain; porencephalitis, or cavity in the brain, produced by a localized inflammation; simple lack of brain development and brain atrophy are the most common causes of this condition. Pneumonia, scarlet fever, measles and general anemia from exhausting diseases, such as cholera infantum, etc., are often predisposing causes. Females suffer more frequently than males, and the majority of cases occur within the first three years of life. Those cases which occur during birth are undoubtedly caused by trauma.

WHAT IS THE PATOLOGICAL ANATOMY?

This depends upon the brain disease which has produced the palsy. Atrophy, a cavity within the brain due to a localized inflammation, anemia and softening due to thrombosis and the results of cerebral hemorrhage are among the changes found.

WHAT ARE THE SYMPTOMS?

Convulsions.—These may be general or confined to one-half of the body or to one member which will later be involved in the paralysis, and show the cortical origin of the lesion.

Paralysis.—Paralysis is the chief characteristic symptom. It may appear suddenly, with loss of consciousness, or it may come on immediately after a convulsive seizure. It may be confined to one member, or to the members on one side of the body. It may be complete for several days, and then improvement may commence, the leg improving more rapidly than the arm. In some cases the paralysis may entirely pass away so that no signs of it are left.

Aphasia.—This defect of speech is a symptom which often complicates these palsies. It is often permanent, and is one of the causes of mutism.

Mental Defects.—Defects of the mind are observable to a greater or less degree in all cases. They manifest themselves either in loss of moral sense or may go on to extreme idiocy.

Rigidity of Muscles.—Muscular rigidity is a late symptom of the paralysis and is associated with contractures which lead to the various forms of club-foot, especially equino-varus. Contractures of the flexor muscles of the elbow, wrist and fingers may be present. Wax-like rigidity of the member affected is a characteristic symptom.

Exaggerated Reflexes.—These are observed in both upper and lower extremities.

Athetosis.—Occasionally present in these cases.

Defective Growth.—The paralyzed member is limited in its development, the limb remaining shorter and smaller in circumference than normally.

Cranial Deformities.—Irregularities in development, such as asymmetry, are frequently seen.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

This disease may be sometimes confounded with polio-myelitis-anterior, which is similar to it in many respects, both as to method of onset and in the paralysis which remains. In polio-myelitis-anterior there are no mental symptoms, no cranial asymmetry, no rigidity of the muscles, the member hanging relaxed and flaccid. The atrophy in this disease is also more marked than in cerebral palsy. There is also a loss of reflex action in the paralyzed member, while in cerebral palsy the reflexes are exaggerated.

From birth palsy or infantile meningeal hemorrhage it is differentiated by the history of a distinct onset after birth.

WHAT IS THE PROGNOSIS?

The initial convulsions are sometimes followed by death. If the patient survives the convulsions, and if these have not been severe, recovery may be more or less complete. In the majority of cases paresis, contractures, exaggerated reflexes, and mental defects are observable during the rest of the patient's life. Many of these cases develop epilepsy later in life.

WHAT IS THE TREATMENT?

GENERAL.—The little patient should be kept as quiet as possible during the initial stage of the disease. Cold applications to the head may be used during the convulsions: hot water to the feet, or the feet may be immersed in hot mustard water. As the little one recovers from the convulsion and the paralysis manifests itself the paralyzed member should be kept warm by wrapping it in cotton or in a warm flannel blanket, as it is inclined to become cold. When contractures come on, they may be overcome by the use of the faradic current. Orthopedic surgery and appliances may be necessary to straighten deformed limbs.

EDUCATIONAL.— This is the main treatment for the mental impairment due to cerebral palsy. It cannot be general, but must be special for the individual case. A teacher is required for each pupil, who should understand thoroughly all of the patient's peculiarities. Discipline is most essential, for unless the child be made to mind the

moral sense will become obliterated and a wayward, unmanageable child will be the result of this neglect. The kindergarten method of training is most excellent in these cases and is the system used in the homes for feeble-minded children, where large numbers of them are to be found.

DIETETIC.—In the initial stage milk, boiled rice, oatmeal and flour gruels are advisable. Later, a mixed diet, both meat and vegetables, is necessary. Meat should be allowed but once a day and in small quantities. Rice pudding, corn starch, Indian meal pudding, and bread and butter, should form the basis of diet. These patients should be well fed and at regular intervals.

REMEDIAL.—*Aconite.*—When the convulsions and paralysis come on suddenly, with great nervous excitement, excessive restlessness and tossing about; jerking of leg and arm; grinding of teeth; heat; startings; twitchings of single muscles; child gnaws its fists; body rigid and bent backwards; fist clinched across throat; gnashing of teeth; eyes drawn up spasmodically; great muscular weakness; weariness; prostration and total inability to stand.

Arnica.— Paralysis depending upon extravasation of blood in the brain; left-sided; general sinking of strength; can scarcely move a limb; sensation of being bruised in the paralyzed parts; bed feels too hard; paralyzed limb generally painful. This remedy may be continued for some time with great benefit in helping to restore the power of the paralyzed member.

Cuprum.—Convulsions beginning with cramps in the lower extremities, and drawing in of the fingers and toes; throwing about of limbs; frothing at the mouth and choking at the throat; restless; tossing about and constant uneasiness; starting and grinding of the teeth; stiffness of the whole body; violent spasm similar to epilepsy; red face; head and face puffed up; shrill shriek before the attack; paralysis of tongue; stuttering; deficient speech; paralyzed limbs grow thinner; complicated with unyielding contractures.

Gelsemium.—Infantile paralysis; complete relaxation of the whole muscular system; great drowsiness; loss of rest; great muscular weakness; tingling, pricking, crawling sensation in the limbs; trembling of hands when lifting them up; mental exertion causes a sense of helplessness from brain weakness; paralytic symptoms in throat and limbs.

Calcarea carbonica.—Great loss of power on walking, especially in limbs, with exhausting sweat; great exhaustion on waking in the morning from a deep sleep; child languid, yellow and pale; sensation as if about to faint, with headache; paralysis of voluntary muscles; easy relapses; child does not continue to convalesce after the onset of the disease; frequent severe spasms especially in the evening and at night, with coldness of thighs; muscular twitching, clonic spasm and epileptic paroxysms.

Kali phosphoricum.—Paralysis dependent upon exhaustion of nerve power in recent cases resulting from scarlet fever, measles or diphtheria; general debility, with nervousness and irritability; starting on being touched or at sudden noises; paralysis with atrophy; paralyzing pain in the limbs, made worse by external warmth and motion.

Strychnia.—For contractures, athetosis and clonic spasms of the muscles in the later stages. This remedy gives best results when used in the 200th potency.

ENCEPHALITIS.

WHAT IS ENCEPHALITIS?

It is an inflammation of the substance of the brain, occurring very rarely alone, but usually complicated with meningitis.

WHAT ARE ITS CAUSES?

Idiopathic encephalitis is practically unknown. Injury may set up an inflammation of the brain as well as the meningitis. Occasionally the membranes escape and the brain alone becomes involved in the inflammatory process. Punctured wounds may cause circumscribed inflammation; operations upon the brain, such as trephining or the removal of tumors; new growths within the brain may cause inflammation of adjacent tissue by their pressure; acute diseases, such as erysipelas, diphtheria, and typhoid fever, are common causes. Sunstroke, great mental anxiety and alcoholism also produce it.

WHAT IS THE PATHOLOGICAL ANATOMY?

Red softening is present in all cases. The consistence of the brain tissue is lessened on account of the disintegra-

tion of the tissue elements by effused liquid and the separation of the particles. The affected area is usually swollen and puffed up. The nerve fibres, ganglion cells and neuroglia cells become degenerated. After complete disintegration of tissue elements there is left a fatty emulsion in the cavity formed by the inflammatory process.

WHAT ARE ITS SYMPTOMS?

Headache, occasionally vomiting, general convulsions and acute delirium develop rapidly. There are great cerebral excitement, incoherent speech, attempts to escape from the bed. Temperature may range from 102° to 106°, according to the intensity of the delirium. Somnolence and coma may follow the delirium. Mental weakness is left after the attack.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

From acute meningitis it is differentiated by the absence of hyperesthesia and stiffness of the muscles of the back and of the neck.

From acute mania by the height of the temperature and the suddenness of onset.

WHAT IS THE PROGNOSIS?

This depends upon the severity of the inflammation. If delirium is great and temperature high the result is usually fatal. In milder cases recovery may begin after a few days, but some mental weakness is usually left.

WHAT IS THE TREATMENT?

GENERAL.—Absolute quiet in bed in a darkened room; applications of cloths wet in cold water to the head, and the avoidance of everything that would be apt to produce excitement; exclusion of all unnecessary friends from the room, and no loud talking or whispering in the patient's presence.

Dietetic.—Avoidance of all stimulants such as tea, coffee and alcohol. Milk, eggs beaten in milk, thick broths, rice, oatmeal, sago, or barley boiled a long time, may be

given every two or three hours in small portions. Water, cold, ad libitum.

REMEDIAL.—*Aconite.*—Idiopathic cerebral inflammation, especially from lying with the head exposed to the direct rays of the sun, particularly when asleep: violent burning pains through the brain, especially in the forehead: fever: delirium: red, bloated face; burning as if the brain were moved in boiling water.

Aethusa cynapium.—Delirium; imagines he sees cats and dogs: jumps out of the window; great anxiety and restlessness, bad humor, irritability: wants to escape: sensation as if both sides of the head were in a vice; headache with vertigo.

Anacardium.—For the sequela of brain fever: total loss of memory: weakness of special senses: dullness and confusion of head: incomplete paralysis of muscles subject to volition: tendency to use profane language.

Belladonna.—Rambling delirium: fear of imaginary things: wants to run away from them: sees ghosts, hideous faces, and various insects: tears the clothes from his body and runs naked into the streets in broad daylight. When closing the eyes, when not asleep, the patient sees fierce, wicked-looking, large animals: delirium worse in the evening: disposition to bite, spit, strike and tear things: jumping out of bed with fear: trying to run away and hide.

Glonine.—Sensation as if the head were enormously expanded; pain ascending from below upward, and from without inward: sensation as if warm water were running upward from the nape of the neck: sudden sensation as if the head were crowded with blood: brain feels as if too heavy and too large for the skull, with raving headache: racking pain with raving; heat in head: redness of face: red, protruding eyes: wiry pulse.

Hyoscyamus.—Stupor, loss of consciousness: talking of the affairs of the day: picking of bed-clothes: delirium with physical restlessness: will not stay in bed: great restlessness; piercing, staring look: raving, scolding, singing: chatters day and night.

Stramonium.—Unconscious and stupid: weakened intellect: loss of memory: does not recognize his friends and family: confusion of mind: delirium, bland, murmuring,

foolish, joyful, loquacious, incoherent, chattering, with open eyes; frightful fancies take hold of his mind; his features express fright and terror; eyes red and inflamed, wide open and staring; shrinking look as if from fear; dilated pupils; pain in the head and nausea; desire for light; throbbing headache.

Veratrum viride.—When caused by sunstroke, with prostration; fever; intense cerebral congestion; feeling as if the head would burst open; buzzing and roaring in the ears; double vision; convulsions of all the limbs; trembling of the whole body coming on suddenly; frothing at the mouth and violent jactitations of all the voluntary muscles.

ACUTE SUPPURATIVE ENCEPHALITIS.

WHAT IS ACUTE SUPPURATIVE ENCEPHALITIS, OR ABSCESS OF THE BRAIN?

It is a collection of pus either on the surface of the brain or within its substance, forming an abscess.

WHAT ARE ITS CAUSES?

It is more common in men than in women, and may occur at any time of life, but most frequently during the third decade. Injury; suppurative inflammation, near or distant, from which septic material is carried to the brain; disease of the bones of the skull, and middle ear diseases; chronic disease of the nose; abscess of the liver; abscess of the lungs; imperfectly resolved pneumonia, and odium albicans, or thrush in the mouth. In about half of the cases no cause can be found.

WHAT IS THE PATHOLOGICAL ANATOMY?

The abscess cavity may be as large as a walnut or a hen's egg. If it be near the surface of the brain the membranes become thickened by inflammation and form one wall of the abscess. The cavity may be irregular and the capsule forms after a time, first being thin and delicate, then gradually increasing in thickness and firmness, its inner surface smooth and its substance composed of connective tissue elements. Outside of the capsule the brain tissue is sometimes softened

and there may be some fatty degeneration of the cerebral elements. After the capsule is formed pus cells still increase in the abscess cavity. The pus has a greenish tinge. The majority of abscesses have capsules, but some do not. Multiple abscesses may be present, but they are usually small. In the larger number of cases the abscesses are single. Abscess occurs in the cerebrum four times as fre-

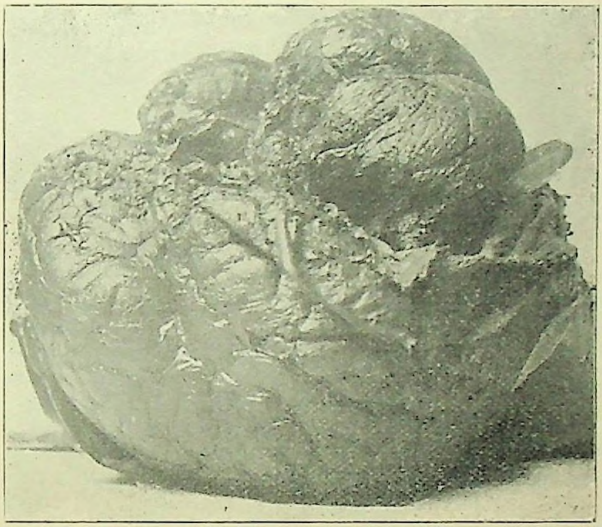


Figure 22.

Abscess of cerebellum. No symptoms until three hours before death.

quently as in the cerebellum, and is rare in the pons or medulla. The cause of cerebellar abscesses is always suppuration of the middle ear. If the abscess is of any size it causes pressure in the neighboring parts of the brain, but not to such an extent as tumor. Abscesses may become calcified and may remain in the brain many years.

WHAT ARE THE SYMPTOMS?

The first symptoms in acute cases are those of cerebral inflammation: but in chronic cases there may be a latent period which does not at first manifest itself. All degrees

are met with from severe cerebral inflammation to a slowly forming abscess.

Headache.—Headache is a frequent symptom; it may be extremely severe or moderate in degree. Patients have been known to die from the severity of the pain. It is increased by muscular exertion and dependent positions of the head. The pain usually corresponds with the seat of the disease, but sometimes is referred to a different part of the head. It may occasionally change its position from day to day, but usually when it once becomes seated it remains. Percussing the skull over the seat of the abscess will sometimes cause intense pain.

Vomiting.—Emesis is generally associated with the headache. It is more frequent in cerebellar abscess.

General Convulsions.—Convulsions may occur either at the beginning or at the end of the disease. If at the end, they are usually due to rupture of the abscess.

Paralysis.—Paralysis occurs in some cases. It may be only slight and is not often complete.

Sensation.—Not often affected.

Mental Symptoms.—Common at the terminal period following rupture; stupor, delirium, followed by coma or mental depression or failure of the mental faculties, may take place.

Temperature.—Raised near the end of the disease. It is often accompanied by rigors, followed by sweating, which is characteristic of fever due to septic poisoning.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

It is extremely difficult to diagnose this disease, for we may have an ordinary attack of encephalitis which produces the abscess followed by a period of latency, which leads us to believe that the patient has fully recovered. Usually there is sudden rupture, with terminal symptoms. Chronic abscess must be differentiated from tumor. When the causes which produce abscess are absent but symptoms of it are present the diagnosis is in favor of tumor.

From meningitis it may be distinguished by its longer duration and from the fact that the cranial nerves are not so frequently affected. The two, however, may be so nearly

alike in their symptomatology that it will be impossible to differentiate them.

From chronic ear disease it is difficult to distinguish abscess, because the meningitis is so frequently a complicating condition.

Cerebellar abscess may not produce any symptoms at all, particularly when situated in the lobes of the cerebellum.

An abscess within the centrum ovale may exist for years without manifesting more than trifling symptoms, not pronounced enough to enable us to make a diagnosis.

WHAT IS THE PROGNOSIS?

When the diagnosis is certain the prognosis is grave unless surgical measures can be brought into use. The terminal stage may be precipitated at any time and death result.

WHAT IS THE TREATMENT?

GENERAL.—Surgery, when of avail, is the most important measure to be used. In middle ear diseases or in mastoid abscess a free opening may relieve the trouble. The health of the patient should be kept up as much as possible by meeting symptoms as they arise.

DIETETIC.—An abundance of good nutritious food, consisting of meat, vegetables, and a general mixed diet, is of great importance before the terminal period.

REMEDIAL.—*Belladonna.*—Stabbing through the head as if with a double-edged knife: stabbing in the right side of the head: head aches as if the sutures of the skull were being torn open, as if a lever were being applied to force the bones of the skull asunder: pains come on suddenly, last indefinitely and cease suddenly: headache makes patient first blind, then unconscious: violent pain in the ear: face flushed: cannot keep any food on the stomach: cannot hold his head up on account of nausea: vomiting of watery, slimy, bilious fluid.

Calcarea carbonica.—Hammering in the head, weight on top of head, violent throbbing on vertex, has to lie perfectly still, is aroused from sleep every morning at five o'clock by a violent aching pain in vertex, one-sided headache with belching, internal and external sensation of cold-

ness of various parts of the head as if ice were on it, with pale, puffed face; severe heat in head and great orgasm of blood, nocturnal sweats of head; copious, exhausting sweats all over; hectic fever, with alternate chills and heat; sweats easily.

Hepar sulphur.—Constant pressive pain in one-half of the brain, as of a plug or nail; lancinating headache, worse when walking in the open air; extreme sensitiveness of the scalp, cannot bear to have anything touch the head; whizzing and throbbing in the ears; darting pain in the ears; discharge of fetid pus from ears; over-sensitiveness to pain; fainting from a slight pain; hectic fever, with intermittent paroxysms; internal chill, with weariness and soreness in all the limbs; dry, burning heat, with violent thirst.

Mercurius.—Congestion to the head; head feels as if it would burst, with fullness of brain, as if constricted in a band; head feels heavy and swollen, as if getting larger and larger; hoop-like feeling; head feels as if in a vise, with nausea; sticking and burning deep in the ear; bloody and offensive matter flows from the ear, with tearing pain; stitching, tearing, pressing, burning pains deep in the ear, extending to cheek; involuntary motions of head and hands.

Silica.—Violent headache, with loss of consciousness or reason; headache wakes him up at night; loud cries, nausea to fainting from the severity of pain; vibratory shaking sensation in head when stepping hard; purulent discharges from the ear; caries of mastoid process; nausea and vomiting of what is drunk; chilliness; constant internal hectic fever, particularly during long suppurating processes; sweat on head from the least exertion.

INTRA-CRANIAL TUMORS.

WHAT IS AN INTRA-CRANIAL TUMOR?

A tumor within the cranial cavity, either upon the inner surface of the cranial bones, or within the membranes of the brain, or in the brain substance itself.

WHAT ARE THE CAUSES OF INTRA-CRANIAL TUMOR?

The causes of these tumors are sometimes as obscure as the causes of tumors in other parts of the body. Males

seem to suffer more frequently than females, except in sarcomatous growths. Tumors may occur at any time of life, but they are not common in old age. The greater number of cases occur during childhood and active adult life. A majority of those occurring during childhood are of a tubercular nature. They may, however, occur up to seventy years of age. Gliomatous growths also occur during active adult life, most frequently between twenty and forty years of age. Sarcoma occur at about the same time as glioma. Parasitic tumors generally occur between ten and twenty. Carcinoma is most frequently met with between 40 and 60. Tubercular and syphilitic growths depend upon a constitutional dyscrasia. In the former there is usually an hereditary history of consumption; and in the latter, a personal history of syphilis. Trauma is sometimes an exciting cause of a growth, but there must be some predisposing condition back of it to induce the growth.

WHAT IS THE PATHOLOGICAL ANATOMY?

Intra-cranial tumors may be of several varieties: tubercular, syphilitic, glioma, sarcoma, myxoma, carcinoma, fibroma, osteoma, cholosteatoma, lipoma, vascular, psammoma, neuroma, parasitic, and simple cysts. They occur in frequency about in the order named.

Tubercular.—Occur in solid, firm, rounded masses, regular in shape, surrounded by a softened layer of brain matter. They vary in size from that of a pea to that of a hen's egg, and upon section present an opaque cheesy aspect, softened here and there. They occur in the cerebral substance, and usually without any connection with the membranes; and also within the cerebellum. There is usually more than one tumor; sometimes two or three.

Syphilitic.—Irregular and nodular in shape, and vary in size from that of a pea to that of a walnut. They present upon section a cheesy appearance, irregularly distributed through the mass. Sometimes they appear shrunken, hard, fibroid, and surrounded by a hardened capsule. In favorable cases sometimes only a cicatrix is left. They are situated most commonly within the cerebellum or the pons, and are usually superficial and attached to the pia mater.

Glioma.—Have a glue-like consistency and their elements resemble these of the neuroglia. Round, oval, fusiform cells are visible. Their tint is gray or reddish-gray and appears very much like that of the brain tissue. Soft-

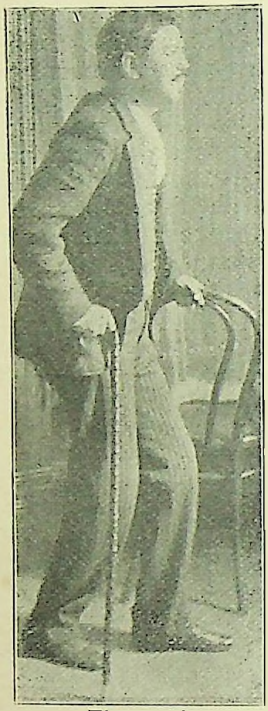


Figure 23.
Attitude in syphilitic gumma at base of brain.

Figure 24.
Same patient four years ago.

ening sometimes occurs which converts the growth into a cyst. Hemorrhage may also take place into these growths. They are irregular in shape and throw out processes into the surrounding brain tissue. They are usually single.

Sarcoma.—May arise from the inner surface of the cranial bones, from the membranes, or from the brain. When within the brain substance they have a well-defined limit and the brain tissue is usually somewhat softened around

them, so that they may be easily removed. They may be either hard or soft.

Myxoma.—A mucoid growth much like a glioma.

Carcinoma.—Usually soft and may arise from the dura mater or may be contained within the cerebral hemispheres. They infiltrate to some degree the brain substance, but usually displace it. They are usually single.

Fibroma.—Rare, and occur most frequently in the cerebellum.

Osteoma.—Met with in the cerebral hemispheres.

Cholesteatoma.—A form of cystic tumor arising from the pia mater within the recesses of the brain.

Lipoma.—Composed of soft, firm lobular masses of adipose tissue bound together by fibrous septa, and occur on the surface of the corpus callosum.

Vascular.—Rarely met with, but when present are usually in the cerebral hemispheres.

Psammoma, or *Sand Tumors.*—Found in the cerebral meninges and contain granular calcareous concretions.

Neuroma.—Small growths containing nerve elements.

Parasitic.—Either hydatid or cysticerceous. The hydatids may be outside of the membranes, but they are generally within the hemispheres. The cysticerci are within the membranes or in the cortex.

Simple Cysts.—The result of hemorrhage or softening, their contents, effused blood or broken down nerve elements, having become absorbed.

These growths produce certain pathological effects upon the brain. They destroy the nerve elements which are near them by their pressure and by the effect of the growth of the morbid tissue elements. They also produce distant pressure and affect all the parts of the brain, those nearest the growth being the most affected. Internal hydrocephalus is sometimes produced on account of the pressure of the growth in the aqueduct of Sylvius. Inflammation in adjacent brain tissue is caused by the irritation of the growth. Meningitis may also be set up. Thinning of the cranial bones and actual perforation have been observed by the constant erosion of the growth.

WHAT ARE THE SYMPTOMS OF INTRA-CRANIAL TUMORS?

They are of two kinds, general and local.

General Symptoms.—*Headache.*—Generally constant, with acute exacerbations. It may be dull or rending, stabbing, tearing, boring pain preventing sleep, and sometimes producing insanity; increased by any muscular exertion. It may be felt in all parts of the brain, and in the front or back of the head or on one side of the head. If the tumor be upon the surface of the brain, the pain generally corresponds with its situation. If the growth be in the centrum ovale, the pain is most often frontal; when below the tentorium, the pain is occipital. Gentle tapping over the seat of the disease, if the tumor is superficial, will sometimes cause intense agony, in which case the pain is usually due to irritation of the meninges.

Optic Neuritis.—Occurs in the majority of cases of intracranial tumor, wherever the growth may be; and it does not matter whether it be small or large. There is probably an extension of tissue irritation to the optic tract and nerves, which produces an inflammation of the papilla. The neuritis may develop rapidly or slowly, and it may go on to atrophy.

Mental Symptoms.—Coma and symptoms of cerebral apoplexy are common in the last stages of tumor. Mental failure, loss of memory, great mental depression and irritability are frequent. Sometimes delusions may occur when the tumor is in the frontal lobe.

Vomiting.—A common symptom, particularly when the growth is in the medulla or in the cerebellum.

Dizziness.—Attends the majority of cases, and is sometimes extremely troublesome.

Affections of Speech.—Slowness of speech, a tendency to separate words, difficulty of articulation, and aphasia are present with tumors in the white substance.

Local Symptoms.—*Paralysis.*—May be either a hemiplegia, monoplegia, or may affect the cranial nerves.

Contracture.—Due to excess of myotatic irritability, frequently comes on with the paralysis and usually accompanies the loss of power. Unsteadiness in standing, but most marked in walking, is common in tumors of the cerebellum. The patient may sway backwards or forwards, or stagger like a drunken man.

Convulsions.—May be general, with loss of consciousness, similar to ordinary epilepsy, or they may involve first only one member, and then gradually spread to others, or even be limited to the one in which it first commenced.

SENSORY SYMPTOMS.—Hemianesthesia may be present with a tumor situated in the posterior part of the internal capsule. Numbness, tingling and pain are sometimes produced by cerebral tumor.

Figure 25.
Attitude in cerebral tumor beneath the cortex.

Loss of Smell.—May occur from tumors in any part of the brain.

Strabismus.—Either paralytic or spasmodic occurs when the third, fourth or sixth nerves is affected by the growth.

Neuralgia.—Sometimes most intense, with extreme hyperesthesia, is present when the fifth nerve is irritated, and may be followed by anesthesia.

The symptoms are always of gradual onset, and progress with more or less regularity. There may be latent periods during which the disease may in a measure pass away. Sensory symptoms may give way to those of paralysis, and the nerve fibres become degenerated after a long period of irritation.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

In every case of brain tumor we have to determine, first, that there is some organic disease of the brain; second, whether it be a tumor or not, if so its seat and its nature. The gradual onset of the symptoms, the headache, optic neuritis, vomiting, convulsions and the local symptoms are usually enough to diagnose the disease from anything else.

Abscess of the brain has many symptoms similar to tumor, but the local symptoms are not often present. The history of suppura-

tive disease, such as mastoid disease or middle ear abscess, or the results of injury, are usually present in abscess.

To determine the nature of the growth is usually a comparatively easy matter. The presence of tubercle in other parts of the body would lead us to believe that the growth is tubercular, or if there is an hereditary tubercular history. If acquired syphilis does exist or has existed the tumor will most probably be syphilitic. The results of treatment may also determine the syphilitic character of the growth. Cancer or fatty tumors in other parts of the body would probably be of the same nature as that in the brain when co-existing with it. A tumor which is very slow in developing in the cerebral hemispheres will probably be a glioma.

WHAT IS THE PROGNOSIS?

Syphilitic tumors may be stopped. All other growths usually go on to a fatal termination, unless they are of such a character and in such a position that surgical measures may be applied for their removal.

WHAT IS THE TREATMENT?

SURGICAL.—Tumors with well-defined limits, of firm consistency, and not infiltrating, such as tubercular, lipoma and sarcoma, situated near the surface of the brain, may sometimes be removed with good results.

REMEDIAL.—*Belladonna.*—Headache increased at night, makes patient crazy, he has to run up and down, and often falls; incessant, dull, pressive pain on one or the other side of the head; violent pressing in the whole head from within outward; congestion to the head with delirium; red face; heaviness and paralytic feeling in upper limbs; numbness and pricking in one hand; weakness of the whole arm; convulsive movements of limbs; loss of co-ordination in muscles of both upper and lower limbs; violent convulsions followed by sound sleep; attacks similar to epilepsy; sleepy yet cannot sleep; great restlessness at night.

Baryta carbonica.—Great mental and bodily weakness; whining mood; stupid; pressure in brain under vertex, towards occiput on waking, with stiffness of neck; buzzing and ringing in the ears. Useful in fatty and sarcomatous

tumors; excessive irritation of all the nerves; general weakness of nerves and body; too weak to even chew his food.

Calcarea carbonica.—Stupefying, pressive pain in the forehead, with confusion of senses and dullness of whole head while reading; tearing headache above eyes, down to nose, with nausea; violent throbbing on vertex; has to lie perfectly quiet; headache begins in occiput and spreads to top of head, so severe that she thinks head will burst and that she will go crazy; tearing in bones of head; head too large, fontanelles not having closed in childhood; weakness and paralysis of left arm; nightly lacerating and drawing pain in the arm; frequent paralysis of fingers; heaviness and painful weight in limbs and great fatigue on walking; great loss of power on walking, especially in limbs, with exhausting sweat; loss of flesh and general muscular weakness. Especially useful in tubercular subjects.

Conium.—Complete indifference; takes no interest in anything; sad and gloomy for days, then excited; cannot endure any kind of excitement as it brings on physical and mental depression; lancinating pains especially in the vertex; pain in occiput with every pulsation as if pierced with a knife; sensation in right half of brain as of a large foreign body; paralyzed feeling in all the limbs; numbness of both fingers and toes; staggering gait; muscular paralysis without spasms; drowsiness by day; stupid in the morning. Useful in fibroma and carcinoma.

Hydrastis canadensis.—Moaning, with occasional outcries from pain; sharp cutting in temples and over the eyes; myalgic headache in integument of scalp and muscles of neck; great despondency; intense lancinating pains in the head; general debility; cancerous cachexia.

Kali iodatum.—Terrible hammering pains in forepart of head; lancinating and darting over left eye; pains in side of head as if screwed in; vision dim and foggy; sees objects indistinctly; violent jerking of limbs and muscles of thigh; boring, tearing pains in temporal bone; numbness of mouth; pains all worse at night; great general debility. Useful in syphilitic growths.

Mercurius.—Tearing, drawing pains in the head, having their seat in the periosteum and bones; scalp tense and con-

tractive; tearing pain from forehead to neck; lancinating, tearing and stinging in bones of scalp; whole external head is painful to touch; trembling of extremities; involuntary jerking in limbs; weakness and weariness in limbs; general tremors with stammering of speech. Useful in syphilitic subjects.

INTRA-CRANIAL ANEURISM.

WHAT IS AN INTRA-CRANIAL ANEURISM?

It is a tumor formed by a localized dilatation of an artery within the cranial cavity.

WHAT ARE ITS CAUSES?

It occurs more frequently in males than in females, and between ten and sixty years of age. There are sometimes hereditary tendencies toward the formation of aneurism. Injury, such as a blow or fall on the head, causing an arteritis which may so change the wall of the artery that it becomes easily dilated. Syphilis and embolism are frequent causes.

WHAT IS THE PATHOLOGICAL ANATOMY?

The walls of the artery are usually thin and bulging at different points, producing an aneurism. The aneurism may vary in size from that of a pea to that of a hen's egg. They are usually round, sometimes oval. As it grows in size it compresses the brain tissue and thus damages it to a greater or less degree. It is this pressure which produces the symptoms. In case of rupture the blood escapes into the cerebral tissue and into the membranes. The middle cerebral artery is most frequently involved, and next the basilar. The internal carotid, anterior cerebral, posterior cerebral, vertebral, and the communicating arteries may also be involved.

WHAT ARE THE SYMPTOMS?

Aneurism of considerable size may exist within the brain for many years without giving any indication of its presence until rupture takes place. Where symptoms are present they are usually those which are characteristic of tumor, such as headache, vomiting, giddiness, optic neuritis,

affections of speech, mental disturbances, symptoms of pressure upon the cranial nerves and other local symptoms.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

It is impossible to diagnose intra-cranial aneurisms until pressure symptoms are present, and even then we may not be able to differentiate between aneurism and tumor until we are able to distinguish the aneurismal murmur which in rare cases has been heard.

WHAT IS THE PROGNOSIS?

When the diagnosis is certain the prognosis is always grave, and it is impossible to tell at what moment death may occur from rupture.

WHAT IS THE TREATMENT?

GENERAL.—Avoid as much as possible any excessive muscular exertion, such as lifting heavy weights, or running, stooping over, or lying with the head low. All mental worry should be avoided as much as possible, and every means used to prevent either active or passive congestion of the brain.

DIETETIC.—Good, plain, wholesome food without much sweets or pastries, which are likely to produce hyperemia of the liver and, secondarily, hyperemia of the brain.

REMEDIAL.—The remedies used in intra-cranial tumors will be found useful in the treatment of aneurisms.

DEGENERATIONS OF THE BRAIN.

CYSTIC DEGENERATION OF THE BRAIN.

WHAT IS CYSTIC DEGENERATION OF THE BRAIN?

It is the formation of cavities within the white substance of the brain. These cavities are probably enlarged peri-vascular spaces, and are usually filled with broken down nerve tissues and the remains of small extravasations

due to the rupture of vessels within them. These cavities usually occur in the shrunken brains of the old, and there may be numbers of them. It does not matter how great the number of these cavities, for they usually give no symptoms.

CHRONIC PROGRESSIVE SOFTENING OF THE BRAIN.

WHAT IS CHRONIC PROGRESSIVE SOFTENING OF THE BRAIN?

It is a slow progressive softening involving the white matter of the brain, and is a primary process. The change is a simple white softening, and the microscope will show the products of degeneration. The areas affected are usually sharply limited from the normal brain tissue. While the softening usually commences in the white matter it may in some cases involve the gray matter of the cortex.

WHAT ARE THE CAUSES?

It occurs in both sexes, and is most frequent between sixty and eighty years of age. No special causes have as yet been determined as the disease is rare.

WHAT ARE THE SYMPTOMS?

A gradually increasing hemiplegia and hemianesthesia. The weakness may commence in one member and gradually involve the whole side without any special sensory loss at first. Numbness, tingling, and formication in the limbs are sometimes present. Rigidity of the members paralyzed may come on at first, but it passes away when the paralysis is fully developed. There may be attacks of vertigo, which soon pass away. The intellect may not be much affected, though in some cases it may become blunted toward the last.

WHAT IS THE PROGNOSIS?

It is invariably fatal, running its course in from two or three months to two years.

Little can be said with regard to treatment. The main method of procedure is to nourish the patient as much as possible, and to use such remedies as may be indicated from time to time for the general symptoms.

DISSEMINATED OR INSULAR SCLEROSIS, OR MULTIPLE SCLEROSIS.

WHAT IS DISSEMINATED SCLEROSIS?

It is the formation of scattered islets of hardened tissue due to proliferation and increase of connective and neuroglia tissue in the brain and spinal cord.

WHAT ARE ITS CAUSES?

It occurs in both sexes with about the same frequency and at any period of life, but especially in the first half of adult life. The larger number of cases commence between twenty and thirty-five. Heredity is an occasional cause, such as a family history of insanity, epilepsy, etc. Many cases develop without any cause that can be determined. Exposure to cold, mental worry, over-exertion, both mental and physical, acute diseases and trauma are often exciting causes. It has been known to follow typhoid fever, small-pox, diphtheria and erysipelas. One of the author's cases occurred during pregnancy.

WHAT IS THE PATHOLOGICAL ANATOMY?

The hardened islets are irregular as regards their location, being scattered anywhere through the central nervous system, but almost exclusively in the white matter. They are irregular in shape, and in size are as large as a pea or may be as large as a walnut. They are reddish-gray in color. The central ganglia are frequently involved in the sclerosis. The cerebellum has but few hardened patches. In the spinal cord the diseased spots appear on the surface and extend for some distance into the substance of the cord. They consist of fibrous tissue and are of firmer consistency than the surrounding tissues.

WHAT ARE THE SYMPTOMS?

As these islets of sclerosis are widely scattered the symptoms are correspondingly various. Paresis which goes on to paralysis in different members of the body, associated with jerky irregularity of movement, are prominent symptoms at the beginning of the disease.

Paresis.—Begins usually as a simple weakness in one limb, or maybe both limbs on one side, but the legs are most frequently affected. After a time this weakness becomes an absolute loss of power.

Intention tremor.—An irregular, jerky tremor upon attempting to use the affected member. If the patient tries to lift a glass of water to his lips he is likely to spill its contents on account of this tremor. It ceases entirely when the patient is at rest.

Nystagmus.—One of the manifestations of tremor.

Exaggerated Reflexes.—Present, with a spastic condition of the lower extremities.

Articulation.—Jerky in character, syllables being dropped or indistinctly pronounced, due to inco-ordination of the muecles of the tongue. It is sometimes called "scanning speech."

Oscillation of the Head.—May be so pronounced that the patient is not able to hold his head up or even to sit up. Sometimes the muscles of the trunk are involved in like movements.

Impairment of Sight.—Occurs in one or both eyes, without any demonstrable changes in the optic nerve, at first due to an islet of sclerosis in the optic nerve or in the optic chiasm. Later, however, atrophy of the nerve occurs.

Paralysis of the Eye Muscles.—Due to a paralysis of the sixth nerve, occurs in a large number of cases, and sometimes there may be paralysis of the third nerve.

Handwriting.— Jerky and tremulous like the speech, due to inco-ordination.

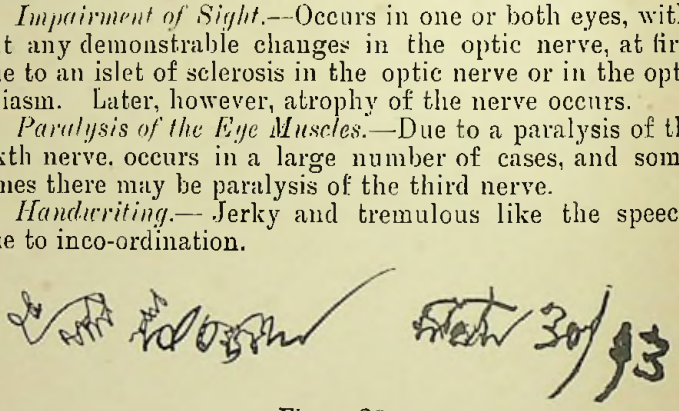


Figure 26.
Handwriting in disseminated sclerosis.

Anesthesia.— Irregularly distributed anesthetic patches may occur whenever a sensory nerve is affected.

Mental Changes.—Characterized by failure of memory and an undue complacency and contentment which are entirely out of place with the existing condition of affairs.

Headache, dizziness and vertigo are usually present to a greater or less degree.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The characteristic symptoms of jerky inco-ordination, combined with progressive weakness in the limbs and nystagmus, make it easy of diagnosis.

In paralysis agitans the tremor is regular in character, and persists even when the patient is quiet.

Disseminated sclerosis is sometimes confounded with hysteria, but the jerky inco-ordination, the exaggerated reflexes and the nystagmus should enable one to determine the difference between the two.

WHAT IS THE PROGNOSIS?

Always grave. In some cases it progresses steadily from first to last toward a fatal termination, though usually periods of apparent rest alternate with those of progress. The duration is from two to fifteen years, but the average is from three to six years. The cause of death is the involvement of the medulla by the sclerosis, which interferes with its function, preventing swallowing and even respiration itself.

WHAT IS THE TREATMENT?

GENERAL.—Patients suffering from this disease should exert themselves as little as possible, rest a great deal, and take life easy. Hygienic measures should be rigidly enforced. Electricity should not be used when spastic symptoms are present.

DIETETIC.—Good, nourishing, wholesome food, without pastries or sweets, at regular intervals and in sufficient quantity, is the proper diet for such cases.

REMEDIAL.—*Argentum nitricum.*—Great weakness of mind; tremulous weakness accompanied by general debility; periodical trembling of the whole body; lassitude and weariness of forearms and legs; legs feel as if made of wood; inco-ordination of muscles with staggering gait.

Baryta carbonica.—Great mental and bodily weakness with glosso-pharyngeal paralysis; paralysis of upper and

lower extremities; great drowsiness during the day; weakness of sight; irregular contraction of pupils.

Causticum.—Paralysis of single parts or single nerves coming on gradually; obscuration of sight as if a veil were drawn before the eyes; impairment of deglutition and speech; contraction of muscles; exaggerated reflexes; unsteady walking.

Crotalus horridus.—Tremulous weakness all over; easily tired by slightest exertion; sudden and great prostration of vital force; starting, jerking, trembling and cramps of lower extremities, with coldness of feet; contraction of flexors; numbness of hands and feet.

Gelsemium.—Paralytic symptoms in throat and in limbs; complete relaxation and prostration of the whole muscular system, with entire motor paralysis; unsteady gait; staggers as if drunk, due to inability to control muscular movements; eye-balls oscillate laterally when using them (nystagmus); paresis of ocular muscles; great heaviness of lids.

Ignatia.—Mentally and physically exhausted by long continued grief; trembling of hands when writing; great weakness of whole body; paralysis after great mental emotion; trembling of limbs with great langour; staggering gait; jerking and twitching in various parts of muscles.

Silica.—Trembling of legs with extreme nervousness; walks like a hen (grassus gallinaceus); weakness of knees; cramps in calves of the legs; sense of great debility; wants to lie down; limbs go to sleep easily.

Tarentula.—Weakness of legs, not allowing feet when walking to be placed squarely on the ground; difficulty to move legs, they do not obey the will; disorderly and irregular muscular movements of limbs; trembling of the body.

PROGRESSIVE BULBAR PARALYSIS.

WHAT IS PROGRESSIVE BULBAR OR LABIO-GLOSSO-LARYNGEAL PARALYSIS?

A progressive degeneration of the motor nerves which supply the muscles of the mouth, fauces, pharynx and larynx, producing an associated palsy of these muscles. The nerves involved are the fibres of the facial which supply the lower part of the face, the hypoglossal which

supplies the tongue, the spinal accessory which supplies the larynx and palate, and the glosso-pharyngeal which supplies the pharynx.

WHAT ARE THE CAUSES?

Men and boys are more liable to it than women and girls. It occurs more frequently in the second half of life. Mental worry and depression, combined with great physical exhaustion, exposure to cold, lack of proper food, injury, such as a blow upon the back of the neck, and excessive use of the muscles involved are some of the immediate causes.

WHAT IS THE PATHOLOGICAL ANATOMY?

The muscular fibres present fatty and granular degeneration and are much narrower than normal, some fibres more so than others. The nuclei of the sheaths and of the interstitial tissue are increased, and a reddish pigment, the result of degeneration of fibres, is scattered throughout the muscle. The main lesion is found in the nuclei of origin of the hypoglossal, glosso-pharyngeal, pneumogastric and spinal accessory nerves within the medulla or bulb, hence the name of the disease. There is sometimes degeneration of the anterior pyramids. The nerve cells are wasted and their processes shrunken.

WHAT ARE THE SYMPTOMS?

The first noticeable symptom is indistinctness of articulation. The patient is unable to articulate the consonants l, n, t. The tongue cannot be protruded from the mouth but a little way. The lips are also weak, and the patient cannot sound the consonants b, p, m. He cannot whistle. The under lip hangs down and the saliva dribbles from the mouth. There is difficulty in swallowing solids. After a time the lips are paralyzed to such a degree that the patient cannot shut his mouth. The expression of the patient's face changes: the upper part has an expression of anxiety and suffering, and the lower part is motionless and without expression. There is great dryness and stiffness about the throat, and after a time the ability to swallow is lost and articulation also. Mastication is difficult, because the

tongue is not able to guide the food in the mouth. There are no sensory symptoms and the mind is unimpaired; there is, however, a great tendency to weep, which might naturally be the case when the patient realizes the hopelessness of his condition.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The muscles affected, the slowness of onset, and the progressive character of the disease distinguish it clearly from any other condition.

Tumors within the membranes are differentiated from bulbar paralysis, by the symptoms beginning upon one side, while in bulbar paralysis they are bilateral.

From diseases of the brain, such as polio-encephalitis, softening, and cerebral lesions, it is diagnosed by the absence of hemiplegia and of any sensory symptoms.

WHAT IS THE PROGNOSIS?

Grave. It usually goes on to complete degeneration of the nuclei of the bulb, producing such a degree of paralysis that the patient is not able to take the proper nourishment and dies from inanition, or on account of the paralysis of the muscles of the throat particles of food pass into the bronchi and a fatal bronchitis is set up.

WHAT IS THE TREATMENT?

GENERAL.—Rest, massage and faradization of the muscles of the throat and tongue, and abundant nourishment are of great importance in these cases. The so-called "rest cure" sometimes retards the progress of the disease and is therefore of value.

DIETETIC.—As the disease progresses the patient is not able to take solid food, but broths, long-boiled rice or oatmeal, milk, soft boiled eggs, gruels and meat, bread, potatoes, vegetables, macerated in a mortar by adding broth, are of value.

REMEDIAL.—*Anacardium.*—Unable to speak; makes only unintelligible sounds; apt to choke when eating and drinking; swallows food and drink hastily; heaviness of tongue

and sensation as if it were swollen: water flows out of the mouth; low-spirited; disheartened; despairs of getting well.

Baryta carbonica.—Paralysis of tongue; inability to speak; tongue cracked and sore; dryness of mouth; hawks mucus; water runs from the mouth all day; much troubled with tongue phlegm; salivation; inability to swallow.

Belladonna.—Paralytic weakness of organs of speech; inarticulate speech from hindered mobility of the tongue; trembling of the tongue when protruded.

Causticum.—Speechlessness from paralysis of the organs of speech; stuttering; difficult, indistinct speech; paralysis of tongue; saliva runs from the mouth more after eating; paresis of laryngeal muscles and vocal chords; laryngeal muscles refuse to act; sudden loss of voice.

Cocculus.—Tongue seems paralyzed; speech difficult; paralysis of face, tongue and pharynx; great difficulty in swallowing; has to speak slowly in order to be understood.

Phosphorus.—Speech difficult and weak; answers questions with difficulty; stutters when endeavoring to articulate; painless twitching of the muscles of the tongue: much watery saliva in the mouth.

Mercurius.—Speech difficult on account of trembling of mouth and tongue; quick, stuttering, tremulous speech; saliva fetid, or tastes metallic, coppery, tenacious, soapy and stringy; fetid odor from the mouth.

ATROPHY OF THE BRAIN, OR MICROCEPHALY.

WHAT IS ATROPHY OF THE BRAIN?

A wasting of the brain substance which may involve the whole brain or only a part of it.

Atrophy of the whole brain is congenital, while partial atrophy may be acquired.

The skull is usually small as well as the brain, some having thought that the small skull was the cause of the small brain, but this is probably not true, as the skull grows as rapidly as the brain.

There is also more or less atrophy in the senile brain, which may not be altogether a diseased condition.

Mental defects are constant in this condition and the subjects are usually idiotic. Weakness, athetoid movements, and inco-ordination also co-exist.

Figure 27.
Microcephalic Idiot.

This condition of the brain is not a distinct disease and has no place in neurology as such. Its causes are difficult to define and the treatment for the condition is unsatisfactory, as nothing can be done.

HYPERTROPHY OF THE BRAIN.

WHAT IS HYPERTROPHY OF THE BRAIN?

A condition in which the brain is of abnormal size. Hydrocephalus and rickets are usually causes of this condition. The brain sometimes increases abnormally in older children and in adults, but nothing is known of the causes.

The treatment may be directed toward the relief of the hydrocephaloid condition or of the rickets if these causes have produced the trouble.

PART IV.

DISEASES OF THE CRANIAL NERVES.

OLFACTORY.

WHAT CONDITIONS RESULT FROM DISEASE OF THE OLFACTORY NERVE?

Anosmia.—When not due to a disease of the nasal mucous membrane anosmia may be produced by lesion in the olfactory nerve in the medulla, or in the olfactory centre in the cortex of the brain, or it may be produced by a meningitis, tumor of the brain, or hydrocephalus. It sometimes occurs in locomotor ataxia as the result of atrophy of the olfactory nerve. Excessive stimulation of the olfactory nerve may cause its paralysis. Tumors of the brain may produce an olfactory neuritis. Anosmia may be bilateral or unilateral. In the former case, it is called hemianosmia.

Hyperosmia.—A symptom which often manifests itself in hysteria, and in hypnotized subjects.

Parosmia.—Also a symptom of hysteria, or it may be an hallucination indicating the beginning of insanity. Everything smells alike, and everything has a disagreeable odor.

WHAT IS THE TREATMENT?

REMEDIAL.—For *anosmia:* Anacardium, Calcarea. Hyoscyamus, Natrum muriaticum, Plumbum, Silica and Zincum.

For *hyperosmia:* Agaricus, Aurum, Belladonna, Coffea. Conium, Mezereum, Nux vomica, Sepia and Tabaccum.

For *parosmia:* Nux vomica, *odor of cheese;* Anacardium, Calcarea and Veratum viride, *odor of manure;* Aurum, Belladonna, Creosote, Kobalt and Nitric acid, *putrid odor;* Pulsatilla, *odor of tobacco;* Agnus castus, *odor of musk.*

OPTIC NERVE.

INFLAMMATION OF THE OPTIC NERVE, OPTIC NEURITIS OR PATILLITIS.

WHAT ARE THE CAUSES OF OPTIC NEURITIS?

Tumors of the brain are the most frequent cause. Meningitis, abscess, thrombosis, myelitis, nephritis, diabetes, multiple neuritis, lead poisoning and hemorrhage are other causes.

WHAT ARE THE SYMPTOMS?

No subjective symptoms may be reported by the patient, as the sight may remain good for a long time. The disease will have to be determined by the ophthalmoscope. The ophthalmoscopic symptoms are, increased redness of the disc, with its borders obscured, swelling, and later, the form of the disc is entirely lost. The arteries are partly concealed by the swelling of the disc. The inflammation may go on and produce atrophy of the optic nerve.

WHAT IS THE PROGNOSIS?

This depends upon the disease of the brain which produces it. In some cases the inflammation passes away and vision is left intact. In unfavorable cases there is finally absolute blindness.

WHAT IS THE TREATMENT?

The only satisfactory treatment is that applicable to the disease which produces it.

ATROPHY OF THE OPTIC NERVE.

WHAT CAUSES ATROPHY OF THE OPTIC NERVE?

It may be primary or secondary.

When primary, it is a part of either locomotor ataxia, multiple sclerosis, hemorrhage or alcoholism.

When secondary, it is a result of optic neuritis.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is a parenchymatous degeneration of the nerve, with loss of its fibres and an overgrowth of the connective tissue of the nerve.

WHAT ARE THE SYMPTOMS?

Gradual decrease of vision, color blindness and dilatation of the pupil. The ophthalmoscope shows the disc to be of a pearly-white color and cup-shaped. The vessels are decreased in size and are fewer in number.

WHAT IS THE TREATMENT?

The treatment is that of the disease which produces it, according to the symptoms presented.

AMBLYOPIA AND AMAUROSIS.

WHAT ARE THEIR CAUSES?

Great shocks to the nervous system, mental anxiety, worry, hysteria, concussion of the brain and exhausting hemorrhages. There are some drugs which produce these conditions, such as quinine, salicylic acid, alcohol and tobacco. Toxic blood-states, such as the consequences of uremia, may also cause them.

WHAT ARE THE SYMPTOMS?

Dimness of sight or total loss of vision, usually coming on suddenly and lasting but a short time. It generally involves both eyes, but in hysteria it may be greater in one than in the other.

WHAT IS THE PROGNOSIS?

Usually good.

WHAT IS THE TREATMENT?

The conditions which produce the blindness should be treated. Such remedies as Aurum, Belladonna, Causticum, Chelidonium, Phosphorus, Plumbum, Silica and Zincum may be indicated.

HEMIANOPSIA.

WHAT ARE ITS CAUSES?

They may be functional or organic.

Its functional causes are sick-headache, gout and lithemia.

Its organic causes are tumors of the brain, hemorrhages, softening, and inflammations which involve part of the optic nerve, the optic tract, or the chiasm.

The treatment is that suitable for the disease which produces it.

MOTOR NERVES OF THE EYE.

OPHTHALMOPLEGIA.

WHAT IS OPHTHALMOPLEGIA?

Progressive paralysis of all the muscles of the eye.

WHAT ARE THE CAUSES OF OPHTHALMOPLEGIA?

Meningitis of the base of the brain, tumors of the brain, injuries, exposure to cold, the poison of diphtheria, hemorrhage into the sheath of the nerve, syphilitic poisoning, locomotor ataxia, excessive use of tobacco, excessive exposure to light, and the use of morphine and alcohol. Rheumatism may also be the cause.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE THIRD NERVE?

When all the branches of the nerve are paralyzed there are ptosis, double vision, and paralytic strabismus; also dilatation of the pupil, which does not contract to light.

When the branch which supplies the internal rectus muscle is paralyzed there is divergent strabismus.

When the branch which supplies the superior rectus is paralyzed there is a deviation of the eye downward, with a slight divergence.

When there is paralysis of the branch supplying the inferior rectus the eye deviates upward and slightly outward.

When there is paralysis of the branch supplying the inferior oblique there is an inability to move the eye upward and inward.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE FOURTH NERVE?

There is an inability to move the eye downward and inward, with convergent strabismus when the patient attempts to look downward.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE SIXTH NERVE?

There is loss of power in the external rectus muscle, which produces a convergent strabismus. Diplopia is a common symptom in all forms of strabismus.

WHAT IS THE TREATMENT OF OPHTHALMOPLEGIA?

LOCAL.—Electricity applied over the affected muscle is often a most useful adjuvant. The faradic current is the one most used in these cases, and only the weakest possible current should be employed. It may be applied over the conjunctiva, which must first be treated by cocaine, or to the eyelid in the region of the affected muscle. One pole should be placed over the nape of the neck and the other near the muscle to be faradized. It makes no difference which pole is used over the muscle. Surgical measures sometimes have to be employed, as severing some of the fibres of the opposite muscle, which may allow the eye to go back into place.

REMEDIAL.—*Alumina.*—Strabismus due to weakness of the internal rectus muscle.

Gelsemium.—Paralysis of the third and sixth nerves; double vision controlled by strength of will; diplopia due to pregnancy.

Other remedies must be selected according to the totality of the symptoms produced by the cause of the trouble.

INSUFFICIENCIES OF OCULAR MUSCLES.

WHAT IS MEANT BY INSUFFICIENCES OF OCULAR MUSCLES?

An inability of the muscles of the eye to maintain their equilibrium, whereby the visual axis is kept parallel without an effort.

WHAT ARE ITS CAUSES?

It is usually congenital, or may be present in neurasthenic persons.

WHAT ARE THE SYMPTOMS?

A deviation of the visual axis outward, inward or upward. The result of the strain of the ocular muscles may produce headache, nausea, vomiting, epilepsy, chorea, and even insanity.

WHAT IS THE TREATMENT?

LOCAL.—Surgery in extreme cases. Prismatic glasses to enable the muscles to overcome the insufficiency.

IRRITATION OF THE OCULAR NERVES.

WHAT ARE THE CAUSES OF IRRITATION OF THE OCULAR NERVES?

The first stages of any of the diseases which produce paralysis of the nerves may cause irritation; also reflex conditions, such as worms in children and hysterical patients.

WHAT ARE THE SYMPTOMS?

Spasm of the muscles which the nerves supply. The spasmodic form of strabismus is a most common symptom. The strabismus when due to spasm is the opposite of that produced by paralysis; for instance, spasm of the external rectus would produce a divergent strabismus; paralysis of the external rectus would produce convergent strabismus. Nystagmus is often a symptom, and, also, conjugated deviation of the head and eyes. The deviation of the head is due to the efforts of the patient to overcome the effects of the strabismus.

WHAT IS THE TREATMENT?

SURGICAL.—Severing the fibres of the muscle affected may benefit some cases.

REMEDIAL.—*Belladonna.*—Strabismus due to spasmodic action of muscles, or when resulting from brain affections; the eye-balls turn convulsively in a circle.

Cina.—Strabismus dependent upon worms; child has a pale, sickly look: blue rings around the eyes; pains about the umbilicus: frequent urination: boring at the nose.

Cyclamen.—Convergent strabismus arising from irritation produced by worms; convulsions; convergent strabismus after measles; double vision.

Hyoscyamus.—Spasmodic action of the internal rectus muscle, with diplopia.

TRIGEMINUS.

WHAT DISEASES MAY AFFECT THE TRIGEMINUS?

Paralysis, spasm, anesthesia, paresthesia, trophic disturbances, neuralgia and tic douloureux.

PARALYSIS.

WHAT ARE THE CAUSES OF PARALYSIS OF THE MOTOR BRANCH OF THE FIFTH NERVE?

Disease within the pons, such as hemorrhage, softening, tumors, and sclerosis; disease of the base of the brain, such as tumors, meningitis, and caries of the bone; neuritis, caused by a syphilitic gout or exposure to cold; injury of the trigeminus, due to fracture of the skull and gun-shot wounds through the mouth or nasal cavity.

WHAT ARE THE SYMPTOMS?

There is paralysis of the temporal, masseter, and pterygoid muscles which diminishes the power of mastication upon the affected side and the ability to move the lower jaw toward the healthy side. If the nerve is diseased there is also muscular wasting.

WHAT IS THE TREATMENT?

When the paralysis is due to injury surgical measures may be necessary. If due to an inflammation of the nerve remedies which are suitable for the various forms of neuritis should be administered. Electricity, both galvanic and faradic currents, is useful.

SPASM.

WHAT ARE THE CAUSES OF SPASM OF THE FIFTH NERVE?

Spasm due to primary disease of the nerve itself is extremely rare, but it is symptomatic of some general condition and may be clonic or tonic.

Tonic spasm occurs in tetanus, producing trismus or lock-jaw, and also in hysteria.

Clonic spasm is present in general convulsions.

ANESTHESIA.

WHAT ARE THE CAUSES OF ANESTHESIA OF THE FIFTH NERVE?

It is most commonly found in syphilitic diseases of the brain. It may also occur in hysteria with anesthesia of other parts of the body, and in organic disease of the nerve centres. Degeneration of the nerve, due to a neuritis or injury which divides the nerve, will produce anesthesia as well as motor paralysis.

PARESTHESIA.

WHAT ARE THE CAUSES OF PARESTHESIA OF THE FIFTH NERVE?

It occurs in persons who are extremely nervous or hysterical, and also in those suffering from anemia. It is one of the symptoms, too, of neurasthenia.

WHAT ARE THE SYMPTOMS?

A peculiar numbness, tingling, or formication which does not amount to pain, and is not exactly of the character of anesthesia. It is nearly constant and is extremely annoying. It follows the course of the trigeminal nerve.

The treatment will be given under tic douloureux.

TROPHIC DISTURBANCES.

WHAT TROPHIC CHANGES OCCUR FROM DISEASE OF THE FIFTH NERVE?

The secretions from the mucous membranes, the lachrymal and salivary glands are lessened in paralysis of the fifth nerve and are increased by its irritation. Wounds of the

cheek heal slowly, and there is a tendency toward ulceration. Sometimes the teeth become loosened. Inflammation of the eye-ball, opacity of the cornea, and corneal ulceration are due to the lack of secretion of the lachrymal duct which usually washes all foreign substance from the eye. Herpes zoster along the course of the nerve has been observed. Flushings and pallor of the face, and swellings are also due to irritation of these nerves.

NEURALGIA.

HOW MANY FORMS OF FACIAL NEURALGIA ARE THERE?

Two. The symptomatic and the essential, or tic douloureux.

WHAT ARE THE CAUSES OF SYMPTOMATIC FACIAL NEURALGIA?

Any conditions which tend to lower the vital energies of the patient, such as anemia, over-work, frequent child-bearing, lack of proper nutrition, exposure to cold, diseased teeth, diseases of the eye and nose, gout, diabetes, syphilis, malaria, rheumatism, injury, epilepsy, and neurasthenia. Females suffer more frequently than males, and the majority of cases occur in the first half of life. The attacks occur with greater frequency during the winter and spring.

WHAT ARE THE SYMPTOMS?

Sharp, intense pain along the nerve, with frequent exacerbations and remissions. It is lancinating in character, though it may be dull and heavy at times, and may last for days. When the pain is due to anemia it is usually on the top of the head. When caused by eye troubles it is over the orbits. When caused by decayed teeth it is in the region of the teeth, or in the temporal or cervical region. When due to gastric disturbances it is between the eyes.

WHAT IS THE TREATMENT?

The relief of the condition which produces the neuralgia will stop it. The remedies will be given under tic douloureux.

TIC DOULOUREUX, PROSOPALGIA, EPILEPTIFORM NEURALGIA, FOTHERGILL'S NEURALGIA.

WHAT ARE THE CAUSES OF TIC DOULOUROUX?

It occurs only in adult life, and women are more liable to the disease than men. Any influences which impair the general health may produce the disease. Over-fatigue, either of body or mind, prolonged emotional excitement, excessive use of the eyes, errors of refraction, exposure to cold, local or general; carious teeth; various toxemic influences, such as alcoholism, lead poisoning, diabetes, malaria and *la grippe*, may cause it.

WHAT IS THE PATHOLOGICAL ANATOMY?

A low grade of neuritis has sometimes been found, but as a rule the nerve does not show much change. Diseases of the brain, such as tumors, are occasionally present.

WHAT ARE THE SYMPTOMS?

The pains are of an intense, darting character, which usually commence in the upper lip and at the side of the nose. If the ophthalmic division of the nerve be affected the pain is referred to the supra-orbital region, radiates over the front of the head and is sometimes felt in the eyeball itself and also in the eyelid. If the superior maxillary division is involved the pain is in the region between the orbit and the mouth, and it is then called infra-orbital neuralgia. The term "epileptiform" is given to some of these neuralgias on account of the sudden and intense onset, lasting a short time, and then passing suddenly away to return again in a little while. Many trophic disturbances accompany this form of neuralgia. The pain is made worse by eating, talking, protruding the tongue and breathing in cold air. It is confined to one side of the head, The pain is usually accompanied by lachrymation and fluent discharge from the nose, and the expression of the patient is that of intense agony.

WHAT IS THE TREATMENT?

Local.—Surgical measures are sometimes used, such as resection of the nerve; but such a procedure is not usually

advisable. The removal of the cause should be accomplished if possible. Hot applications, especially dry heat, are often of great service.

REMEDIAL.—*Aconite.*—Neuralgia of the left side; face red and hot; restlessness; anguish; rolling about; screaming; numbness; heavy feeling of the whole face; burning and numbness of lips and mouth.

Belladonna.—Violent neuralgia originating in the right temple, spreading over the orbit to the right cheek, worse from the slightest motion of the jaws, such as chewing, talking; pains come suddenly and disappear as suddenly as they come.

Chelidonium.—Supra-orbital neuralgia of the right side; eyes water; throbbing pains; shivering; temples tender on pressure; better from cold water, worse from warmth and at night in bed; violent tearing in the right zygoma.

Cedron.—Chronic, intermittent prosopalgia always coming on at 7 or 8 p. m. and lasting two to four hours with spasmodic distortion of the muscles corresponding to the affected region; pain starts from a carious tooth, and is of an intense, burning character.

Cinchona officinalis.—Violent tearing, laming, burning pain in the left side of the face every morning; skin sensitive to the least touch; pains excessive in the supra-orbital region.

Kalmia latifolia.—Facial neuralgia involving the teeth of the upper jaw, not from caries but after exposure to cold; pains rending, agonizing or stupefying; worse by worry or mental exertion.

Mezereum.—Lightning-like pains extending from the left infra-orbital foramen to the temples, corners of the mouth, into the cheek and teeth, down the neck into the shoulder, with increased lachrymal secretion; pains come on suddenly while talking or eating; injected conjunctiva; great sensitiveness of the parts to touch.

Platinum.—Tearing, boring pain in the right side of the face near the canthus of the eye, and extending along the nasal branch of the nerve; profuse lachrymation and swelling of face; attack generally comes on at night.

Gelseminum.—Facial neuralgia in nervous women; pain in right temple gradually growing more severe extend-

ing into the ear, and spreading to the eye and side of the head; sudden darting, acute pains from region of ear and side of head to supra-orbital region; jaws affected spasmodically.

Spigelia.—Prosopalgia, mostly left-sided, with tearing, shooting, burning into eye, malar bone and teeth, periodical from morning to sunset, worse at noon, worse from motion or noise, with lachrymation.

FACIAL NERVES.

WHAT ABNORMAL CONDITIONS OF THE FACIAL NERVES ARE OBSERVED?

Irritation, producing over-action or spasm of the muscles to which the nerve is distributed; or loss of function which produces paralysis of the muscles.

FACIAL SPASM.

WHAT ARE THE CAUSES OF FACIAL SPASM?

It may be idiopathic or due to some organic disease. Idiopathic spasm occurs after twenty years of age. That which succeeds paralysis alone occurs only in childhood. Ordinary facial spasm usually begins between forty-five and sixty. There may be an inherited neurotic tendency to insanity or epilepsy. Women suffer more frequently than men. There is usually a neuropathic constitution. Irritation of the fifth nerve by disease of the eyes or teeth, may by reflex action produce it. Organic diseases, such as tumors affecting the nerve nucleus in the pons or in the cerebral centres, cause a symptomatic spasm, but not true facial spasm. There may be a spasm due to cortical lesions of the brain. Shock, injuries or exposure may also produce the idiopathic form.

WHAT ARE THE SYMPTOMS?

The spasm usually comes on gradually, simply a momentary contraction of the muscles now and then which does not cause the patient much annoyance. The orbicularis palpebrarum and the zygomatici muscles are usually the first attacked, the eye is half closed, the angle of the

mouth drawn upwards, and the naso-labial furrow is deepened. The corrugator supercilli are sometimes involved. The spasm is clonic in nature and consists of lightning-like twitches which last for a second or two, when there is a short cessation after which the spasm is renewed. There is no pain, and the spasm is made worse by great nervous excitement, conversation, mental emotions, and exposure to light and cold. The spasm may be confined to one side of the face and is usually so confined at first, and then later the other side is involved. There is no wasting of muscles and no paralysis.

There are two kinds of facial spasm which are quite common, blepharospasm, a tonic spasm of the orbicularis palpebrarum, and nictitating spasm, a winking movement of the lids.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

There is no difficulty in diagnosing the disease, the only difficulty being in determining the cause of the spasm, whether due to organic disease of the brain or to causes within the nerve itself. If due to brain troubles there are usually other symptoms accompanying it. Idiopathic facial spasm is chronic, unilateral, and unaccompanied by pain or paralysis.

WHAT IS THE PROGNOSIS?

The prognosis is usually unfavorable, as the disease is very rarely cured, especially if it has lasted for some time. If there be a reflex cause and that cause can be relieved the prognosis will be more favorable.

WHAT IS THE TREATMENT?

REMEDIAL.—*Belladonna.*—Spasm of facial muscles; continual winking and trembling of eyelids; convulsive movements of the facial muscles, with distortion of the mouth.

Such remedies as *Agaricus*, *Arnica*, *Baryta carbonica*, *Hyoscyamus*, *Ignatia*, *Opium*, *Pulsatilla*, *Sulphur* and *Strychninum* will be of the most benefit.

FACIAL PARALYSIS.

WHAT ARE THE CAUSES OF FACIAL PARALYSIS?

The nucleus of the nerve within the pons may be damaged by various focal lesions; the nerve itself may be compressed at the base of the brain by tumors, or injured by meningitis or hemorrhage. Within its canal in the temporal bone it may suffer from the various forms of inflammation of the ear, and diseases of the ear in children in cases where there is caries of the bone and suppuration of the middle ear. Fracture of the base of the skull which passes through the petrous portion of the temporal bone may cause the nerve to be torn or bruised. A blow on the face may also cause paralysis. Inflammation of the nerve, due to cold, is a common cause. Pressure upon the nerve by sleeping with the head upon the hand may induce a true pressure paralysis; also the pressure of forceps upon the side of the face during delivery. Syphilis is sometimes a cause. Hemorrhage into the nerve sheath often produces it, and it has also been observed in rare cases of tabes.

WHAT IS ITS PATHOLOGICAL ANATOMY?

When the disease is due to a peripheral neuritis inflammation attacks most of the peripheral filaments of the nerve, though sometimes the inflammation may be more central. When the disease is due to organic lesions within the brain the pathology is, of course, characteristic of these conditions.

WHAT ARE THE SYMPTOMS?

The disease may come on suddenly or gradually, according to the conditions which produce it. When due to a neuritis caused by exposure to cold it will come on suddenly; but where pressure from tumors is the cause it will come on more gradually. In meningitis we would expect a sudden onset, as well as in fracture of the skull. The paralyzed side is smooth, flaccid, and without expression; the patient cannot completely shut the eyes, and when attempting to chew upon the affected side the food goes in between the cheek and teeth and he is unable to get it out. As the conjunctiva is not properly protected from dust and other for-

eign bodies floating in the air it becomes inflamed and extremely painful. The mouth is drawn at first toward the sound side. When the patient laughs this drawing of the mouth is most noticeable. If the patient is asked to close his eyes tightly the eye of the palsied side is not closed, but the eye-ball turns up, showing the sclerotic. The nostrils of the affected side do not expand during inspiration, saliva dribbles from the paralyzed side and the patient cannot pucker the mouth in whistling. Taste is lost in the anterior part of the tongue on the affected side. After a time there is wasting of the muscles of the face to a slight degree; reactions of degeneration are usually observed.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

We have to determine whether the paralysis be due to a lesion within the cerebrum, within the pons, at the base of the brain, or whether it be peripheral. If the cerebrum be affected the upper branch of the nerve is usually not much affected and the patient can close the eye. Disease involving the nuclei within the pons is rare and usually involves other cranial nerves. When due to lesions at the base of the brain there are usually brain symptoms which help to differentiate it. When the disease is at the base it is usually either syphilitic or tubercular.

WHAT IS THE PROGNOSIS?

When due to a peripheral neuritis it is generally good, although complete recovery does not take place. The disease may last for from three to five months; sometimes cases get well within a few weeks. If normal electrical irritability begins to return after a few weeks the prognosis is good.

WHAT IS THE TREATMENT?

LOCAL.—Electricity; the galvanic current after about two weeks, just strong enough to produce muscular contractions for about five minutes every other day, the negative pole being placed over the muscle to be treated.

REMEDIAL.—*Aconite.*—When due to exposure to cold winds, especially at the very beginning of the disease.

Belladonna.—In paralysis of the right side of the face; face flushed and hot; patient irritable; sleepless and worried.

Causticum.—Paralysis of the face from taking cold; right-sided.

Other remedies such as *Cocculus*, *Graphites*, *Kali muriaticum*, *Nux vomica*, *Rhus toxicodendron*, and *Stramonium* may be given according to general symptoms that may arise at the time of the attack.

AUDITORY.

WHAT DISTURBANCES OF THE AUDITORY NERVE OCCUR?

Diminished function producing deafness; increased action, causing auditory hyperesthesia or hyperacusis; and irritation of the nerve, causing tinnitus aurium.

NERVE DEAFNESS.

WHAT ARE THE CAUSES OF NERVE DEAFNESS?

It may be congenital, as in a very large number of deaf-mutes, or it may be the result of the disease in early life. Heredity is sometimes a cause, especially when there have been marriages with near relations; also disease of the labyrinth, such as acute or chronic inflammation, syphilitic disease, degenerative processes, hemorrhages, and calcareous formations. Loud noises may produce permanent deafness; as may occasionally diseases at the base of the brain, such as meningitis, morbid growths or syphilitic diseases; morbid conditions of the nerve itself, such as tumors and interstitial hemorrhage; primary degeneration, as in locomotor ataxia; softening within the pons affecting the nuclei; tumor of the corpora quadrigemina, and diseases of the temporo-sphenoidal lobe. Deafness is of functional origin in hysteria, and also occurs as a result of severe hemorrhage.

WHAT ARE THE SYMPTOMS?

The main symptom is loss of hearing, which may be accompanied by vertigo and tinnitus. In a case of nerve deafness the vibrations of the tuning-fork cannot be heard when the handle of the fork is placed in contact with the skull over the mastoid process, but can be heard when held

close to the external auditory meatus. If the deafness be due to a disease of the middle ear, or to some obstruction in the meatus, the vibrations cannot be heard when the tuning-fork is held opposite the meatus but can be heard when the handle is brought in contact with the skull. The tinnitus is a most constant symptom in these cases and it seems to the patient as if it would drive him crazy, being often very severe and intense. Nerve deafness may be either unilateral or bilateral.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The only disease from which this would have to be differentiated is when deafness is caused by disease of the middle ear, or by an obstruction of the external auditory meatus. In nerve deafness the vibrations of the tuning-fork are heard most plainly when the fork is held opposite the meatus. When due to middle-ear disease the vibrations of the tuning-fork are heard most plainly when it is in contact with the skull.

WHAT IS THE TREATMENT?

REMEDIAL. — *Calcarea carbonica.* — Ringing, buzzing, singing and hissing, or thundering in the ears; deafness with feeling as if something lay in front of the membrana tympani; deafness after use of quinine; right-sided deafness in scrofulous subjects.

China.—Ticking sound in the ear as of a distant watch; hardness of hearing with humming or roaring in the ears; something seems to be constantly before the ear.

Manganum.—Deafness as if ears were stopped, better by blowing nose, worse during cold and rainy weather: fullness of ears with difficult hearing and cracking when blowing nose or swallowing.

Phosphorus.—Difficult hearing, especially of human voice; ticking of watch only heard when held close to the ear; a sound as of the roaring of rushing waters; sensation as if foreign body were lodged in the ears.

Platinum.—Nervous deafness: great variety of noises in the ears; reports in right ear like distant thunder: ringing,

roaring or rumbling sound in the ears; sensation of coldness in the ears.

Silica.—Deafness of nervous origin; comes on suddenly after a faint ringing in the ears, and deafness with paralysis; inflammation of the labyrinth after cerebro-spinal meningitis.

AUDITORY HYPERESTHESIA.

WHAT ARE THE CAUSES OF AUDITORY HYPERESTHESIA?

It occurs most frequently as a symptom in hysteria, with acuteness of other senses. It is a common symptom in acute cerebral and general diseases.

TINNITUS AURIUM.

WHAT ARE THE CAUSES OF TINNITUS AURIUM?

Conditions which lead to disease of the labyrinth, of which gout is frequent. Neuropathic constitutions, sufferers from neuralgia and periodical headaches often have this symptom. Accumulation of cerumen, growths in the external meatus, inflammation of the middle ear, congestion, causing increased pressure within the labyrinth, irritation of the nerve endings, pachymeningitis, sunstroke, alcoholism, thickened arteries in old people with imperfect brain nutrition may produce the condition. Neurasthenic states and reflex irritations are other causes.

WHAT ARE THE SYMPTOMS?

Roaring, hissing, buzzing, ringing, singing, whistling, or thundering sounds, which seem to be in the head. The sound is usually heard in one ear but may be in both. There is no cessation of these sounds, and the patient becomes worn and weary.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The symptom itself is easily discerned, but the principal point is to discover its cause. The ear should be most carefully examined, both as to the function of hearing and its general condition, to determine whether an obstruction is in the ear which would produce the symptom, or if it be due to some disease within the labyrinth or the brain.

If the abnormal sound seems to originate in the head, and is not complicated by deafness, it is probably of central origin.

Musical sounds are also of central origin. Roaring, pulsating sounds are due to congestion or hemorrhage in the labyrinth. Gurgling, bubbling, and boiling sounds are usually due to disease of the middle ear with exudation or catarrh of the Eustachian tube.

WHAT IS THE TREATMENT?

GENERAL.—See that the ear is thoroughly cleansed of any accumulation of cerumen, pus or blood that may perhaps cause the abnormal sounds.

REMEDIAL. *Ambra grisea.*—Roaring, whistling in the ears in the afternoon.

Belladonna.—Roaring, tingling, humming and murmuring in the ears; extreme sensibility of hearing.

Chelidonium.—Sensation of wind rushing out of ears; loud roaring in ears as of a distant storm; ringing in left ear while walking; humming in ears.

Elaps.—Illusions of hearing; hears whistles and ringing; continued buzzing as if a fly was enclosed in auditory meatus.

Graphites.—Violent nocturnal roaring; ears feel stuffed at times; thundering, roaring sound before the ears; sound like air passing into the ears; hissing sound.

Nux vomica.—Humming, ringing, hissing, roaring, whistling; a whirring noise like that of a mill.

Thuja.—Noise in ears as from boiling water.

GLOSSO-PHARYNGEAL.

WHAT AFFECTIONS OF THE GLOSSO-PHARYNGEAL NERVES ARE THERE?

As the function of this nerve is not fully understood, and as its nucleus of origin is so closely associated with those of the pneumogastric and spinal accessory nerves, and as it has extensive communication with the fifth and seventh nerves, and, too, as the nerve is rarely affected alone, it is impossible to say what symptoms might occur from disease of this nerve.

PNEUMOGASTRIC.

WHAT DISEASES MAY AFFECT THE PNEUMOGASTRIC NERVE?

Its nuclei may be affected by softening, hemorrhages or degeneration. The nerve, as it emerges from the medulla, may be compressed by tumors, thickened membranes, or aneurisms of the vertebral artery. The trunk of the nerve may be injured by gunshot wounds, or by incised or lacerated wounds. It may be also damaged in surgical operations by being tied with the carotid, or divided in the removal of tumors. Neuritis following diphtheria is another condition.

WHAT ARE THE SYMPTOMS OF LESIONS OF THE PNEUMOGASTRIC NERVE?

They are of two kinds: Spasm and paralysis.

Spasm due to the irritation of the nerve causes laryngeal spasm, vomiting, and depression of the heart's action.

In paralysis there is increased frequency of the heart's action, diminished frequency of respiration, and Cheyne-Stokes breathing.

PHARYNGEAL BRANCHES.

WHAT ARE THE SYMPTOMS OF DISEASE OF THE PHARYNGEAL BRANCHES?

Paralysis of the Pharynx.—Manifested by difficulty of swallowing. Food lodges in the pharynx instead of passing into the esophagus. Small particles of liquid sometimes enter the larynx and produce spasm and choking. Liquids may pass into the nose. Food made into a pulp is swallowed better than solids.

Spasm of the Pharynx.—A functional disturbance, often due to hysteria producing the so-called "globus hystericus."

LARYNGEAL BRANCHES.

WHAT ARE THE SYMPTOMS OF DISEASE OF THE LARYNGEAL BRANCHES?

The larynx receives sensory fibres from the pneumogastric and motor fibres from the accessory portion of the spinal accessory. The branches supplying the muscles of the larynx are the superior laryngeal, which supplies the

crico-thyroid muscle and the epiglottis, and the inferior laryngeal, which supplies all the other muscles of the larynx. Disease of one or the other of these branches manifests different phenomena. The vocal cords are abducted and the glottis opened mainly by the posterior-cricoid muscles. They are adducted and the glottis closed by a number of muscles, but mainly the crico-arytenoid.

Paralysis of the Laryngeal Muscles.—This produces alteration or loss of voice, derangement of the regulation of the entrance of air during respiration, and defective movements of the vocal chords.

Spasm of the Larynx.—Commonly affects children, and may be accompanied by difficult breathing and cyanosis. It is usually nocturnal.

It is often called spasmodic croup, or laryngismus stridulus.

PULMONARY BRANCHES.

WHAT ARE THE SYMPTOMS OF DISEASE OF THE PULMONARY BRANCHES?

The muscular fibres of the bronchi are supplied by the pneumogastric. Spasmodic contractions of these fibres produce asthma; there is now no doubt but that the majority of cases of asthma are due to some irritation of the pulmonary branches of the pneumogastric.

CARDIAC BRANCHES.

WHAT ARE THE SYMPTOMS OF DISEASE OF THE CARDIAC BRANCHES?

These branches regulate the action of the heart by inhibition, and any irritation of them would produce a slowing of the heart's action. This sometimes occurs in meningitis and in rapid compression of the brain. Excessive rapidity of the heart's action is produced by paralysis of the cardiac branches, and has been observed in diphtheritic neuritis and as the result of injury of the nerve. Persons suffering from sexual neurasthenia may be troubled with irregular heart's action, due to disturbances of nutrition of the pneumogastric. Angina pectoris is caused by irritation of these branches.

ESOPHAGEAL AND GASTRIC BRANCHES.

WHAT ARE THE SYMPTOMS DUE TO DISEASE OF THESE BRANCHES?

These branches are not often disturbed in function. Vomiting may result either from reflex stimulation of the gastric branches, or by direct irritation of the pneumogastric. Gastralgia is sometimes due to irritation of these branches. The gastric crises which occur in locomotor ataxia are undoubtedly due to irritation affecting the nuclei of the pneumogastric.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The main symptoms diagnostic of disturbances of the pneumogastric are laryngeal paralysis, retarded respiration, accelerated heart's action and vomiting. The seat of the lesion can be determined only by the distribution of the symptoms.

If one vocal cord be paralyzed it would indicate pressure upon one inferior laryngeal, or upon the spinal accessory at the medulla.

Bilateral paralysis of the larynx would denote a lesion within the medulla.

WHAT IS THE PROGNOSIS?

This depends entirely upon the nature of the disease that is producing the trouble.

WHAT IS THE TREATMENT FOR THESE CONDITIONS?

GENERAL.—In pharyngeal or laryngeal paralysis, also in spasm of the pharynx and larynx, rest of the muscles is the most important consideration. In irritation of the cardiac and gastric branches attention to diet will often very largely remedy the difficulty. But for any of the lesions, whether they be organic or functional, homeopathic remedies will be of inestimable service in alleviating the distressing symptoms which are present.

REMEDIAL.—*Aconite.*—Laryngismus stridulus, after exposure to dry, cold winds; awaking in first sleep; child in agony, impatiently tossing about; loud breathing during expiration; larynx sensitive to touch and to expired air as if

denuded; voice husky, can scarcely speak; hoarseness after singing or speaking.

Causticum.—Aphonia, with rawness and tickling in the throat; dry cough; pain in the throat; must swallow continually; feels as if the throat were too narrow; paralysis of the esophagus; mucus collects in the throat and can not be raised by hawking.

Gelsemium.—Painful sensation of a lump in the esophagus that cannot be swallowed, in hysterical women; spasmodic sensations and cramp-like feeling in the gullet; paralytic dysphagia; inability to swallow or speak; spasmodic affections of the throat; nervous aphonia with dryness and burning in the throat; laryngismus stridulus.

Ipecac.—Spasm of cords; constant alternate contraction and relaxation of cords following each other in rapid succession; spasm of glotitis; blueness of face and coldness of extremities; rattling noises in air-passages during respiration; voice hollow.

Phosphorus.—Irritable acute weakness of vocal organs; cannot talk on account of pain in the larynx; laryngeal croup; aphonia; rapid sinking; rapid respiration; suffocative pressure in the upper part of the chest; larynx sensitive.

Rhus toxicodendron.—Hoarseness with roughness, scraping, or raw sensation in the larnyx from over-straining the voice; muscular exhaustion of larynx from loud and prolonged exercise of the voice; throat feels sore and stiff after straining it.

Sambucus.—Spasm of glottis; breathing is of a wheezing, crowing character, worse after midnight and from lying with head low; breathing anxious, loud and quick; child suddenly awakes nearly suffocated, sits up in bed, turns blue, gasps for breath, which he finally gets, spell passes off, and the child lies down again, to be aroused sooner or later in the same manner.

Spongia.—Great dryness of larynx; talking, singing or swallowing hurts larynx; starts suddenly from sleep with contraction of larynx; paroxysms of dyspnea and cough; constriction and suffocation during sleep; sawing respiration.

SPINAL ACCESSORY.

EXTERNAL PART.

This is really a spinal nerve and supplies the sterno-cleido-mastoid and trapezius muscles. It is purely motor and its diseases are characterized by paralysis or spasm.

WHAT ARE THE CAUSES OF DISEASES OF THE SPINAL ACCESSORY NERVE?

The nuclear cells are sometimes degenerated in progressive muscular atrophy, and in polio-myelitis-anterior. Exudation in meningitis, intra-cranial tumors or enlarged glands may compress the nerve in its course and cause various symptoms. In fractures of the skull and in diseases of the vertebræ the nerve is sometimes injured.

WHAT ARE THE SYMPTOMS?

Paralysis of the sterno-cleido-mastoid and trapezius results from degeneration or disintegration of the nerve. In paralysis of the sterno-cleido-mastoid there is an inability to rotate the head to the opposite side. In paralysis of the trapezius the power of supporting the head in the upright position is impaired, especially if the paralysis be bilateral, and it readily falls forward so that the chin rests upon the sternum. When both sterno-mastoids are paralyzed there is a tendency for the head to fall backward. The trapezius is never completely paralyzed from disease of the spinal accessory, as this nerve only supplies the upper portion of the muscle. In paralysis of the upper portion there is alteration in the contour of the outer side of the neck. It assumes a concave curve instead of a nearly straight line, as in the normal condition. In paralysis of the middle portion of the trapezius there is a slight drooping of the shoulders, the scapula recedes from the spine and the lower angle is rotated inwards on account of the unopposed action of the rhomboideus and the levator anguli scapulæ. Elevation of the arm is also somewhat impaired.

WHAT IS THE TREATMENT?

General.—Electricity and massage are of benefit in these cases.

HYPOGLOSSAL.

WHAT DISTURBANCES OF THE HYPOGLOSSAL OCCUR?

Paralysis, spasm and hemiatrophy. Paralysis is caused by degeneration of the nuclei, and forms a part of bulbar paralysis. The root fibres are sometimes damaged by softening or tumor within the medulla, and outside of the pons the fibres of origin are damaged by meningitis and various growths.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE HYPOGLOSSAL NERVE?

If one nerve only be affected the tongue deviates toward the affected side. In bilateral paralysis the tongue lies motionless within the mouth, articulation is impaired and mastication is hindered because the tongue is not able to keep the food between the teeth. There is also wasting of the tongue when the nucleus of the nerve is diseased. Neither sensation nor taste is impaired.

WHAT ARE THE SYMPTOMS OF SPASM OF THE HYPOGLOSSAL NERVE?

There are stuttering and stammering on attempting to speak.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

There is no difficulty in differentiating this disease, as the paralysis and hemiatrophy, if incomplete, the affection of one nerve, and paralysis of the whole tongue, which is usually a part of bulbar paralysis, are all easily determined.

WHAT IS THE PROGNOSIS?

The paralysis depends entirely upon the causing lesion. The spasm is usually improved after a time.

WHAT IS THE TREATMENT?

In paralysis of the hypoglossal the treatment is the same as in bulbar paralysis. The spasm is often treated by educational methods, as teaching the patient to control the action of the tongue as much as possible.

PART V.

DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

SPINAL MENINGITIS.

WHAT IS SPINAL MENINGITIS?

It is an inflammation of the membranes of the spinal cord. It may be acute or chronic.

INTO WHAT TWO FORMS IS SPINAL MENINGITIS DIVIDED IN REGARD TO THE DURA MATER?

External meningitis, which begins outside of the dura mater, and internal meningitis, which begins within the dura mater.

EXTERNAL SPINAL MENINGITIS.

WHAT IS EXTERNAL SPINAL MENINGITIS?

It is an inflammation of the outer surface of the dura mater, sometimes called external pachymeningitis, or peripachymeningitis.

WHAT ARE THE CAUSES OF EXTERNAL SPINAL MENINGITIS?

It is generally due to extension of some contiguous disease, such as caries of the bones of the spine, syphilitic disease of the vertebræ, deep sacral bed-sores, ascending neuritis, psoas abscess, retro-pharyngeal abscess, and fractures of the spinal column.

WHAT IS THE PATHOLOGICAL ANATOMY?

The inflammation may be simple or purulent. If simple the dura mater is only reddened and slightly opaque,

with a small amount of lymph upon its surface. When purulent it is covered by a layer of pus; the fat outside the dura mater becomes quickly absorbed, and the membranes after a time are adherent to the bone.

WHAT ARE THE SYMPTOMS?

These are often complicated by those of the primary disease. They consist mainly of symptoms of irritation of motor and sensory roots, followed by symptoms of compression of these and of the spinal cord, pain in the back, in the region of the loins, which is increased by movement, stiffness of the muscles of the spine, and hyperesthesia of the skin. The rigidity of the muscles of the back is sometimes followed by paralysis, and the hyperesthesia is followed by anesthesia. There is also loss of reflex action of the paralyzed muscles, with flaccidity of the muscles. There is generally paralysis of the sphincters. Trophic disturbances manifest themselves by bed-sores and lividity of the skin. There are radiating pains extending down the limbs, with twitching, followed by paresis of the lower extremities, which goes on to paraplegia. There is also a bending backward of the trunk upon the hips.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis is made upon the presence of a primary disease followed by the symptoms just enumerated.

WHAT IS THE PROGNOSIS?

It is usually grave, because the cause of the trouble is generally a serious one.

WHAT IS THE TREATMENT?

GENERAL.—In caries of the bone, in which there is angular curvature of the spine, the treatment for Pott's disease will be of benefit.

The remedies for this disease will be given under internal spinal meningitis.

ACUTE INTERNAL SPINAL MENINGITIS.

WHAT IS ACUTE INTERNAL SPINAL MENINGITIS?

It is an inflammation of the inner surface of the dura mater, the arachnoid, and the pia mater.

WHAT ARE ITS CAUSES?

Local, such as injuries to the spine, fracture or dislocation, concussion, puncture of a spina bifida, exposure of the back to cold, extension of adjacent inflammation, inflammation due to morbid blood states, such as septicemia, or the virus which produces cerebro-spinal meningitis, tuberculosis, syphilis, typhoid fever, exposure to cold and also isolation.

WHAT IS THE PATHOLOGICAL ANATOMY?

The inflammation may be simple or purulent. It is usually wide in extent. It may be localized at certain levels. The spinal fluid is generally increased in amount. If the disease lasts for some time the pus becomes absorbed in a measure, the connective tissue is increased, and the dura, arachnoid and pia are bound to the cord. The roots of the spinal nerves are compressed by the inflammatory product which surrounds them. In tubercular meningitis the exudation is of a fibrinous character and tubercular granules are found on the pia mater, arachnoid, and on the inner surface of the dura mater.

WHAT ARE THE SYMPTOMS?

The first symptoms may be a slight pain in the back and general prostration, which are followed by a chill, fever and severe pains in the back, varying according to the locality of the inflammation; they may be felt along the whole length of the spine.

Pain.—Pain, due to irritation of the nerve roots, is eccentric, that is, radiating from the centre to the periphery, extending down the upper and lower extremities and around the trunk, comes in paroxysms and is intense. It is sharp, darting, burning, or constricting. There is usually constant pain, which is increased at times by movement or by pressure.

Muscular Spasm.—Rigidity of the muscles of the back usually accompanies the pain. It first manifests itself in the neck, as that is the most mobile part, the head is drawn backward and there is also slight stiffness of the neck. Sometimes spasm is so severe that there is complete opisthotonos, such as that which occurs in tetanus.

Hyperesthesia of the Cutaneous Surface.—This is sometimes extreme, and the least movement or pressure of the limbs, particularly the legs, will cause great pain.

Dyspnea.—Oppression of the breathing is present when there is severe spasm of the thoracic muscles.

Pulse.—This may be frequent or slow.

Temperature.—The body heat if raised will be raised only a degree or two.

Headache, Delirium and Coma.—These symptoms occur when the inflammation has extended to the membranes of the brain. If the Cheyne-Stokes respiration be present it will show that the medulla has become involved. As the disease progresses the symptoms of hyperesthesia and spasm give place to those of anesthesia and paralysis. Reflex action disappears. In a fatal termination there is sometimes considerable rise in temperature.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The pain in the back, rigidity of the neck and spine, hyperesthesia and spasm of the limbs, which are made worse upon attempting to move them at the beginning of the disease, associated with fever, are characteristic symptoms. The only difficulty in the diagnosing of the acute form of the trouble is when it runs an almost latent course and there is but little tendency to invade the nerve structures.

From myelitis it is differentiated by the severe pain which is not present in myelitis. In myelitis the paralysis comes on early.

From tetanus it is differentiated by the fever at the onset in meningitis.

WHAT IS THE PROGNOSIS?

It is grave in all cases. The greater the severity of the acute symptoms, and the earlier the symptoms of irritation give way to those of paralysis, the more serious is the prog-

nosis. The disease may terminate in a day or two, or it may last for two or three weeks and then end in death or possibly in recovery. Homeopathic treatment does more to change an unfavorable prognosis than anything else, extremely serious cases very frequently recovering under this method of treatment.

WHAT IS THE TREATMENT?

GENERAL.—Perfect rest and quiet are absolutely important throughout the whole course of the disease. Light and sound should be excluded from the room as much as possible, and all bodily and mental exertion avoided. Spinal ice-bags are sometimes of use. Occasionally the patient may find relief from some of his symptoms for a little while by lying in the prone position, but the muscular exertion which is necessary to get into the position may do more harm than good.

DIETETIC.—Nourishment should be given frequently and in small quantities. Milk, flour gruel, corn-meal gruel, and beef juice if there is not much fever, are the main articles of diet. Oat-meal or rice boiled for several hours may be given hot or cold, as the patient desires. The patient may have as large quantities of water as he wishes, but not too near the times of taking food. In cases that go on to recovery milk toast, blanc mange, plain custard, farina, eggs and wine jelly may be given.

REMEDIAL.—*Aconite.*—After exposure to cold; stiffness of the back; burning, shooting pains in the spine; numbness in the small of the back extending into lower limbs; shooting, tearing pains in legs; legs almost powerless; after sitting, numbness; excessive restlessness and tossing about.

Belladonna.—Burning, throbbing pain in the spine; back aches as if it would break; cramp-like sensation in the lumbar region; convulsive movements of the limbs; frequent startings as if electric shocks were running through the limbs.

Bryonia.—Painful stiffness of the back, worse upon the slightest motion; pain extends through the thorax and lower portion of the sternum.

Cicuta.—Pain in nape of neck; spasmodic drawing of head backwards, with tremor of the hand; back bent backwards like an arch; complete powerlessness of limbs after

spasmodic jerks; spasmodic contortions and fearful jerking of limbs; trembling of upper and lower limbs.

Cocculus.—Stiffness of the cervical muscles and great weakness; constant pain in the back shooting through the body on both sides and along the spine to the occiput; tenderness on pressure upon the vertebral spines; trembling in the back; numbness and paralytic feeling in the arms; paralytic rigidity of extremities.

Hypericum.—After a fall; slightest motion of arms or legs extorts cries; cervical vertebra very sensitive to touch; consequences of spinal concussion; lies on back, jerking head backwards; crawling in hands and feet as if they were numb.

Mercurius.—Violent pains in the spine, worse from motion; involuntary jerking in the limbs; tearing and stitching in the lower limbs at night and during motion, with sensation of coldness; paralysis of lower extremities, bladder and rectum.

CHRONIC INTERNAL SPINAL MENINGITIS.

WHAT ARE THE CAUSES OF CHRONIC INTERNAL SPINAL MENINGITIS?

This is a rare disease, occurring most frequently in adults, and in men more frequently than in women. Exhausting and debilitating influences predispose to the disease; also severe and prolonged over-exertion. Repeated exposure to cold, trauma, and the results of concussion are sometimes causes. Syphilis, chronic alcoholism and tuberculosis are other causes.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is an increase of connective tissue cells, thickening and opacity of the membranes. There is also inflammation of the nerve roots, and sometimes the spinal cord itself may be involved, producing what is called a meningo-myelitis. Induration of the spinal cord, constituting a peripheral sclerosis, is found.

WHAT ARE THE SYMPTOMS?

These, like the symptoms of the acute form, are produced by irritation of the membranes and of the spinal

nerve roots. They are much the same as those of the acute form, but come on more slowly and less severely. Pain in the back (which is made worse by movement) of a dull, heavy character, with stiffness of the back and tenderness along the spine is the most prominent symptom. Eccentric pains, sharp, severe or rheumatic in character, come in paroxysms, but are usually worse at night. Hyperesthesia of the skin may exist to a greater or less degree. The pains may also extend down the extremities, and after they have persisted for months there is a gradually increasing weakness and wasting of muscles, with the reaction of degeneration for a time, but complete loss of electrical irritability later. Inco-ordination of the lower extremities is sometimes present. There is paralysis of the sphincters, and bed-sores form later.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The pain and stiffness are sometimes taken for spinal irritation, but in the latter condition the spine is tender only in spots, the pains are not radiating, and there are no symptoms of paralysis.

In muscular rheumatism there is no tenderness of the spinal column and no increased sensitiveness of the cutaneous surfaces.

In progressive muscular atrophy the wasting is not preceded by severe pain as it is in meningitis; neither is there any hyperesthesia or anesthesia.

In locomotor ataxia there is a greater degree of ataxy but no weakness, as in meningitis.

WHAT IS THE PROGNOSIS?

As in the acute form, much can be done with homeopathic remedies.

WHAT IS THE TREATMENT?

GENERAL.—Rest is most important. In severe cases it is absolutely necessary. Everything must be done to prevent increase of pain. The prone position can be maintained with better results than in the acute form and is extremely helpful in many cases.

DIETETIC.—Good, nourishing food is of the utmost importance; and almost everything that the patient wishes that is not hard to digest can usually be given.

REMEDIAL.—*Calcarea carbonica.*—Stiffness and rigidity at the nape of the neck: pain in the small of the back; can scarcely rise from his seat; violent, boring, tearing, burning pains extending down the back, with inclination to move about; painful stiffness in the back, making change of posture very difficult; weakness and lameness of extremities; heaviness and painful weight in limbs, with great fatigue on walking; sweating of lower extremities.

Nux vomica.—Backache in lumbar region worse in bed; sudden loss of power in legs in the morning; hands and feet go to sleep easily; stiffness and tension in hollow of knees; numbness and formication along the spine and into extremities; ataxic symptoms in meningitis from alcoholism; incomplete paralysis of upper and lower extremities; aching, drawing or bruised pain in the limbs, worse during motion and at night.

Rhus toxicodendron.—Stiffness in small of back, painful on motion; while sitting small of back aches, as also after long stooping or bending; pain as if bruised in small of back whenever he lies quietly upon it or sits still; sensation as if something were grasping the back and as if the flesh had been beaten; better when moving slowly about; inflammation of the membranes from getting wet or sleeping on damp ground; sensation of stiffness on first moving limbs after rest.

Secale cornutum.—Lassitude, weakness, heaviness, trembling of limbs; most violent convulsive movements of limbs occur several times a day; paraplegia; violent pain in the back, especially in sacral region; anesthesia and paralysis of limbs; painful contraction of flexor muscles; paralysis of bladder and rectum.

SPINAL HEMORRHAGE.

INTO WHAT PART OF THE SPINAL CORD MAY HEMORRHAGE TAKE PLACE?

It may occur outside of the dura mater, between it and the bones, when it is called extra-meningeal; or it may occur within the dura mater, being then called intra-meningeal;

within the arachnoid, when it is called sub-arachnoid; or into the cord itself, hematomyelia.

WHAT ARE THE CAUSES OF SPINAL HEMORRHAGE?

It may occur in newly born children or in adults. If in a newly born child it is due to rupture of the vessels during birth. Immediate causes of hemorrhage are injuries, such as fracture of the vertebra, blows or falls on the back, and falls on the feet or buttocks. It may take place as a result of severe convulsions of epilepsy, eclampsia, tetanus, chorea, from strychnia poisoning, and also from severe muscular exertion. Some diseases in which there is hemorrhagic tendency may induce it, such as purpura, acute specific diseases, small-pox, yellow fever, etc.

WHAT IS THE PATHOLOGICAL ANATOMY?

Hemorrhage outside of the dura mater comes from the large plexus of veins which lie between it and the bone. The extravasation may be small or large in extent. The blood usually coagulates wholly or partially.

Hemorrhage within the membranes usually comes from the vessels of the pia mater. It may surround the cord for a few inches, or it may fill the whole sub-arachnoid cavity. The spinal fluid is often blood-stained, and the spinal cord is discolored and compressed.

When with the cord itself there is usually a fatty degeneration of the coats of the vessels. The clot may be absorbed and a cavity left in the cord, or it may be the cause of myelitis.

WHAT ARE THE SYMPTOMS?

Sudden, burning pain in the back, which corresponds in position with the seat of the hemorrhage. This pain is accompanied by pains along the course of the nerves, of a darting, burning character, and of great intensity. There are also sensations of tingling, numbness and formication, accompanied by hyperesthesia.

Muscular spasm generally accompanies the pain and involves the vertebral muscles, causing rigidity of the spine, or even opisthotonos; it also involves the muscles supplied

by the nerves in which the pain is felt, and those muscles supplied from the cord below the seat of hemorrhage.

Later there is weakness or paralysis and anesthesia. There may also be spasmodic retention of urine.

In hemorrhage of the cord the symptoms are those of numbness, weakness, sudden paraplegia, with anesthesia and paralysis of the sphincters. There is not so much rigidity of the muscles as when the meninges alone are affected. In spinal hemorrhage the mind is usually clear and the patient feels the paralysis and numbness commencing in the extremities and gradually extending up the body, involving the trunk, intercostal muscles and diaphragm, when death take place from suffocation, sometimes within a few hours from the onset.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

When the above symptoms follow injury of some kind the diagnosis is usually plain.

Meningitis comes on more gradually and has fever at first; while in spinal hemorrhage if there is any fever it comes on later.

WHAT IS THE PROGNOSIS?

In severe cases death usually takes place in a few hours. If the symptoms have reached their height in a few hours and then gradually begin to subside the patient may recover, if secondary inflammation does not take place. The prognosis may often be determined by the rapidity of onset. Hemorrhage in the upper portion of the cord is extremely grave because the respiratory organs may be interfered with.

WHAT IS THE TREATMENT?

GENERAL.—Absolute rest is the first thing to be enforced. The patient should lie prone or on one side, not on the back. Ice-bags up and down the spine often give much relief.

REMEDIAL.—*Aconite.*—Numbness of small of back, extending into the lower limbs; stiffness of the back; burning, shooting pains in the spine; formication in arms, hands and fingers; great restlessness and nervous excitability; tossing about.

Arnica.—After injury; violent pains in the spine; tingling in the back; back painful, as if it had been beaten; extremities feel weary, as if bruised by blows; great heaviness and soreness of limbs; numbness; swelling or tingling of the limbs; weakness; weariness; sensation of being bruised; bed feels too hard; paralysis of extremities.

Hamamelis.—Severe pains in hypogastric regions, extending down the legs; back feels as if it would break; bruised feeling in upper arms and shoulders; stiffness in arms and shoulders; great soreness of affected muscles; general lassitude and feeling of weakness.

Veratrum viride.—Opisthotonos, with great arterial excitement; hands and feet cold; shocks in limbs; heat and redness down the spine; back of head hot; galvanic shocks of great violence in limbs; severe aching of back of neck and shoulders.

MYELITIS.

WHAT IS MYELITIS, AND WHAT ARE ITS DIFFERENT FORMS?

It is an inflammation of the spinal cord. It may be acute, subacute, or chronic. Acute myelitis is that form in which the symptoms reach their height in less than two weeks. In subacute myelitis the symptoms reach their height in from two to six weeks. In chronic myelitis the symptoms reach their height in a very much longer time than six weeks.

The inflammation is transverse when the whole thickness of the cord is involved in a small vertical extent; when a larger area of the cord is inflamed it is called diffuse; when a small spot is inflamed it is called focal; when many small spots, either near each other or distant, are inflamed it is called disseminated; when the gray matter around the central canal is involved it is called central myelitis.

Acute transverse myelitis is the most common form.

ACUTE TRANSVERSE MYELITIS.

WHAT ARE ITS CAUSES?

It may occur at all ages, and males suffer more frequently than females. Injury, such as lacerations, bruises, punctured wounds, or concussion (producing the so-called "railway skin"); violent action of the muscles of the spinal

cord, exposure to cold when the body is heated, lying on the damp ground or snow, cold bathing, over-exertion, sudden suppression of the menses, or other long-continued discharges; over-action of the cord; toxic blood states, such as typhus, typhoid fever, variola, measles and puerperal fever; syphilis, alcoholism, inflammation of the uterus, bladder, and kidneys; lead or arsenical poison may be causes.

WHAT IS THE PATHOLOGICAL ANATOMY?

The cord at the inflamed part appears soft, swollen, either red and hyperemic, or pale and anemic. If there be extravasation of blood there is a condition called red softening, in which the affected part is diffluent, like cream. If the effusion has lasted for some time there is a chocolate color given to the softened part. After a longer time the blood pigment changes and there is a yellow appearance called yellow softening. In some cases the destruction of nerve elements is very much greater than the extravasation of blood, so that we have a condition called white softening. There is a degeneration of fibres, granules, myelin, and granule corpuscles mixed with numerous red blood discs and leucocytes and other cell elements in the tissues. Connective tissue changes are most noticeable in the white matter. The axis-cylinders and myelin sheaths are often very much disintegrated. After a few weeks the processes of absorption and cicatrization and secondary degeneration occur; and the fatty and granular matters and leucocytes disappear. The axis-cylinder, which is usually the last destroyed, recuperates first; but when the nerve cell is destroyed it never develops again.

WHAT ARE THE SYMPTOMS?

For some days there may be a general malaise, with shivering, headache, depression, loss of appetite, fever, with some weakness of the lower extremities, accompanied by numbness, tingling or burning sensation, pain in the back and limbs, which, however, soon ceases; occasionally cramps and twitchings of the limbs are present.

Paralysis.—This generally comes on rapidly, and may reach a considerable degree in a few hours. If the patient be walking he may suddenly feel his legs becoming very

heavy, as if they were made of lead. If he sits down to rest awhile and then tries to walk again he finds that he cannot stand, and in a few hours is unable to raise his legs. With these symptoms there is numbness and tingling of the extremities. Sometimes the paralysis comes on during the night, the patient going to bed perfectly well and awakening with a complete paraplegia; but generally the onset of the paralysis occupies several days or a week, or even two weeks. When the paralysis has reached its height it is usually complete, but it may be incomplete. If it is incomplete in the lower extremities the flexor muscles suffer more than the extensors. If the cervical region be inflamed there may be paralysis and atrophy of the arms and also of the intercostals. An inflammation above this point causes paralysis of the respiratory organs, and death ensues quickly.

Sensation.—Sensation is lost in severe cases up to the level of the lesion. In slight cases only a partial loss is noticed. Sometimes there is general hyperesthesia. Sensation of a girdle around the body is very distinct, and is due to irritation of the nerve roots. Analgesia is sometimes present.

Reflex Action.—This is sometime decreased at the beginning of the disease, but later becomes excessive. When the lesion is in the lumbar region there is diminished reflex action and flaccidity of the paralyzed muscles, which waste and show reaction of degeneration. If the dorsal region is affected reflexes are present and after a while become exaggerated.

The Sphincters.—They are early affected. For a short time there is retention of urine, and later there is incontinence. If the lumbar region be affected there is incontinence from the beginning.

Cystitis.—Cystitis is due to the inability of the bladder to expel all of the urine, which remains, becomes alkaline, and acts as a source of irritation, thus producing inflammation.

Constipation.—This symptom is present in a very marked degree.

Trophic Disturbances.—The temperature of the paralyzed limbs may be raised at first, but later is lower than normal. The skin is usually very dry, its nutrition suffers

to a very great degree and bed-sores frequently result. The slightest pressure upon the skin may produce a blister, which is afterward followed by sloughing. Atrophy of the muscles involved is usually present.

Temperature.—The temperature is usually from 101 to 103 degrees, reaches its height the second or third day, remains so for several days, then gradually falls and in a week is about normal.

Contractures and Spasms.—These symptoms develop with flexion of the legs, and deformities are produced. If the cervical region be attacked the arms are involved as well as the legs.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

It has to be differentiated from hemorrhage, acute ascending paralysis, multiple neuritis, meningitis, and hysterical paralysis.

Spinal hemorrhage comes on suddenly and does not have fever at first.

In acute ascending paralysis there are no sensory symptoms and no atrophy.

In meningitis there is severe pain in the back with rigidity of the muscles of the back and limbs.

In hysteria there is no atrophy and not much rigidity.

WHAT IS THE PROGNOSIS?

In favorable cases when improvement commences it is very slow and continues for a long time, when recovery may be complete. If there is much damage of the cord, recovery is not complete and there is usually left some weakness with spasm and wasting. Generally there is some improvement in every case, which may be followed by frequent relapses and periods of improvement which continue for some years. After two weeks from the time of onset the muscles may be tested by the faradic current. If there is no response to the current it shows that the gray matter of the cord is inflamed: and if there is no change in electrical irritability from normal the muscles will probably recover their tone, as it shows there is no nutritive change taking place in the nerves and that the gray matter is not involved. Under this condition the prognosis is favorable.

In favorable cases the improvement begins to take place in a week or two. Cystitis and bedsores may produce such a degree of exhaustion as to cause death. In very severe cases paralysis of the respiratory organs occurs and death takes place quickly.

WHAT IS THE TREATMENT?

General.—Rest is of the first importance, as in other spinal cord diseases. Everything possible must be done to prevent bedsores. The bed clothing should be clean, and great care used to keep it free from wrinkles. The patient should be bathed frequently, and no urine should be allowed to remain upon the surface of the body. Frequent change of position is desirable to prevent bedsores. Solutions of tannin, alum, or alcohol may be used upon the dependent surfaces to prevent chafing.

Dietetic.—Milk, eggs, rice, toast, farina and blanc mange may be given in the first part of the disease, being followed during convalescence by a full nutrition diet of potatoes, meat, such as beef and mutton, chicken, fish broiled, and such green vegetables as lima beans and string beans. Over-feeding should be avoided, because it is likely to make the constipation worse.

Remedial.—*Aconite.*—When due to exposure to cold; tingling commencing in the feet and spreading upward; legs stiff when moved; almost powerless after sitting, with numbness; creeping pains in the fingers; great restlessness; whole body feels as heavy as lead; faintness on attempting to sit up; formication now in one part, now in another.

Anacardium.—Paralysis of single parts; wants to lie or sit continually; can scarcely move a hand; trembling weakness of limbs; sensation as if a band were tied around the body; cramp-like pain in muscles, with contraction of joints.

Arnica.—When due to injury; tingling in the back; pain in spine as if not capable of carrying the body; formication; lame feeling in lower limbs; must change position often; bed or chair seems too hard; tingling in the legs; great heaviness in the limbs; tearing pain in limbs, with soreness, trembling, numbness, swelling, or tingling; great sinking of strength.

Arsenicum.—Burning in the spine; paralysis of the lower third of the spinal cord; loss of strength in the small of the back; weakness and numbness of lower extremities with paralysis: swelling of the feet: general lack of will power in upper and lower extremities: numbness and sensation of heaviness; cold hands and feet.

Dulcamara.—Myelitis after taking cold during menstruation; pain in small of back as after stooping a long time; paralysis of arms, they are icy cold, especially during rest: weariness, prostration and languor; urine passed involuntarily.

Gelsemium.—Dull aching in lumbar and sacral regions; cannot walk; muscles will not obey; paraplegia; early stage of myelitis; tired sensation of the arms which steadily increases; fatigue of lower limbs after slight exertion: heaviness; weight; loss of voluntary motions; calves of the legs feel bruised: paroxysmal pains in lower extremities: trembling of all the limbs; great nervous excitement.

Rhus toxicodendron.—Myelitis in rheumatic subjects: great heaviness, weakness, and weariness of legs, with aching pains and inability to rest in any position but for a moment: when walking legs feel as if made of wood: sensation of stiffness on first moving limbs after rest: paresis of limbs with numb sensation and difficulty of moving the back: cramp-like pain in the limbs; spine affected from getting wet.

CHRONIC MYELITIS.

WHAT ARE THE CAUSES OF CHRONIC MYELITIS?

The disease occurs most commonly in middle adult life, and more frequently in women than in men. Frequent exposure to cold or wet continued for a long time is a very common cause of the chronic form. Injury, repeated overexertion, sexual excess, shock, infectious fever, syphilis and lead poisoning may produce it: or it may be secondary to the acute form of myelitis. Chronic alcoholism is also a cause.

WHAT IS THE PATHOLOGICAL ANATOMY?

The pia mater is generally thickened over the inflamed portion, the cord has a grayish, discolored look, and may also

be shrunken. There is an irregular increase of interstitial tissue which causes a wasting of the nerve fibres, which undergo destructive changes from the beginning. After a time all fibres disappear in the inflamed area.

WHAT ARE THE SYMPTOMS?

They come on very gradually. The patient notices first that he gets tired very easily after walking a little while, his legs are heavy, with a prickling and numb sensation in the feet and sometimes slight pain in the back; or there may be a feeling of constriction about the trunk like a girdle. The legs feel stiff and there are exaggerated reflexes. There is loss of sexual power, and a tendency toward retention of urine. After a few months there is paresis of the lower extremities with some rigidity of the limbs, anesthesia, analgesia, and occasionally pain is felt in the back. There may be also some wasting of the muscles. Later the urine has to be drawn. When the patient walks, it is with a stiff, shuffling gait. The arms become weak and stiff, and there is also some wasting, anesthesia and pain. In the last stages, the paraplegia becomes complete and the patient has to keep to his bed. There is marked atrophy of the legs, contractures and rigidity. Cystitis and inflammation of the kidneys often develop, or the patient dies from some intercurrent disease, such as pneumonia or typhoid fever.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

From progressive muscular atrophy it is distinguished by the involvement of the sphincters and the sensory disturbances.

In meningitis there is more pain in the back, with rigidity of the muscles of the back.

In locomotor ataxia there is no marked weakness of the legs, and there is the ataxic gait.

WHAT IS THE PROGNOSIS?

It may last for three to fifteen or twenty years. It is usually considered incurable, but proper hygienic and medicinal treatment may stay the progress of the disease very materially for many years. The longer it takes the disease

to reach its height, and the greater the destruction of the spinal cord, the less liability to amelioration.

WHAT IS THE TREATMENT?

GENERAL.—Moderate exercise as long as the patient can keep about, is of great value; but all exercise should stop short of fatigue: plenty of sleep and good, nourishing food are of the utmost importance. Freedom from mental worry so far as possible.

REMEDIAL.—Remedies which were suitable for the acute form will be found of value here.

POLIO-MYELITIS-ANTERIOR, ACUTE ATROPHIC PARALYSIS, INFANTILE SPINAL PARALYSIS.

WHAT IS POLIO-MYELITIS-ANTERIOR?

It is an inflammation of the gray matter of the anterior cornua of the spinal cord.

WHAT ARE ITS CAUSES?

It occurs at all ages but is by far most frequent during the first ten years of life. The great majority of cases occur during hot weather. Over-exercise and chilling the body when heated may sometimes cause it. Measles, scarlet fever, injuries, such as a fall, striking on the head or back, are other causes. Sometimes the child seems perfectly well before the attack comes on and no cause can be found.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is an acute exudative inflammation with destruction of tissue without suppuration in the anterior horns of gray matter, particularly in the lumbar and cervical enlargements. The multipolar cells are destroyed by inflammation, the spinal nerve fibres degenerate as a result of defective nutrition, and the muscles which they supply undergo rapid fatty degeneration and atrophy. Later there is sclerosis of the lateral columns of the cord.

WHAT ARE THE SYMPTOMS?

Prodromal.—There may be first vomiting, loss of appetite, fever, and all the symptoms of indigestion; or possibly

a diarrhea may have been present for a few days before the active symptoms manifest themselves.

Paralysis.—The child may have been put to bed at night after suffering from gastric symptoms, or he may have been perfectly well on retiring but when taken up in the morning all of the limbs are found to be paralyzed, or only the lower extremities, or even only one arm or one leg. In some cases the child may be walking along and suddenly fall down with both legs powerless. Improvement will begin slowly in one leg and wasting in the other. Sometimes a tingling will be felt in one arm and in a few minutes the whole arm may become powerless; the other arm soon becomes affected also, then the lower extremities, until all of the members are paralyzed. Improvement usually begins in the member affected last, and only the member first affected may remain weak and wasted. Occasionally there will only be left weakness and wasting of certain sets of muscles in the limbs. The paralysis reaches its height from one to four days, remains at its height from one to six weeks, when improvement gradually commences.

Figure 28.
Infantile paralysis from polio-myelitis-anterior.

Atrophy.—In two or three weeks wasting of the paralyzed limb or sets of muscles will be noticed.

Reflex Action.—This is lost in the paralyzed limb and no knee-jerk can be obtained.

Fever.—Fever is usually present, temperature running up to 101° or 102°, or 104° in severe cases. It may last a few hours or days, and then pass away.

Convulsions.—Convulsions may attend the onset in young children, and be followed by paralysis of one or more members. Sometimes the convulsions are followed by coma and the child may remain in that state for three or four days, and then gradually begin to recover. These cerebral symp-

toms always pass away with the other symptoms of general disturbance.

Electrical Reactions.—At the end of the first week there is loss of faradic irritability in the paralyzed muscles, but they soon begin to manifest reactions of degeneration.

Deformities.—Tendons which support joints become lax when the muscles are paralyzed, the articular surfaces fall apart, and dislocation may result. This is more common with the shoulder joint. Talipes equinus is common, on account of the paralysis of the muscles on the anterior portion of the leg and contraction of the posterior muscles. In the forearm the pronators may become involved and the supinators escape. There are also apt to be paralysis of the extensors of the hand and contraction of the flexors. Retarded growth of the bones in the affected limb, causing the limb to remain shorter than normal, frequently occurs. Permanent shortening of muscles may produce displacement of the parts to which they are attached.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis of polio-myelitis-anterior can easily be made by remembering the age of the patient, the sudden onset of the paralysis, which quickly begins to improve: the absence of sensory symptoms and bladder or rectal symptoms, the reaction of degeneration in the paralyzed muscles and the arrested growth of the limb.

From progressive muscular atrophy it is distinguished by the preceding paralysis, which was rapidly developed, and the reactions of degeneration in the paralyzed muscles.

From pseudo-hypertrophic paralysis it is distinguished by the initial fever, and atrophy of muscles instead of hypertrophy.

From multiple neuritis by the absence of sensory symptoms such as pain, anesthesia, paresthesia, etc.

WHAT IS THE PROGNOSIS?

If the inflammation be in the cervical region, there may be paralysis of the muscles of respiration, and death will be the result, but such a condition is extremely rare. After ten days from the onset of the disease if there is no response

to the faradic current in the paralyzed muscles, and if there are reactions of degeneration and if atrophy has commenced, these muscles will probably not recover their tone. whether they be all of the muscles of a limb or only certain sets of muscles. In the chronic stage the amount of recovery depends upon the degree of wasting, and the duration of the case. If there is no sign of returning power within three months, there will not be a great amount of recovery. If at the end of a couple of months there is some response to the faradic current, improvement may be considerable at the end of a few months more.

WHAT IS THE TREATMENT?

GENERAL.—The same as that of myelitis, which has been given.

REMEDIAL.—*Aconite.*—May be given during the stage of fever, but will not be of use after the paralysis manifests itself.

Belladonna.—Paralytic, drawing pressure, with weakness in right upper arm and forearm, with feeling of weakness of the whole arm; heaviness and lameness of legs and feet; paralysis of lower extremities; tired feeling in the limbs; hands and feet become cold.

Causticum.—Paralytic weakness of limbs; contraction of limbs; paralysis of upper extremities; paralysis of single parts caused by exposure to cold wind or draught; restlessness of the body in the evening; gradually appearing paralysis.

Cocculus.—Knees sink down from weakness; totters while walking and threatens to fall to one side; legs become more and more useless until complete paralysis is present; paralytic immobility of lower limbs extending from sacrum; great lassitude of the whole body; prostration and exhausting sweat of the whole body.

Gelsemium.—Paraplegia; heaviness and weight in the limbs; loss of voluntary motion; great fatigue of lower limbs after slight exertion; coldness of extremities, especially of feet, as if in cold water; spinal weakness from exhaustion in the early stage of polio-myelitis-anterior.

Phosphorus.—Loss of power in all the limbs; paralysis confined to upper and lower extremities; complete paralysis

after exanthematic diseases; cerebro-spinal system depressed; over-sensitiveness of all the senses.

Rhus toxicodendron. — Paralysis of lower extremities after exposure to wet; great heaviness, weakness, and weariness of the legs; paralysis of the limbs after great physical exertion; great restlessness; is obliged to turn in every direction on account of internal uneasiness.

ACUTE ASCENDING PARALYSIS.

WHAT IS ACUTE ASCENDING PARALYSIS?

It is a rapidly developing motor paralysis which commences first in the lower extremities, then quickly involves the trunk, arms, and finally the muscles of respiration.

WHAT ARE THE CAUSES?

It occurs more frequently in females, and generally between the ages of twenty and forty. Alcoholism, severe exposure to cold, toxemic states of the blood during convalescence from small pox, diphtheria, typhoid fever, after the febrile condition has passed away for some weeks, are causes. It may sometimes follow traumatic conditions, usually after an interval when the wound seems to be healed. It may sometimes occur in syphilitic patients.

WHAT IS THE PATHOLOGICAL ANATOMY?

Even by the most careful observers no pathological changes have been found after death. It is probable that the symptoms of paralysis are due to poisoning of the nerve elements by some substance within the blood.

WHAT ARE THE SYMPTOMS?

There may be premonitory symptoms, such as general malaise, pains in the head and back, accompanied by numbness and tingling in the extremities about to be paralyzed. Soon the patient notices a rapidly increasing weakness of the lower extremities, which may go on to complete paralysis in a few hours. The feet are first paralyzed, then the legs, and finally the thighs. After the lower extremities have completely lost their function, the trunk is next involved,

then the upper extremities, and finally the muscles of respiration. Complete paralysis may not take place in all the members of the body for two or three days. There may also be an inability to swallow, so that the patient has to be fed through a tube. The muscles of articulation become involved to such a degree that the speech is unintelligible. The lips may be paralyzed. The eye muscles may also be affected so that there may be an inequality of the pupils or a slight strabismus. The tingling in the extremities may be followed by hyperesthesia of the skin and tenderness of the muscles. There may also be some blunting of the sensibilities, but no complete anesthesia. In the early stages reflex action is lost, but as the case recovers it returns. The muscles in the paralyzed limbs are flaccid and relaxed, and there is usually no wasting and no change of electrical irritability. The sphincters are not involved. The mind is perfectly clear, but the patient may not be able to express himself either by speech or sign.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The rapidity of onset, with the ascending character of the paralysis, loss of reflex action, without much pain or loss of sensation, and the absence of atrophy of the muscles if the patient survives, usually make the disease easy to diagnose.

From polio-myelitis-anterior it is distinguished by the absence of atrophy of the muscles and the reactions of degeneration.

From meningeal hemorrhage by the absence of pain.

WHAT IS THE PROGNOSIS?

The disease may run its course and end fatally in forty-eight hours, death being due to paralysis of the respiratory muscles. The greater majority of fatal cases end in about a week. Sometimes the disease does not attain its height for two, three or even four weeks, and then it begins to abate, the limbs gradually regaining their power. Recovery of the muscles is usually opposite to that of their invasion. In favorable cases recovery may be complete in three or four months, sometimes in a few weeks.

WHAT IS THE TREATMENT?

GENERAL.—Perfect rest in bed is absolutely necessary. If the paralysis be due to exposure to cold, hot water bottles at the feet and around the trunk and limbs, with plenty of covering over the patient, will help to mitigate the symptoms.

DIETETIC.—Food which is easily assimilated should be administered; and when there is difficulty in swallowing liquid food should be given through a stomach-tube. Milk, eggs beaten up in milk, soups, gruels and long-boiled rice will be the articles most commonly used during the first few weeks.

REMEDIAL.—*Aconite.*—When due to exposure to cold, with excessive restlessness and tossing about; great muscular weakness and prostration; numbness and tingling in the paralyzed limbs.

Alumina.—Great exhaustion of strength after a long walk, with faint and tired feeling which compels him to sit down; sitting increases the weakness; and finally complete paralysis with numbness in the limbs takes place.

Cocculus.—Great weakness of the whole body, with trembling and exhausting sweat from the least exertion; knees sink down from weakness; paralysis of extremities comes on; soles of feet go to sleep while sitting; hands and feet cold and face pale.

Conium.—Paralysis of the voluntary muscles creeping from below upwards, unaccompanied by pains or derangement of intellectual faculties; paralysis of the muscles of deglutition and respiration.

Gelsemium.—Complete relaxation and prostration of the whole muscular system, with entire motor paralysis; numbness and coldness of hands and feet; speech thick; great drowsiness; loss of sight; tingling, pricking and crawling of the limbs; mental exertion causes a sense of helplessness from brain weakness.

Rhus toxicodendron.—Paralysis of the limbs with numb sensation, in consequence of getting wet or after great exertion, with lameness and soreness of the muscles; twitchings of the limbs and muscles; great restlessness at night.

DIVERS' PARALYSIS OR CAISSON DISEASE.

WHAT IS DIVERS' PARALYSIS OR CAISSON DISEASE?

It is a form of paralysis which occurs in persons working beneath the water when they are exposed to considerable pressure, the paralysis coming on soon after their return to the surface. Persons so affected have generally been working at a depth of forty to ninety feet below the surface and have been subject to a pressure of about four atmospheres.

WHAT IS THE PATHOLOGICAL ANATOMY?

There are usually congestions and small hemorrhages of the spinal cord, with some destruction of nerve tissue. There may also be some occlusion of small vessels with softening of different portions of the cord and brain. A myelitis may ensue as the result of reactive inflammation.

WHAT ARE THE SYMPTOMS?

Soon after the patient has returned to the surface, within a half hour or an hour, there is noticed considerable pain in the ears and in the larger joints. The legs feel heavy and are also weak; and in a few minutes the patient is unable to move them. There is anesthesia in the paralyzed members; and in severe cases there is paralysis of the sphincters. The paralysis may take the form of paraplegia or hemiplegia, and may be complete or incomplete according to the severity of the case. The arms are seldom involved alone. In cases of hemiplegia, the paralysis is not as severe as in paraplegia and passes away in a few hours or a few days. In some cases there is sudden loss of consciousness which soon deepens into a comatose condition with irregular breathing and signs of paralysis of the heart.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The method of onset of the paralysis occurring in divers upon their return to the surface, after having worked for some time at great depths in the water, are sufficient indications for the diagnosis.

WHAT IS THE TREATMENT?

The main treatment is prophylactic. Men working at great depths below the water should work only a half hour or an hour and then rest for several hours. The same treatment, both general and remedial, which was outlined in acute ascending paralysis will be beneficial here.

LOCOMOTOR ATAXIA—TABES DORSALIS.

WHAT IS LOCOMOTOR ATAXIA—TABES DORSALIS?

It is an inability to co-ordinate the limbs when moving from place to place.

WHAT ARE THE CAUSES?

A neuropathic heredity is found in some cases, which is manifested in the form of epilepsy, insanity, or other diseases of the nervous system. Males suffer ten times more frequently than females. About one-half of the cases begin between thirty and forty years of age, and about one-quarter between forty and fifty, and some cases between twenty and thirty. One cause more than all others produces the disease and that is the results of syphilis. It is found that a large majority of cases give a history of chancre, though the disease is not strictly speaking a syphilitic disease, but is secondary to the results of syphilis. Locomotor ataxia occurs more frequently in the city than in the country, because syphilis is more frequent in the city. Injury such as produces concussion of the spine, exposure to cold and wet, great mental anxiety, excessive fatigue and over-exertion, various acute diseases, such as rheumatism and typhoid fever, and alcoholic excesses produce the disease. There is a secondary form of locomotor ataxia, especially in syphilitic subjects, which follows some other spinal cord diseases, such as myelitis and syphilitic gummata of the cord.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is first a thickening of the coats of the spinal arteries with a change in their calibre. The lumen of the posterior arteries is increased in size, while that of the anterior is decreased. As a result of this change in the size of

the vessels, there is an increased quantity of blood in the posterior portions of the cord. On account of this hyperemic condition of the posterior portions of the cord there is an increase of the connective tissue in that portion of the cord which, after it becomes organized, begins to contract and causes atrophy and destruction of the nervous elements of the cord—a true sclerosis or hardening. The most marked changes are found in the lower dorsal and upper lumbar segments of the cord. In these positions the columns of Burdach are most affected; but when the change is in the cervical segments the columns of Goll are more markedly changed. The posterior nerve roots are atrophied up to their ganglia. As these changes occur in the posterior portions of the cord the symptoms must necessarily be those affecting sensation and co-ordination, and this we find to be the fact.

Figure 29.
Attitude in locomotor ataxia. front view.

WHAT ARE THE SYMPTOMS?

Fulgurating Pains.—During the stage of invasion a sharp, shooting, lightning-like pain is felt, perhaps in the anterior tibial portion of the leg, which is extremely severe, lasts but a second, and leaves a sore feeling behind it. In a few months the same pain may occur again, in a few weeks still again, and so on, increasing in frequency until it occurs several times a day. This may be the first symptom of the disease, and may have been present for months before inco-ordination comes on.

Inco-ordination of Lower Extremities.— The first symptom of inco-ordination may be noticed when the patient is bending over the washstand with his eyes closed while washing his face. He finds that there is a

tendency to fall forwards, and he has to brace himself in order to prevent his doing so. A little later he finds that he cannot walk in the dark because he is not able to place his feet in the proper position to walk unless he can see them. If asked to close his eyes and walk along a straight line he will not be able to do so. There is no muscular weakness or wasting with these symptoms of inco-ordination.

Reflex Action.—This is lost early in the disease on account of the interference of the function of the motor-sensory arc.

Bladder Symptoms.—During the first stage of the disease there may be an inability to void urine freely, micturition being sluggish. This may be followed by a tendency toward incontinence and the bladder may not be perfectly emptied. The retention may be complete or there may be overflow-incontinence.

Figure 30.
Attitude in locomotor ataxia, side view.

Sexual Symptoms.—There may be loss of sexual power early in the disease, or it may be much increased; but this latter condition is rare.

Constipation.—This is an extremely common symptom, but there may be a paralysis of the sphincter ani.

Gait.—When walking these patients usually lift their feet high, as if stepping over something, and bring them down heavily upon the floor. There is difficulty in keeping the balance when turning quickly, and the patient may fall if he is not careful. The eyes are kept fixed constantly upon the ground so as to watch each place where the step is to be taken. The foot is raised too high, thrown forward too far, and brought down too suddenly, the whole foot striking the ground at one time, giving a peculiar stamping noise.

Attitude.—When standing the feet have to be placed far apart in order to increase the base of support and to maintain the equilibrium.

Brach-Romberg Symptom.—This symptom is present in the majority of cases.

Inco-ordination of Upper Extremities.—The arms are sometimes affected in the same manner as the legs, but usually not to such a degree, and some cases may escape entirely. There is first a difficulty in feeding one's self—an inability to guide the food to the mouth. When attempting to button and unbutton his clothing there is a fumbling of the fingers with the buttons, which prevents the patient from accomplishing what he desires to do. The handwriting may be materially changed in some cases.

Anesthesia.—Anesthesia is frequently observed in the lower extremities, especially in the soles of the feet, so that the patient is not able to determine whether his feet are upon the ground or not.

Perverted Sensation.—When walking along the street there may be a sensation as if there were pebbles in the shoes; the feet feel as if there were cushions filled with air under them, or as if the patient was stepping on velvet with his bare feet.

Delayed Sensation.—An interval of from two to thirty seconds may be noticed between the contact of a pin in the extremities before the pain is felt by the patient.

Argyll-Robertson Pupil.—This symptom is present when the cilio-spinal axis is affected.

Paralysis of Ocular Muscles.—This may be transient, lasting for a few days or weeks, or it may be permanent and complete, and may occur at any stage. Ptosis and strabismus, and even ophthalmoplegia, may be present.

Atrophy of the Optic Nerve.—Sometimes an early symptom, commencing even before inco-ordination is developed, and perhaps the condition for which the patient consults the physician instead of the inco-ordination, which at this time may be very slight.

Tropic Disturbances.—Local sweating of the palms and soles, or of one side of the head, herpes, thickening of the soles of the feet, with blisters and perforating ulcers of the foot, may be present. Changes in the bones of a joint have been noticed, the bones becoming brittle. There is also a tendency towards inflammation and swelling which is followed by induration and ossification. There may be a wear-

ing away of the cartilage and wasting of the heads of the bones, and ossification of the ligaments. There may be also either an atrophy or hypertrophy. The hypertrophy is due to the development of new osseous tissue. There is a tendency towards spontaneous fractures and dislocations.

Gastric Crises.—Occasional attacks of severe pain in the epigastrium, passing through to the back, accompanied by vomiting and irregularity of the heart's action, have been observed.

Laryngeal Crises.—There may be true laryngeal spasm, with loud inspiration and expiration, cough and dyspnea similar to those whooping-cough.

WHAT IS THE CAUSE OF THIS DISEASE?

It is divided into three stages: (1). In which there is simple loss of knee-jerk, fulgurating pains, Argyll-Robertson pupil and the Brach-Romberg symptom. (2). Distinct inco-ordination upon attempting to walk. (3). In which walking can be accomplished only by the aid of another person. This third stage may go on until there is an absolute inability to stand. This is also a stage of complications in which involvement of the cerebral vessels, acute myelitis, and general paralysis of the insane may come on.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis of tabes is determined by the combination of the symptoms given. When there is loss of knee-jerk with fulgurating pains and the Brach-Romberg symptom there is reason to suspect locomotor ataxia.

In myelitis there are paralysis and exaggeration of reflexes.

WHAT IS THE PROGNOSIS?

Some patients have been cured when in the first stage. In the last stage not much can be done except to make the patient as comfortable as possible until death intervenes, usually from some intercurrent disease. Locomotor ataxia may last for years.

WHAT IS THE TREATMENT?

General.—Hygienic measures should be carefully attended to. Frequent bathing, plenty of sleep, regular work

and exercise, if the patient is able to carry them out, should be insisted upon. Sometimes the patient must give up business in order to be relieved. Every means which tends to conserve vital energy should be used. Electricity is of use as a general tonic, both galvanic and faradic currents being used.

Dietetic.—Wholesome, nutritious food which is easily digested should be given. During the gastric crises liquid food may be of most use. Soft-boiled rice, milk, gluten flour, gruels of various kinds, farina, meat broths and beef juice are most suitable.

Remedial.—*Alumina.*—Great muscular weakness and impairment of co-ordination; weakness of bladder and sexual organs; body totters when the eyes are closed; ptosis and diplopia; pain in the soles of the feet on stepping as if they were soft and swollen.

Argentum nitricum.—Cannot walk in the dark without reeling; legs feel as if made of wood, or padded, with insensibility to touch; tottering, irresolute gait; diminished warmth; cannot walk with eyes shut; stands unsteadily; loss of pupillary reflexes; atrophy of the optic nerve; gastric crises; retention of urine; loss of sexual desire; fulgurating pains.

Belladonna.—In the early stage, with inco-ordination of both upper and lower extremities; raises the feet slowly and puts the heel down with great force; when walking raises his legs as if he had to pass over an obstacle; fulgurating pains.

Phosphorus.—Atrophy of the optic nerve with flashes of light; trembling, especially of the hands while writing; great nervous prostration; fulgurating pains in different parts of the body, excited by the slightest chill; great sexual excitement.

Picric acid.—In the early stages when there is great sexual desire; great weakness of the legs which tremble; numbness and crawling in the legs, with trembling and pricking as if from needles; extremities cold.

Physostigma.—Neuralgic pains, sometimes in the upper arms and again in lower limbs; pain, dull, grinding, crushing; gradual loss of motion of limbs and great prostration of the whole muscular system; staggering gait, as if drunk; he must see where he is going.

Rhus toxicodendron.—Loss of power of co-ordination of lower extremities; staggers; takes long strides; steps higher than usual; tearing pains during rest.

Secale cornutum.—Absence of knee-jerk; fulgurating pains; ataxia; difficult, staggering gait; complete inability to walk, not from want of power but on account of the peculiar unfitness to perform light movements with limbs and hands; feeling as if walking on velvet.

Zincum.—Beginning of locomotor ataxia; fulgurating pains are marked and intense; twitchings in various muscles; the whole body jerks during sleep; lassitude, prostration, and pains in the limbs, with aching in the lumbar region; burning pain in the tibia; impotency.

PRIMARY SPASTIC PARAPLEGIA, OR PRIMARY LATERAL SCLEROSIS.

WHAT IS PRIMARY SPASTIC PARAPLEGIA?

It is degeneration of the antero-lateral columns of the spinal cord, producing weakness of the lower extremities with spasm.

WHAT ARE ITS CAUSES?

An inherited tendency toward neuropathic conditions, syphilis, concussion of the spine, such as a fall on the back, in which the spastic and paraplegic symptoms do not come on for two or three years after the fall: repeated exposure to wet and cold: childbirth or abortion; inflammation of the knee-joints, and, in the congenital form, injury to the brain during birth. The disease occurs most frequently between the ages of twenty and forty, and with about equal frequency in males and females. There is a congenital form or infantile form of some authors which takes place during the early years of childhood.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is a degeneration of the pyramidal tracts, with the usual increase of connective tissue and wasting of nerve fibres. In some cases there are large numbers of granule cells in the diseased parts. The degeneration undoubtedly begins in the nerve elements themselves, and many fibres

can be seen scattered through the hardened area. This degeneration may be traced into the medulla, pons, and cerebral hemispheres.

WHAT ARE THE SYMPTOMS?

Weakness of the Legs.—Gradually increasing weakness is the symptom first noticed. The patient finds that he gets tired very easily, that after a short walk his legs feel heavy and that if he gets very tired he has to drag one after the other with great difficulty. Sometimes there may be a weakness of one leg before the other is affected.

Rigidity of the Extremities.—With the gradually increasing weakness rigidity of the extremities is noticed; there is a slight stiffness of the legs on getting out of bed in the morning, which passes away after the patient has moved around a little while. After a few months it is noticed that the limbs become rigid after the patient has been sitting a short time, and it is only with considerable difficulty and after continued flexing and extending of the limbs that he is enabled to walk. The spasm is more noticeable in the extensor muscles, and therefore the limb is kept in a straightened position when the patient is sitting. By grasping the leg firmly above the knee it may be gradually flexed and extended until the rigidity passes away. If, however, during this process of flexion the leg be fully extended it will spring back quickly into a rigid position, much like the blade of a clasp-knife when it is opened; hence it is called "clasp-knife rigidity."

Exaggerated Reflexes.—The reflexes are most marked. The knee-jerk is excessive, and sometimes by tapping the patella tendon when the leg is straight and the patient is lying in bed the whole limb will be raised from the bed. Ankle-clonus is also marked. If the patient in sitting happens to rest the ball of the foot upon the floor there will be extremely severe clonic contractions of the muscles of the lower leg until he grasps the knee with his hands and forces the heel down to the floor.

The Gait.—The gait is extremely characteristic. In advanced stages when the patient attempts to walk he has to carry the leg forward as a rigid whole, and scuffs the foot along the ground because he is not able to flex the knee or

ankle. There is also a tendency for the legs to become locked, on account of spasm of the adductor muscles of the thigh.

Sensory Symptoms.—Sensory symptoms are generally absent, there being at most only a heavy, dull pain in the extremities due to the spasm of the muscles.

Trophic Changes.—The muscles are sometimes enlarged on account of their continued over-action. There is never any atrophy.

The Infantile Form.—This form resembles very closely the same condition in the adult. There are the same spasm of the extensors and exaggerated reflexes, but there is not such a degree of rigidity and neither is there apt to be an ankle-clonus. Contracture of the calf muscles is usually so marked that it is a very serious hindrance to walking, even though the strength of the limbs may be enough. The child after a time is usually able to overcome the spasm and will be able to walk, although very late. Sometimes there is a swinging oscillation of the body which lasts through life.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The weakness, rigidity, and exaggerated reflexes of the lower extremities, without trophic changes or sensory symptoms, will always be sufficient to make the diagnosis complete, as there is no other disease which has this tripod of symptoms.

WHAT IS THE PROGNOSIS?

If the disease has not reached an advanced stage there may be an arrest of the symptoms and even considerable improvement. Complete recovery does not often occur, although in slight cases it may. If the rigidity has lasted for some time and is very marked, with excessive reflex action and great weakness, there will as a rule not be much improvement. The prognosis of the infantile form is better than that of the adult.

WHAT IS THE TREATMENT?

General.—Absolute rest will sometimes be of great service. In any case the patient must avoid over-fatigue in walking. Rubbing will sometimes relieve the spasm of the

limbs to a very great degree. A course of Turkish baths will sometimes be of great service. Electricity should never be used, as it only increases the spasm and does just what we do not wish it to do.

REMEDIAL.—*Angustura.*—Gradual increasing heaviness and weakness in the lower limbs; cannot walk quickly; limbs feel stiff; threatening paralysis of the legs with trembling of the feet; drawing in the limbs with soreness; limbs get stiff after sitting a while; a painful tension of the muscles of the thighs when moving.

Arsenicum.—Excessive weakness and exhaustion of the limbs, obliging him to lie down; contraction of limbs from paralysis of the extensors; limbs stiff, lame and cold, with occasional cold feeling all over the body; restless; constantly moving about; cannot remain seated long at one time.

Calcarea carbonica.—Weakness and trembling in the legs, especially above and below the knees; cramps of the muscles of the legs; spasmodic contraction of the tendo Achillis, with violent pain; weariness of feet so great that it seems as if they could not bear the body; sensation in feet and legs as if he had on damp stockings; contraction and rigidity of the muscles of the legs.

Lathyrus.—Paresis of lower extremities with tremulous tottering gait; tendon reflexes exaggerated; no wasting of muscles.

Phosphorus.—Paresis of the lower extremities, with partial contraction of the affected muscles; formication and tearing in the limbs; pain and stiffness in the spine, preventing walking; heaviness and sensation of fatigue, especially when ascending steps; great irritability and nervousness.

Plumbum.—Paralysis, with trembling of the lower extremities; extensor muscles more affected than flexors; painful contractions of the limbs and cramps of the muscles, with shooting and tearing pains; feet cold.

Rhus toxicodendron.—Lameness of lower extremities and joints, with stiffness on rising after being seated for a long time; paresis of the lower extremities, with dragging, slow, difficult walking; when caused by exposure to wet, strains or excessive exertion; painful stiffness; tingling and numbness, with pains in the small of the back.

ATAXIC PARAPLEGIA.

WHAT IS ATAXIC PARAPLEGIA?

It is a weakness of the lower extremities, with ataxia or inco-ordination, produced by a combined lateral and posterior sclerosis of the spinal cord.

WHAT ARE ITS CAUSES?

Syphilis occasionally; heredity rarely; exposure to cold, such as sitting in a draught after perspiring freely, severe exertion, concussion of the spine, and great sexual excess. In some cases no causes can be traced at all. It occurs most frequently in males and usually between thirty and forty years of age, but may commence as early as nineteen and as late as sixty.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is a sclerosis of both the posterior and lateral columns of the cord. It differs from the sclerosis in locomotor ataxia in not involving the posterior root zones. In the lateral columns it is the direct pyramidal tracts that are affected. It is not, strictly speaking, a true systemic disease, because it is not limited to a single system of fibres, although the pyramidal tracts are the most affected. The cerebellar tract is sometimes also affected, and there is usually some increase of connective tissue in the unaffected columns; but there are no morbid changes to be found in the gray matter.

WHAT ARE THE SYMPTOMS?

The beginning symptoms are very much like those of spastic paraplegia plus inco-ordination.

Weakness of Lower Extremities.—A symptom which gradually increases until the patient is almost unable to walk. There may be some weakness of the upper extremities, but often the legs suffer alone. The patient gets tired easily after walking a short distance.

Ataxia.—The patient becomes very unsteady on turning or walking in the dark, cannot stand with his feet together, and falls if his eyes are then closed. When lying he cannot touch a designated object with his foot when the eyes are closed on account of the inco-ordination.

Gait.—There is not the high movement as if he were stepping over something which is characteristic of locomotor ataxia, but the patient has to steady himself with a stick or catch hold of something to prevent him from falling, on account of his inability to maintain his equilibrium.

Reflexes.—There is great increase of the knee-jerk and also some ankle-clonus, which steadily increases with the inco-ordination and weakness.

Sensory Symptoms.—There is sometimes a dull pain in the legs, in the sacral region, or in the spine after fatigue. There is no loss of sensation.

Inco-ordination of the Upper Extremities.—This symptom with cramp-like spasm of the hands is marked in some cases.

Articulation.—There may be an irregular tremulousness of the mouth and tongue which produces a slight impairment of the speech.

Sexual Power.—It may be lost in the early stages of the disease.

Sphincters.—There is apt to be an inability to empty the bladder, which consequently may become largely distended, with excessively alkaline urine.

These symptoms may all gradually increase to a certain point when the ataxia is lost sight of by the increase of the paralysis and the spasm; so that in the last stages of the disease the symptoms of spastic paraplegia are most marked.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

From locomotor ataxia it may be diagnosed by the increase of knee-jerk, and also the weakness of the lower extremities.

From primary spastic paraplegia the presence of the inco-ordination will give the diagnosis.

From myelitis it is distinguished by the absence of sensory symptoms.

WHAT IS THE PROGNOSIS?

It does not cause death. Usually some intercurrent trouble such as bedsores, disease of the kidneys, or cystitis may complicate the condition. The patient may live for

many years comparatively comfortable, with the exception of his inability to move about.

WHAT IS THE TREATMENT?

It is essentially the same as that which has been given for locomotor ataxia and spastic paraplegia.

HEREDITARY ATAXIA—FRIEDREICH'S DISEASE.

WHAT IS HEREDITARY ATAXIA?

It is a form of ataxic paraplegia which occurs at an early age, and in several members of the same family. It is called Friedreich's ataxia because this physician first described the characteristic symptoms of the disease. It is a combination of lateral and posterior scleroses of the spinal cord.

WHAT ARE ITS CAUSES?

Heredity is undoubtedly one of the most prominent causes, as it occurs so frequently in several members of the same family. There may be no direct hereditary ataxia, but there is an inherited neuropathic tendency in the patient. The disease occurs between the ages of two and twenty, the seventh and eighth years of life being those in which the disease most often begins. Next frequently it occurs about the age of puberty, from twelve to sixteen. Females suffer about as frequently as males. The same sex in families are most likely to suffer. Several brothers may be affected and the sisters all escape; or several sisters may be affected and all of the brothers escape. Isolated cases occur in families, but adult cases are rare. Immediate causes cannot usually be traced.

WHAT IS THE PATHOLOGICAL ANATOMY?

It is the same as in ataxic paraplegia and locomotor ataxia combined. There is degeneration of the lateral columns as well as of the posterior columns, but it is largely in the latter. The posterior nerve roots are also usually affected. The pia mater over the posterior columns is generally thickened. There may also be some general shrinking and induration of the pons and medulla.

WHAT ARE THE SYMPTOMS?

Inco-ordination.—This symptom, commencing first in the legs and afterward in the arms, is generally the first symptom manifested. It is shown by the unsteadiness in standing and walking. It may be slight at first, but gradually increases until the feet have to be placed far apart in standing in order to increase the base of support. In walking the patient often staggers like a drunken person. When closing the eyes the unsteadiness is largely increased in some cases, but in others it makes no difference.

Jerky Inco-ordination.—Manifests in the upper extremities of arms, hands and fingers. If the patient attempts to carry a tumbler of water to his lips all kinds of angles are described by his hands before the desired point is reached. Jerking of the head upon attempting a movement is noticeable in most cases, and sometimes may amount to an irregular tremor.

Paresis of Lower Extremities.—Marked after the disease has been in existence for a little while.

Speech.—Speech is impaired later in the disease. It is of an eliding character. Syllables and words are run together, or it is jerky like the extremities.

Nystagmus.—When the eyes are moved sideways or upward. It is not present when the eyes are at rest or looking straight forward. This comes on after the limbs have become affected, but usually occurs early in the disease. There is no strabismus, no optic nerve atrophy, no diplopia, and the pupils are usually normal.

Sensory Symptoms.—Dull rheumatoid pains in the legs sometimes occur, but are never severe. There are no fulgurating pains such as are present in locomotor ataxia. Sensibility is usually normal.

Lateral Curvature of the Spine.—It sometimes results from unequal weakness of the muscles of the back.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The occurrence of jerky inco-ordination, paresis of the extremities, loss of reflex action, eliding speech, and nystagmus, in a person under twenty years of age, would probably be due to hereditary ataxia.

This disease has to be differentiated from locomotor ataxia by the absence of fulgurating pains and the involvement of the sphincters, and by the presence of weakness of the muscles and the symptoms mentioned above, in a person under twenty years of age.

From ataxic paraplegia, by the absence of the knee-jerk and the age at which the disease commences and also its occurrence in several members of the same family.

WHAT IS THE PROGNOSIS?

Serious in every case, as the disease is essentially progressive. The patient, however, may be made very comfortable and life prolonged many years.

WHAT IS THE TREATMENT?

The treatment is similar to that of other degenerative conditions which have just been mentioned. The remedies prescribed have to be given according to the indications present in the individual case. Such remedies as arsenicum, phosphorus, and argentum nitricum will perhaps be most frequently indicated.

PROGRESSIVE SPINAL MUSCULAR ATROPHY.

WHAT IS PROGRESSIVE SPINAL MUSCULAR ATROPHY?

It is a disease characterized by a slowly progressing wasting of the muscles of the extremities and trunk, with consequent paresis, without any noticeable sensory disturbances, and due to the wasting of the motor and trophic cells in the spinal cord.

WHAT ARE ITS CAUSES?

It occurs more frequently in males than in females and commences between twenty-five and forty-five years of age, but some cases may commence much earlier and others much later. Direct inheritance is not common, but an inherited neuropathic tendency is present in about one-third of the cases. Great mental anxiety, severe fright, exposure to wet or cold frequently repeated, concussion of the spine, and syphilis are some of the causes. Injury of a limb may pro-

duce a wasting of the muscles of that limb, which soon extends to other muscles of the body and finally becomes general. In many cases no cause for the muscular atrophy can be found, and sometimes causes which seem to be totally inadequate to produce the trouble have really done so.

WHAT IS THE PATHOLOGICAL ANATOMY?

In the muscles themselves there may be simply a narrowing of the muscular fibres, with the striæ farther apart than normal. There is fatty degeneration, with a granular appearance of the transverse striæ; as the disease progresses the granules become larger until there are distinct globules scattered through the sheath. Some fibres are seen in which the sheath contains only a few fatty globules. The peripheral nerves contain degenerated nerve fibres and the terminal branches which are distributed to the muscles contain a larger number. These degenerated nerve fibres are found to come only from the anterior roots. The spinal cord is softer than normal at the affected part and the lateral columns may be gray and translucent. There are also changes in the anterior cornua. Most of the large cells are degenerated. The nerve fibrilla waste and there is an increase of the small angular and stellate cells and connective tissue elements. Most of these changes are found in the cervical region when the atrophy begins in the arms; but when it begins in the legs the changes are greater in the lumbar enlargement. There is a distinct degeneration of the anterior root fibres passing from the cornua through the anterior columns. There is also generally degeneration of the pyramidal tracts, the sclerosis varying in extent according to the size of the anterior tract and the distance which it extends down the cord. Most of the fibres of these tracts seem to have completely disappeared in the sclerosed area.

WHAT ARE THE SYMPTOMS?

Pain.— Pain, usually of an aching character but not very severe, may occur in the muscles, which afterwards become wasted.

Weakness and Wasting.—These generally come on together. The weakness may attract the attention of the

patient first, or perhaps the wasting is first noticed. When the muscles are covered by the clothing the weakness is first noticed. If the patient be fat, the wasting may not be noticed for some time after the weakness has been in existence.

Hand.—When the hand is affected first an inability to perform finer movements of the hand and fingers, such as writing, may first attract the attention. Muscular wasting usually begins in one hand first, the adductor longus pollicis, the thenar muscles and the interossei being early affected. The wasting then spreads from muscle to muscle, the ball of the thumb becomes flat, the wasting extends upward, involving the flexors and extensors of the forearm, and later the upper arm and shoulder. The hand becomes thin and flattened, depressions form between the metacarpal bones on the back of the hand, and between the flexor tendons on the palm, due to the wasting of the lumbricales. Sooner or later the hand becomes deformed on account of the predominance of power in the extensors and abductors of the thumb, the so-called "ape-hand" being the result. Atrophy of the interossei and contraction of the long flexor and extensor muscles produce what is commonly known as the "claw-hand."

Forearm.—The extensor muscles are atrophied more frequently than the flexors, and the supinators usually escape altogether.

Upper Arm and Shoulder.—The deltoid first manifests the disease, and the rounded contour of the shoulder becomes changed, the wasting often so marked that the head of the humerus can be recognized beneath the acromion. Wasting of the other muscles of the upper arm and shoulder soon follow that of the deltoid. The biceps suffer more than the triceps. The supra- and infra-spinatus are also affected.

Back.—The muscles of the back are usually involved early in the disease, and in some cases the wasting begins in them. The trapezius usually suffers first, and the rhomboids and erector spinæ later. The serratus, latissimus and pectoralis major are affected later. The muscles that extend the head on the spine are sometimes affected to a very great degree, so that there is difficulty in holding the head erect.

It is generally inclined backwards, so as to balance it on the spine. If it is moved forward, it falls so that the chin rests upon the chest and can only be brought back into an upright position by inclining the trunk backwards, when with a sudden contraction of the sterno-mastoids and a jerk the head is thrown back into an upright position.

Respiratory Muscles.—These suffer in the majority of cases, and are a great menace to the life of the patient. Both the intercostals and the diaphragm may be affected.

Lower Extremities.—Atrophy of the muscles of the legs is not so common as that of the arms. The glutei, extensors of the knees and the muscles in the front and on the outer side of the lower leg are those most commonly affected. The wasting in the lower extremities usually comes on late in the disease.

Face.—The face usually escapes the atrophy and is a marked contrast to the wasted body.

Bulbar Paralysis.—It is present in some cases.

Lordosis.—Lordosis is common when the trunk and hip muscles are involved. It is usually the result of weakness of the extensors of the hip-joint which causes the pelvis to be unduly inclined forward, while the upper part of the trunk is inclined upwards.

Electrical Irritability.—This diminishes as the muscular fibres waste, but there is a response to both the galvanic and faradic currents as long as a muscular fibre lasts to be acted upon.

Fibrillary Twitchings.—They occur in almost every case and consist in repeated and brief contractions of individual parts of muscles. They are most marked when the muscles are tapped with the finger, or when the parts are exposed suddenly to the cold air.

Knee-jerk.—Usually excessive.

Ankle-clonus.—It may be obtained.

The wasting may go on until nearly all of the muscles of the body are involved, and the patient is incapacitated from moving about on account of the progressive weakness.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The gradual onset of the weakness and wasting, and the slow extension to other muscles make the diagnosis easy.

WHAT IS THE PROGNOSIS?

The prognosis is usually grave in every case, and yet there may be an arrest of the symptoms in old age. The danger to life is from extensive involvement of the respiratory muscles, and from bulbar paralysis. If the wasting has remained stationary for six months, it will usually continue unchanged. Sometimes cases may recover to a very great degree, but never completely. The disease usually runs its course in about five years, but may last for a great many years. Death is produced by some intercurrent disease, or by involvement of the muscles of deglutition and respiration.

WHAT IS THE TREATMENT?

GENERAL.— Galvanism, faradism, massage, and warm baths are often of great use. The muscles should be treated by the faradic current, while the galvanic current may be applied to the spine. Each application should not last more than ten minutes, and should not be given oftener than every other day. Never use the current long enough to make the muscles tired.

REMEDIAL.—*Argentum nitricum.*—Lassitude and weariness of forearms and legs; drawing pains in the muscles; arms heavy; wasting, particularly of the legs, with paralytic weakness; great nervousness and prostration; weakness and debility, with rigidity of various muscles; paraplegia.

Arnica.—Arms feel weary, as if bruised by blows; limbs ache as if beaten; great weakness; lassitude and sluggishness of the whole body; scarcely able to stand; everything feels too hard; fibrillation in single muscles; twitching in all the limbs; over-sensitiveness of the whole body.

Arsenicum.—Heaviness of the limbs; painful feeling of fatigue penetrating to the marrow of the bones; aching in all the limbs; excessive weakness and exhaustion in the limbs which oblige him to lie down; limbs stiff and lame; a general lack of will power in upper and lower limbs, with numbness and sensation of heaviness; contractions of limbs from paralysis of extensors.

Calcarea carbonica.—Wasting of thighs and paresis of extensor muscles; heaviness and painful weight in limbs,

and great fatigue on walking; contractions and rigidity of flexors of the arm and fingers; great nervous irritability and nervous excitement; trembling in the limbs and continuous lassitude; great loss of power on walking, especially in limbs, with exhausting sweat; great exhaustion on awaking in the morning after deep sleep.

Causticum.—Tearing and drawing in shoulder-blades; paresis of deltoid; cannot raise hand to head; rheumatic aching in shoulder; bruised pains in thighs and legs in the morning; stiffness of the legs; intolerable uneasiness of the limbs in the evening; paralytic weakness of the limbs, with contractions and rigidity; restlessness and uneasiness of body, with anxiety about heart; faint-like sinking of strength.

Gelsemium.—Fatigue of limbs after slight exercise; heaviness; weight; loss of voluntary motion; calves feel bruised; pain at night; trembling in all the limbs; limbs cold; cold hands and feet; deep-seated, dull aching in limbs and joints, attended with loss of motion; numbness; feeling as if the limbs were going to sleep.

Phosphorus.—Heaviness, weakness, and weariness in lower extremities; unable to move the limbs, which are cold; limbs tremble from every exercise; jerking of single muscles; feeling of weakness in the back as if crushed; both lower limbs so feeble that the patient is only able to stagger for a moment or two with trembling step; unsteady, stumbling gait; arms weak, can hardly move them, they tremble and become numb; wasting of hands, with numbness and insensibility of fingers.

TUMORS OF THE SPINAL CORD.

WHAT ARE THE CAUSES OF TUMORS OF THE SPINAL CORD?

The causes are the same for the most part as those which produce tumors in other parts of the body. Fatty tumors usually grow outside of the dura mater, and occur in early life. Malignant tumors come on late in life. Myomata occur in middle life. Tubercular growths may occur during childhood, but usually between fifteen and thirty. Lipomatous growths are congenital. Syphilis and tuberculosis produce growths within the spinal canal. Parasitic tumors

occur, and are due to the same conditions which produce them elsewhere.

WHAT IS THE PATHOLOGICAL ANATOMY?

The growths may be outside of the dura mater, inside of the dura mater, or within the substance of the cord itself. Growths which are outside of the dura mater are called extra-dural, and spring from the membrane, or from the tissues which lie between the membrane and the bone. Growths within the dura mater may arise from the inner surface of the membrane, from the arachnoid, or from the pia mater; and the growths which develop within the substance of the cord itself may arise from the pia mater. Lipomatous growths are extra-dural, and are due to an overgrowth of fat which lies between the membranes and the bone. Enchondroma, sarcoma, and cancerous growths arise from the bones, or from the intervertebral tissue. Tumors within the dura mater are usually syphilomata, sarcomata, and myxomata. The growths within the substance of the cord are syphilomata and gliomata which are the most common; and sarcomata, myxomata, and tubercular tumors.

Growths without the dura mater are always single, but sometimes are multiple. The growths outside the cord vary in size from a pea to from one to two inches in length. Multiple tumors are small. Neuromata or sarcomata may occur on the nerve roots and are often multiple.

Tumors produce compression of the cord sometimes to such a degree that the cord may be reduced to the size of a quill, and may set up a true pressure myelitis with the tissue changes which occur in that disease. The location for the majority of tumors is the middle cervical region, and the upper and lower dorsal regions.

WHAT ARE THE SYMPTOMS?

Pain.—This is generally the most prominent symptom throughout the whole course of the disease. It may be intense, passing along the course of the nerves which arise from the spinal cord in the region of the tumor, and even in the lower extremities below the lesion. It may be sharp, acute, and of a burning character, or it may be stabbing or rending. Sometimes a dull, aching pain is felt be-

tween attacks of exaggerated pain. During the severe attacks the intensity of the pain is very great. It may be felt first on one side and then on the other. Sensitiveness over the vertebræ is often felt.

Anesthesia.—It follows the pain after destruction of the nerve has taken place.

Formication, Tingling and Numbness.—These are felt in the extremities.

Muscular Spasm.—Common when the tumor arises from the membranes. There may be rigidity of the back with pain in the region of the tumor.

Contractures.—Contractures are apt to be developed in the limbs.

Paralysis.—Paralysis of gradual onset is present in nearly all cases. Paraplegia most commonly exists, but all four extremities may become paralyzed if the growth is in the cervical region. Sometimes one leg becomes weak before the other.

Reflex Action.—If the growth be within the lumbar enlargement, reflex action is lost. If it be above the lumbar enlargement, reflex action is increased.

Atrophy.—Atrophy of the muscles which are supplied by the damaged nerve roots is a prominent symptom in late cases.

All symptoms come on gradually and the location of them depends upon the location of the growth.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis depends upon the symptoms of a focal lesion of slow development, with symptoms of irritation, such as pain and rigidity, followed by anesthesia and the other symptoms just enumerated.

From neuralgia it may be differentiated by the constancy of the symptoms; and their long continuance would lead us to believe that there was organic irritation.

From chronic transverse myelitis the differentiation is sometimes extremely difficult, but in the latter disease there is not the intense radiating pain which occurs with tumor.

The nature of the tumor will have to be determined in the same way as in tumors of the brain. If in a syphilitic subject, the tumor would probably be syphilitic in charac-

ter. If in a tubercular subject, the tumor would probably be tubercular; and in a cancerous subject, cancerous.

WHAT IS THE PROGNOSIS?

This depends upon the nature of the growth. Syphilitic growths are sometimes cured. Other growths, unless they are in such a position that they can be removed, usually go on to a fatal termination.

WHAT IS THE TREATMENT?

SURGICAL.—If the tumor be in such a position that it can be removed, surgical measures may be of use.

REMEDIAL.—*Arnica.*—Cervical vertebræ very sensitive to touch and pressure; formication in extremities; sensation like a heavy weight shooting through the spine.

Arsenicum.—Great exhaustion from the slightest exertion; stiffness and immobility of the muscles of the back; great weakness and restlessness; paraplegia; skin of legs cold, soft and flaccid; the slightest movement impossible; paralysis, with neuralgia of the limbs, and atrophy of the muscles, especially of the lower extremities.

Baryta carbonica.— In sarcomatous tumors: pain in lumbar region of spine, followed by paralysis; stiffness in the back, can hardly raise from the chair; paralysis of flexors of feet, with tension of tendons; dragging in the thighs, particularly when going upstairs, on account of paralyzed feeling in middle of thigh; constantly weak and weary, wishes to lean on something.

Calcarea carbonica.—Where there is a tubercular diathesis, with probability of a tubercular growth; pain in the small of the back, with sensation of numbness on the side of the back upon which he has been lying; weariness and a feeling of stiffness in anterior muscles of thigh in the morning on beginning to walk; coldness of thighs; emaciation of lower extremities with paralysis of extensor muscles; coldness of extremities.

Cicuta.—Frequent jerks in upper portion of the body through the dorsal vertebra and arms; great weakness in arms and legs; twitching of extremities; great depression and prostration of strength; weak and powerless muscles; lassitude and constant sleepiness.

Conium.—Lancinating pains and weight in the back, heaviness and sense of weariness in all the limbs; difficulty in using the limbs; unable to walk; numbness of fingers and toes; piercing and tearing pains in the extremities and in joints; great physical and mental debility; general spinal paralysis.

Graphites.—Pain in small of back as if broken, especially on touching it; pressing, grasping, and twitching in arms and legs; weakness in back and loins on walking; pain from sacrum down to legs; weakness of all the limbs; weak exhaustion of the whole body; atrophy of the affected parts.

Hydrastis.—Stiffness in muscles of lumbar region while bending over for a short time, causing great difficulty when assuming an erect position; dull, heavy, dragging pain across the lumbar region necessitating use of arms to rise from the seat; legs feel weak; frequent sudden attacks of faint spells, with profuse cold sweat all over; great emaciation; cancerous tumors of the cord.

SYRINGOMYELIA.

WHAT IS SYRINGOMYELIA?

It is a disease characterized by the formation of cavities within the spinal cord, and the development of gliomatosu tissue.

WHAT ARE ITS CAUSES?

It is an exceedingly rare disease, and occurs more frequently in men than in women, and in those who follow some manual occupation, laborers, butchers, etc. Trauma may sometimes produce it, and it has been known to follow pregnancy and acute infectious diseases. It occurs usually between the ages of fifteen and twenty-five.

WHAT IS THE PATHOLOGICAL ANATOMY?

The seat of the disease is most frequently in the cervical and upper dorsal regions. The cavities vary in length. They may extend the whole length of the cord, and even up into the medulla and pons. There may be one cavity or more. They may extend irregularly across the cord, backward into the posterior horn of the cord, forward into the

anterior cornua, or laterally into the white substance. In children the cavity may be simply a dilatation of the central canal surrounded by a mass of gliomatous tissue. These cavities may contain a thin gelatinous liquid. The cavities may be formed by the proliferation and subsequent breaking down of masses of gliomatous tissue which have become imprisoned in the posterior, gray matter in the course of development; or they may be formed by the persistence of the primitive tube of which the embryonic cord is composed, and after a while become enlarged by the breaking down of the embryonic tissue forming its walls. Masses of gliomatous tissue are distributed in the region of the cavity. Hemorrhages sometimes take place into the cavities.

WHAT ARE THE SYMPTOMS?

The symptoms are usually bilateral.

Analgesia.—This and the loss of temperature sense, (thermo-anesthesia) with retention of the tactile and muscular senses, are the first symptoms noticed in the upper extremities.

Pain.—Pain of a dull, heavy, aching character is often noticed in the neck and arms, which comes on gradually, and persists with more or less variation.

Atrophy.—Atrophy of the muscles is generally present with the above-mentioned symptoms. It comes on slowly, progresses gradually, much in the same way as if it were a true case of progressive muscular atrophy. It manifests itself in both extremities at about the same time.

Paresis.—Paresis of muscles of the upper extremities corresponds with the atrophy, and may go on to complete paralysis. Paresis of the muscles of the spine produces scoliosis which is common in most all cases.

Spastic Paraplegia.—Spastic paraplegia develops later in the disease, some time after the involvement of the upper extremities.

Trophic Disturbances.—The hands become swollen and red, sometimes with an edematous condition. Eczema, herpes, and bullæ may occur, and painless whitlows manifest themselves on the fingers, which may destroy the last phalanges. Erosions and ulcerations are sometimes present.

The nails become brittle, dry, and drop off. Painless fractures of the bones may occur from slight causes, and ulceration and gangrene of soft tissues which necessitate the amputation of the hand are common. There may be also an enlargement of the capsular ligaments and looseness of the joints.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

In some cases no symptoms of the disease manifest themselves, under which conditions it of course cannot be diagnosed. The main symptoms, progressive muscular atrophy, with thermo-anesthesia and analgesia of the upper extremities, with the trophic changes which have been given, usually distinguish it from any other disease.

From leprosy it may be differentiated by the absence of the characteristic thickening of the skin of the face, producing the so-called "leonine expression." In leprosy the anesthesia is distributed along the course of the nerves, and the nerves themselves are very much thickened. There is no scoliosis in leprosy, and no spastic paraplegia.

WHAT IS THE PROGNOSIS?

Always grave. Nothing can be done to stop the progress of the disease.

WHAT IS THE TREATMENT?

GENERAL.—The patient should observe all hygienic measures necessary to promote health, should have sufficient nourishing food, bathe regularly, obtain plenty of sleep, and take life generally as easy as possible.

REMEDIAL.—The remedies applicable to progressive muscular atrophy will sometimes be of some use in this condition.

MORVAN'S DISEASE—ANALGIC PANARIPIUM.

WHAT IS MORVAN'S DISEASE?

It is a name given to a certain group of symptoms first described by Morvan of Lannelis, Brittany, in 1883, probably of a neuritic character.

WHAT IS THE PATHOLOGICAL ANATOMY?

By some authors it is thought to be identical with syringomyelia, but others claim that it is due to a neuritis. In some cases that have died from what was supposed to have been Morvan's disease, cavities within the spinal cord were discovered upon examination. In other cases a neuritis has been found in the stumps that have required amputation for the trophic lesions.

WHAT ARE THE SYMPTOMS?

Neuralgic Pains.—Pains, neuralgic in character, assail one or both hands or both limbs on one side of the body. They may be slight or severe in intensity, or may even be absent.

Trophic Changes.—Felons, ulcerations which go on to necrosis causing the loss of one phalanx after another. The periods between these ulcerative processes may be weeks or even years, or they may follow each other in rapid succession.

Sensory Symptoms:—Anesthesia, thermo-anesthesia, and analgesia, distributed over the whole arm and adjoining parts of the body and even the face, are present.

Atrophy and Paresis.—These symptoms present in the muscles of the hand and forearm. The muscular wasting does not usually extend above the forearm.

Bulbar Symptoms—Usually present.

Vaso-Motor Changes.—The skin is usually livid or pale and cold to the touch.

WHAT IS THE PROGNOSIS?

The disease is slow in progress, usually extending over many years. It does not generally cause death; neither is recovery apt to take place.

WHAT IS THE TREATMENT?

SURGICAL.—Amputation of the gangrenous phalanges may often be necessary.

REMEDIAL.— *Graphites.*— Finger-nails become thick, black and rough; matrix inflamed, sometimes with throbbing and numbness, no suppuration, eczema on the back of

hands, hands numb and dead with formication extending up the arm.

Lachesis.—Numbness of finger-tips, necrosis of the tendons of the fingers with much discoloration; stinging, pricking intense pains, fistulous openings from which bony splinters are discharged.

Ledum.—Gouty nodosities on hand and finger-joints, boring pains on first joint of thumbs with feeling of stiffness, periosteum of phalanges painful on pressure, consequence of injuries of nails, perspiration in palms of hands.

Mercurius.—Inflammation of cellular tissue beneath cutis, extremely sensitive to heat and cold, fingers of both hands flexed, especially thumb, so that it is completely drawn in, weakness of the arms.

Natrum sulphuricum.—Inflammation and suppuration around roots of nails, tingling, ulcerative pain under nail in tips of fingers, blisters filled with water on the phalanx, pus around root of nail, pain more bearable out of doors.

Silica.—Nails rough, yellow, crippled, brittle; white spots, ulceration around nails, lancinating pains, inflammation extends deep into tendons, cartilages and bones, caries of fingers, atrophy and numbness of fingers, contraction of flexor tendons, profuse sweat in hands.

SPINA BIFIDA.

WHAT IS SPINA BIFIDA?

It is, as its name implies, split spine, depending upon a defect in the closure of the vertebral arches, which leads to protrusion of the membranes of the cord in a sac, forming a tumor external to the spinal column. This tumor is filled with cerebro-spinal fluid.

HOW MANY VARIETIES OF SPINA BIFIDA ARE THERE?

Four; depending upon the degree of deformity.

(1). MENINGOCELE.—In which one or more of the vertebral arches have failed to coalesce. It is the simplest form and does not disable the patient to a great degree. The tumor is covered by the skin of the back and the meninges. The cord does not extend into the sac. This form may become pedunculated, and be cured spontaneously.

(2). MENINGOMYELOCELE.— It is the most common form, and may produce death. If the patient survives there are usually paralysis and deformities in the feet and legs. The membranes and cord both protrude into the sac. On account of defective development of the corium in these cases the posterior surface of the tumor is covered by an exceedingly thin membrane, devoid of hair and sebaceous glands and of all the characteristics of true skin. There is also a failure of development of some of the elements of a part of the cord within the sac, with degeneration of the nerve trunks arising from it. As the result there is paraplegia, club-foot, and sensory and trophic disturbances below the level of the lesion.

Figure 31.
Spina bifida in child of four years. Tumor removed at eighteen.

(3). HYDROMYELOCELE OR SYRINGOMYELOCELE.— In this variety there is a dilatation of the central canal of the cord itself so that the tissues of the cord become the lining layer of the sac.

(4). MYELOCELE.—This is a very rare form. There is not only a failure of the vertebral arches to unite, but the medullary folds have also failed to coalesce, and as the result there is an opening which leads directly into the cerebro-spinal canal; the protuberance which is outside of this opening is not a sac, but a red, pulpy mass of neural tissue, and from its opening cerebro-spinal fluid constantly oozes.

WHAT ARE THE CAUSES OF SPINA BIFIDA?

It is a true developmental defect of the cord, and what the causes are which produces such an effect is not known. Heredity is sometimes a factor. Either one of the parents of the child may have had hare-lip or club-foot or hydrocephalus, showing a defective tendency of development which has taken the form of spina bifida in the individual case.

WHAT ARE THE SYMPTOMS?

There is a tumor which varies in size from one inch to six inches in diameter. It may have a broad base or it may be pedunculated. It almost always occurs in the lumbar and sacral regions because the vertebræ there are the last to become solidified. The skin over the tumor may be normal in character, or may be glossy, tough, thickened or ulcerated.

Headache.—A symptom often so intense as to cause extreme anguish, compelling the child to lie in bed for weeks at a time.

Paraplegia.—Commonly present in these cases.

Anesthesia.—Anesthesia of the regions below the tumor is present in some cases.

Hydrocephalus.—This is sometimes a complication.

Mental Defects.—They are common, owing to changes evidently within the brain itself.

General Conditions.—These children are usually puny, weak, and poorly nourished.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis is usually easy, the only difficulty is in differentiating between it and congenital tumors which may occur in the same locality. Of these growths lipoma and hygroma are the most common. These, however, are firm and resistant, and are composed of solid substances; while spina bifida is cystic and contains a fluid.

WHAT IS THE PROGNOSIS?

Nearly all cases result fatally. The sac generally ruptures, and the child dies from meningitis. Some cases, however, live to attain adult life but they are exceedingly rare.

WHAT IS THE TREATMENT?

Surgical.—Compression by means of elastic bandages or adhesive straps may give some relief to the symptoms, but does not effect a cure. Excision of the sac has been successfully accomplished in a few cases. Iodine injection has also been employed. (See standard works on surgery.)

REMEDIAL.—Remedies directed toward the general symptoms manifested, such as headache, paralysis, and general malnutrition, are sometimes effective in relieving in a measure some of the symptoms and making the patient's life more bearable. Such remedies as arsenicum, calcarea, baryta, and silica may be of use.

PART VI.

DISEASES OF MUSCLES.

ARTHRITIC MUSCULAR ATROPHY.

WHAT IS ARTHRITIC MUSCULAR ATROPHY?

It is a wasting of the muscles which move a joint, due to an inflammation of the joint. The muscles mainly affected are those which extend the inflamed joint. If it be the knee that is inflamed, the muscles of the foot or thigh will be wasted; if the ankle, the calf muscles; if the wrist, the extensor muscles of the forearm; if the elbow, the triceps; if the shoulder, the deltoid. The flexors may sometimes be involved, but the muscles of the limb near by that do not move the joint are not involved.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is simply a narrowing of the fibres of the muscles affected, with occasionally longitudinal striation. The sheath nuclei are proliferated, and the intermediate substance is increased in quantity.

WHAT ARE THE SYMPTOMS?

Simple wasting of the muscles with arthritis. If the inflammation of the joint be acute and severe, the wasting occurs rapidly, and in a week or ten days the muscles may be very greatly diminished in size. The affected muscle is involved throughout its whole length and not only the part near the affected joint. The wasting may increase for two or three weeks, and then become stationary and continue as long as the disease of the joint lasts. Electrical irritability of the diseased muscles is usually normal to both galvanic and faradic currents. There is increased myotatic irritability and increased knee-jerk if the muscles of the thigh are

affected. Ankle-clonus may be elicited when the ankle joint is affected.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The moderate degree of wasting of the muscles which involves the whole length of the muscle, associated with a preceding joint affection, will enable one to make a diagnosis.

WHAT IS THE PROGNOSIS?

If the inflammation of the joint is of short duration, the muscles will undoubtedly recover; sometimes the wasting persists for a long time after the joint has recovered.

WHAT IS THE TREATMENT?

This should be directed toward the inflammation of the joint, and not to the wasting. So long as the inflammation of the joint persists the wasting will continue, this wasting being nature's method of keeping the joint quiet until it can recover. If passive motion of the muscle, or electrical stimulation is used, it will tend to act upon the joint and keep up the inflammation; so that rest of the muscle and rest of the joint are first to be observed until the inflammatory process has subsided, after which passive motion and electrical stimulation of both joint and muscle will assist to bring them back to their normal tone.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

WHAT IS PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS?

It is an apparent enlargement of the muscles with actual wasting of their fibres.

WHAT ARE ITS CAUSES?

It occurs more frequently in males than in females, and during the developmental period of childhood. There are usually several cases in the same family. In some cases while the disease may be congenital, it is not hereditary; but in others there may be a direct heredity, and usually on the mother's side. In one case a sister had two brothers

affected and two sons, but one daughter escaped. In three-fourths of the cases the disease comes on before the tenth year. Occasionally it may not come on before the eighteenth or twentieth year. It may occur as readily among the rich as among the poor; and no general constitutional condition seems to predispose to the disease.

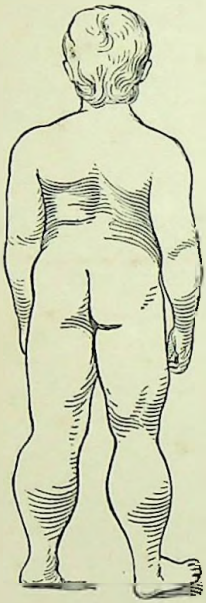


Figure 32.
Pseudo-hypertrophic paralysis.

WHAT IS THE PATHOLOGICAL ANATOMY?

The muscles are pale yellowish in color and often resemble masses of adipose tissue. Fat cells and tracts of nucleated fibrous tissue which contain muscular fibres much narrower than normal appear under the microscope. The fibres preserve their transverse striation for the most part, but when they are narrowed this may have in a measure disappeared, either by granular degeneration or by a simple wasting of the striæ. Empty sarcolemma sheaths may be seen when the narrowing of the fibres is greatest. The motor nerves are found to be normal, and the sensory nerves are lost in the paralyzed muscular tissue. There is an overgrowth of connective tissue in the muscular substance.

WHAT ARE THE SYMPTOMS?

Weakness of the Lower Extremities.—This symptom is first manifested by the child walking clumsily, stubs his toe, falls easily and rises with difficulty. When going up stairs he has to pull himself up by the banisters, or has to put his hand upon his knee after he has raised his foot to the next step above and push himself up in this way. If the child be laid on the floor on his back and asked to rise he will first turn himself over on to the anterior portion of the body, then drawing his knees up under him and placing his hands upon the floor, he lifts his body up until he is resting on his hands and knees. He next draws one leg up under him and then the other, thus resting upon his hands and feet.

The next step is to place his hands upon his knees and push himself up straight by bringing his hands further up his thighs.

Muscular Enlargement.—No changes in the size of the muscles may be noticed for some time after the weakness has come on; or the change in size of may be noticed before the weakness has manifested itself, or the enlargement may come on with the weakness. The muscles in which the enlargement most frequently commences are those of the calf. The extensors of the knee are often enlarged, and occasionally the rectus or the vastus internus alone may be increased in size. The flexors of the knee generally escape. The glutei are usually very large. There is also general increase in the size of the lumbar muscles. Next to the calf muscles the infra-spinatus is the most enlarged. The supra-spinatus and the deltoid are also affected. The triceps and biceps are sometimes enlarged, though sometimes both are wasted.

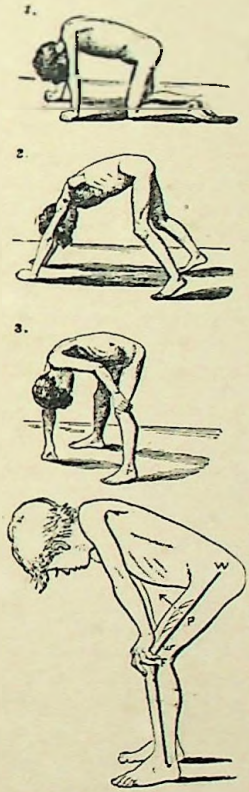


Figure 33.

Method of arising from the floor in pseudo-hypertrophic paralysis.

Gait.—The gait is of a waddling character, with the feet carried far apart in order to maintain the equilibrium.

Lordosis.—This is usually marked, and due to weakness of the extensors of the hip, in which the pelvis is inclined forward, and the upper part of the body is held far back to keep the centre of gravity of the body over the feet.

Deformities.—These frequently occur, due to shortening of the muscles least affected. The knee-joint may become fixed by contraction of the flexors. Talipes equinus may result from contraction of the calf muscles. Lateral

curvature of the spine frequently results from shortening of the muscles on one side of the body.

Knee-jerk.—Diminished and finally lost.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The peculiarity of gait, the mode of rising from the floor, the age of the patient, and the gradual increase of the weakness make the diagnosis sufficiently easy, especially when conjoined with enlargement of the muscles.

WHAT IS THE PROGNOSIS?

The prognosis is most grave. After ten or fourteen years the power of standing becomes lost on account of the weakness and contractions of the muscles. After the patient stops walking, the disease progresses rapidly, but he may live in a helpless condition for several years. Death is usually due to some intercurrent disease, such as acute pneumonia, bronchitis, or consumption. In some cases the muscular power remains fair until after puberty or even up to the age of thirty. The course of the disease is slower in girls than in boys.

WHAT IS THE TREATMENT?

GENERAL.—The patient should keep up exercise as long as possible without becoming over-fatigued. The faradic current is of great use in these cases in keeping up the muscular tone. The hygienic surroundings should be most carefully looked out for and frequent bathing, plenty of rest and good nutritious food should be had. It is only by keeping up the action of the muscles that contractures can be prevented from coming on rapidly.

REMEDIAL.—Remedies are of no avail except so far as they remove any intercurrent trouble which may arise and which has a tendency to lower the vital forces. Arsenicum, calcarea, phosphorus, lathyrus, silica and sulphur may be of use.

SIMPLE IDIOPATHIC MUSCULAR ATROPHY.

WHAT IS SIMPLE IDIOPHATIC MUSCULAR ATROPHY?

It is a form of wasting of the muscles which, while it occurs perhaps more frequently in youth, may also occur

in patients well advanced in years. It is not as frequent as pseudo-hypertrophic paralysis. There are several varieties: Erb's juvenile form, the facio-scapulo-humeral variety of Landuzy and Dejerine, characterized by wasting of the muscles of the face with those of the shoulder girths; another variety in which the affection begins in the legs; and still another form called the peroneal type of family amyotrophy by Charcot and Marie.

WHAT ARE ITS CAUSES?

There are usually no causes to be traced outside of the congenital tendency which is manifested by the occurrence of the disease in several members of the same family. Both sexes suffer. It may begin as early as two or three years and as late as sixty. When the wasting begins in the face the disease usually commences in childhood. It may occur in many generations of the same family; sometimes four or five.

WHAT IS THE PATHOLOGICAL ANATOMY?

Only the muscles themselves are affected and the changes are similar to those in pseudo-hypertrophic paralysis without the increase of interstitial tissue and the absence of fat cells. Multiplication of nuclei is sometimes seen. As the atrophy progresses the connective tissue increases until there is a hard, dense myo-sclerosis.

WHAT ARE THE SYMPTOMS?

The disease usually comes on gradually, weakness and wasting coming together and being noticed about the same time. The wasting generally begins in the upper arms and shoulder muscles, except in the facial form, when it begins in the face first. In some cases the wasting may begin in the legs and be limited to them. When the arm muscles are affected, the weakness and wasting are observed first in the biceps and triceps; but the pectoralis and latissimus dorsi may also be largely wasted. The forearm muscles usually escape, with the exception of the supinator longus. In the face there is a wasting of the zygomatici muscles, with a loss of the labio-nasal furrow; the orbicularis oris

is affected, the lower lip projects, and the face has a dull expression. The muscles of the spine are sometimes considerably wasted. In the legs the flexors of the hip and the extensors of the knee are most commonly affected. In the peroneal type the anterior tibial muscles are especially affected. Electrical irritability diminishes as the wasting progresses. There are no degenerative reactions. Myotatic irritability is also lessened or lost. Shortening of some of the muscles is sometimes noticed, producing deformities. Lordosis is present.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The most important diagnostic points are its presence in several members of the same family, and the onset of the disease before adult life. When the disease begins during advanced life it is extremely difficult to diagnose it from spinal muscular atrophy.

WHAT IS THE PROGNOSIS?

It is difficult to give the prognosis in an individual case. When the disease comes on slowly it may progress for years and then become arrested and the patient live to old age. Sometimes almost every muscle in the body becomes affected and the patient is simply a living, breathing skeleton, unable to move or help himself in any way.

WHAT IS THE TREATMENT?

The same as that for progressive spinal muscular atrophy.

THOMSEN'S DISEASE—MYOTONIA CONGENITA.

WHAT IS THOMSEN'S DISEASE?

It is an hereditary family disease characterized by the development of tonic spasms when the patient attempts voluntary movement. It is a rare disease.

WHAT ARE ITS CAUSES?

It is, as its name implies, congenital and hereditary. Males are most usually affected, and it comes on during youth.

WHAT IS THE PATHOLOGICAL ANATOMY?

The muscular fibres are found to be hypertrophied, the striations indistinct, and the nuclei increased. No other pathological changes are found in any part of the nervous system.

WHAT ARE THE SYMPTOMS?

As soon as the patient attempts to rise a rigidity of the muscles comes on which prevents him from moving for a time. In a few moments the rigidity passes away, to be renewed when the patient attempts to move again. If, however, the movements be continued, the spasm becomes weaker and weaker until it finally passes away, after which time the patient may be able to walk a long distance without becoming fatigued; but after sitting again and attempting to rise, he is seized with the same spasm of the limbs. If the hand is closed tightly, a cramp seizes the muscles and he is not able to open his hand until successive attempts have been made. If the eyes are shut tightly they cannot be opened for some time. The muscles of mastication may also be involved, but the spasm occurs most frequently in the extremities. The spasms are produced by exposure to cold and nervousness, and are overcome by muscular exercise.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

With the symptoms enumerated above present the disease cannot possibly be mistaken for anything else.

WHAT IS THE PROGNOSIS?

It is bad as far as a cure is concerned, but it does not shorten life.

WHAT IS THE TREATMENT?

GENERAL.—Muscular exercise which stops short of fatigue, regular habits, and freedom from mental worry are to be observed as far as possible.

REMEDIAL.—One homeopathic remedy seems to be indicated, and that is strychnia.

PART VII.

DISEASES OF THE SPINAL NERVES.

NEURITIS.

WHAT IS NEURITIS?

It is an inflammation of the nerves.

HOW MANY FORMS OF NEURITIS ARE THERE?

Five.

PERINEURITIS.—In which there is inflammation of the sheath of the nerves.

INTERSTITIAL NEURITIS.—Inflammation of the connective tissue binding the separate fibres together.

PARENCHYMATOUS NEURITIS.—Inflammation of the nerve elements proper.

SIMPLE NEURITIS.—Inflammation of one nerve only.

MULTIPLE NEURITIS.—Inflammation of many nerves.

Any of these forms may be acute or chronic.

WHAT ARE THE CAUSES OF NEURITIS?

Injury by contusions or compression, and by over-extension of the nerves; injuries from dislocations, fractures, and violent contractions of muscles through which the nerves pass; extension from adjacent inflammation such as when a nerve is situated near a suppurating joint or near an inflamed pleura; exposure to cold. General diseases may sometimes produce it, such as diphtheria. Metallic poisons, alcohol, etc., are other causes. These agencies may produce either an isolated or a multiple neuritis. Syphilis, cancer and leucocythemia may also produce it.

WHAT IS THE PATHOLOGICAL ANATOMY?

The changes differ according as the inflammation affects primarily the nerve sheath, the connective tissue, or the nerve elements proper. In acute inflammation the affected nerve is red and swollen. The redness is due to the distended vessels which are seen on the surface. The swelling is caused by the edema or a sero-fibrinous exudation. Leucocyte-like corpuscles surround the vessels and accumulate between it and the nerve. Sometimes there are small extravasations of blood. These changes are limited to the sheath and occur therefore in perineuritis. In interstitial neuritis lymphoid corpuscles are seen in the substance of the fasciculi between the nerve fibres. In the parenchymatous form the changes in the fibres are those of degeneration. The myelin of the white substance breaks up into segments; the axis-cylinders are interrupted; the nuclei of the sheath are increased in number, and the protoplasm around them is also increased in quantity. A little later the myelin is divided into smaller globules, and the axis-cylinder can no longer be distinguished. Finally the sheaths become empty and very narrow, and contain only nuclei at intervals.

Figure 34.
Leprous neuritis.

WHAT ARE THE SYMPTOMS?

Pain.—This is the most prominent symptom, and is felt along the inflamed nerve, and sometimes in the part to which it is distributed. It may sometimes involve the

whole limb. It may be intense, burning, and boring in character, generally worse at night, and increased by movement or by anything that produces passive congestion of the limb, such as coughing, straining at stool, etc. It may radiate to distant parts, and even be felt in the opposite limb. Sensitiveness of the whole limb to which the nerve is distributed may be present. The nerve itself may be felt much swollen at the inflamed part.

Numbness, Tingling and Hyperesthesia.—These symptoms manifest in the part to which the nerve is distributed; and later, anesthesia may be complete as the result of destruction of the nerve.

Paresis.—Paresis of the muscles supplied by the inflamed nerve is usually present.

Eruptions.—Herpes and other eruptions may sometimes occur upon the skin supplied by the affected nerve.

Atrophy.—Usually present if the inflammation passes into the chronic stage.

Electrical Reactions.—They are increased in the inflamed nerve.

Trophic Changes.—Redness of the skin; thickening; atrophy of the skin or "glossy skin"; effusion into the joints producing adhesions, and even ankylosis.

Fibrillary Twitchings.—Present with the muscular atrophy.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The distribution of the symptoms along a certain nerve will readily diagnose the condition. Sometimes the neuritis may be mistaken for a neuralgia, but the neuralgic pain is more lancinating and sharper than that of neuritis.

WHAT IS THE PROGNOSIS?

Where the neuritis is due to a local suppurative inflammation it is grave. Neuritis due to trauma is most apt to recover. Where there is a neuropathic tendency within the individual the prognosis is not so good. Intensity of the symptoms will help us as to the prognosis. When the nerve is completely degenerated it may be many months before regeneration occurs.

WHAT IS THE TREATMENT?

GENERAL.—Rest of the affected nerve is of the most importance. If the neuritis be secondary to some preceding inflammation the primary cause should be dealt with first. If due to injury or abscess in any part those conditions should be met in an appropriate manner. It may be necessary to place the limb in a splint in order to get the most perfect rest.

REMEDIAL.—*Aconite.*—When due to dry, cold weather: pains almost unbearable at night, worse from pressure and heat, better by applications of wet and cold, redness of the skin over affected nerve.

Arsenicum.—Patient extremely weak, pale and haggard from the severity of the pains, pains worse at night, compelling the patient to leave the bed, move the affected part, and walk about; better by external heat, worse from cold and rest, burning pains as if hot oil coursed through the inflamed nerve, periodical aggravation.

Belladonna.—Excruciating pains, worse by the slightest touch, especially in the evening; sharp, shooting, cutting, tearing pains commencing in wrist, shooting to the elbow, always from the periphery to the centre, better by constant motion, paralytic weakness of all the muscles.

Mercurius.—Follows belladonna; inflamed nerve feels like a cord; excessive nocturnal pain worse from the heat of the bed; tearing pains in extremities, and twitching of single muscles.

Nux vomica.—Pains worse after midnight or toward morning; numb sensation in affected parts as if they were asleep; worse from cold; better by warmth.

Pulsatilla.—Jerking, tearing, drawing pains, shifting rapidly from place to place; worse at night and from warmth; caused by protracted wet weather.

Rhus toxicodendron.—Tearing, drawing pains with sensation of numbness or formication in the affected parts; erysipelatous redness of the skin over inflamed nerve; pains worse after midnight from heat of the bed and when resting; better by motion and warmth; rheumatic paralysis from getting wet or from lying on damp ground.

NEUROMATA.

WHAT ARE NEUROMATA?

They are tumors involving nerve trunks or their fibres, consisting of an abnormal growth of nerve fibres, when they are called true neuromata; or of heterologous tissue, when they are called false neuromata. The latter type may be either sarcoma, fibroma, syphiloma, or any other variety of tumor.

WHAT ARE THE CAUSES?

The causes of neuromata are generally obscure. Heredity is sometimes a cause. Pressure, punctured wounds, or division of the nerve may produce them.

WHAT IS THE PATHOLOGICAL ANATOMY?

True neuromata may consist of medullated or non-medullated nerve fibres. Connective tissue between the nerve fibres varies in amount so that the tumor may be firm or flaccid. False neuromata are of various kinds, but fibromata are most common. Myxoma sometimes occurs, and sarcoma and carcinoma occasionally are present. Syphilitic growths occur frequently on the cranial nerves within the skull. The tumors may be single or multiple. They may vary in size from that of a minute swelling to the size of a child's head.

WHAT ARE THE SYMPTOMS?

In some cases the symptoms are entirely absent, the tumor itself being the only evidence of disease.

Pain.—It may be intense along the course of the nerve. It is usually neuralgic in character and sometimes drives the patient almost insane. In other cases it is mild in degree.

Paresis.—Paresis of the muscles supplied by the nerve may be present, and may even go on to complete paralysis of the part.

Anesthesia.—It is present when there has been destruction of the nerve by pressure of the growth. The growth itself may be extremely sensitive or may be without pain. The tumors may sometimes be felt as if they were near the surface.

Numbness and Formication.—These symptoms present in the parts supplied by the nerve.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

If there be pain, numbness, and weakness along the course of the nerve, without any sensitiveness or swelling of the nerve itself, neuroma may be suspected. If it be located superficially and can be felt, the diagnosis is then certain.

WHAT IS THE PROGNOSIS?

The tumors may grow rapidly and cause complete degeneration of the nerve. Unless the growth can be removed by surgical measures, it may produce permanent symptoms.

WHAT IS THE TREATMENT?

SURGICAL.—In the majority of cases excision is the only remedy, but the function of the nerve upon which the growth is situated must be taken into consideration before deciding upon an operation.

Remedies are of but little use except so far as they may relieve in a measure the neuralgic pains which are the result of the pressure of the growth upon the nerve.

DISEASES OF SPECIAL NERVES.

CERVICAL PLEXUS AND ITS BRANCHES.

WHAT DISEASES AFFECT THE CERVICAL PLEXUS OR ITS BRANCHES?

Neuralgia, paralysis and spasm.

CERVICO-OCCIPITAL NEURALGIA.

WHAT IS CERVICO-OCCIPITAL NEURALGIA?

It is a neuralgia referred to a part or all of the distributions of the first four cervical nerves, but it is the great occipital nerve that is most frequently affected.

WHAT ARE ITS CAUSES?

It may result from caries of the cervical vertebra, exposure to cold, strains of the muscles of the back, and it may also occur in hysteria.

WHAT ARE ITS SYMPTOMS?

Pain.—It is usually constant and dull in character, with attacks of sharp pain along the course of these nerves. Tender spots are found at the point of exit of the great occipital nerve between the mastoid process and the spine in the triangle situated between the trapezius and the sterno-mastoid muscles, and above the parietal eminence.

Hyperesthesia of the Scalp.—Prevents the patient from combing his hair, as even the moving of each hair produces pain.

These symptoms are bilateral.

WHAT IS THE PROGNOSIS?

Usually favorable, except when it occurs in patients advanced in life, when it may be a troublesome condition for many years.

WHAT IS THE TREATMENT?

The treatment will be given under the head of Neuralgia.

PHRENIC NERVE.

WHAT DISEASES AFFECT THE PHRENIC NERVE?

Paralysis and spasm.

WHAT ARE THE CAUSES OF DISEASES OF THE PHRENIC NERVE?

Diseases of the spinal cord or of its membranes, producing a disease of the roots of the nerve, may cause impaired functions of the phrenic nerve. Paralysis may sometimes follow exposure to cold. Injury from wounds in the neck sometimes occurs. Tumors of the neck pressing upon the nerve may produce disease. Hysteria may also produce paralysis of the phrenic nerve.

WHAT ARE THE SYMPTOMS?

Paralysis.—It is usually bilateral. There is no movement of the abdomen, and the epigastrium and hypochondrium are retracted. There is dyspnea upon the slightest exertion.

Spasm.—Spasm of the phrenic nerve will produce spasm of the diaphragm which may be either tonic or clonic. It always occurs as the result of tetanus and never alone. Clonic spasm of the diaphragm is commonly known as hiccough or singultus. It may be only slight or very severe, and continue until it produces death. It is usually due to reflex irritation from the abdominal viscera. Disorders of the stomach from over-eating, and gastric, hepatic and uterine diseases may cause hiccough.

Pain.—It may present along the line of attachment of the diaphragm.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The shortened respiration upon the least exertion, with retraction of the epigastrium and hypochondrium during respiration, usually very clearly suggests disease of this nerve.

WHAT IS THE PROGNOSIS?

This depends entirely upon the cause of the disease. In rare cases hiccough may continue for weeks and produce such a degree of exhaustion that the patient dies. Paralysis of the phrenic nerve may sometimes prove fatal; but the respiration may be carried on a long time by the aid of the intercostal and thoracic muscles.

WHAT IS THE TREATMENT?

GENERAL.—For paralysis rest is of great importance because then the difficulty of breathing is not present. The cause which produces the paralysis, whatever it may be, must be taken into consideration in the treatment. In clonic spasm of the diaphram or hiccough stopping respiration for a minute will sometimes stop the spasm. Drinking water, or great emotional excitement may sometimes relieve. Inhalations of nitrite of amyl in severe cases is sometimes of use.

REMEDIAL.—*Agaricus.*—Spasmodic twitching of muscles, especially face and upper extremities; hiccough shaking the whole body, worse evening and when standing; abdomen distended with gas.

Belladonna.—Violent attacks of hiccough so that they jerk the patient up, even with feeling of suffocation; hic-

cough with convulsions of arm and leg alternating; violent hiccough after midnight accompanied by profuse sweat.

Cicuta.—Loud-sounding, dangerous hiccough; nausea in morning and when eating; burning pressure at stomach and abdomen; violent vomiting, with headache; thirst and dryness of throat.

Cyclamen.—Violent hiccough while eating, and for some time afterward, or hiccough-like eructations, particularly in pregnant women; burning in esophagus, and aching pains in stomach extending through the back.

Hyoscyamus.—Hiccough after abdominal operations; violent hiccough at midnight with involuntary micturition and frothing at the mouth; frequent hiccough with cramps and rumbling in the abdomen; heartburn.

Ignatia.—Hiccough from great mental emotions in hysterical subjects.

Nux vomica.—Hiccough brought on by cold drinks, frequently coming on before dinner without any apparent cause; hiccough from over-eating and from his customary tobacco: eructations sour, bitter and rancid.

BRACHIAL PLEXUS AND ITS BRANCHES.

WHAT DISEASES AFFECT THE BRACHIAL PLEXUS OR ITS BRANCHES?

Paralysis, spasm, and neuritis.

BRACHIAL PARALYSIS.

WHAT ARE THE CAUSES OF PARALYSIS OF THE BRACHIAL PLEXUS?

Injury to the plexus itself, pressure of tumors in the neck on the nerve trunks, injury of a single nerve or neuritis. Injuries may be produced by dislocations of the shoulder.

WHAT ARE THE SYMPTOMS OF COMBINED PARALYSIS OF THE BRACHIAL NERVES?

The arm feels heavy and numb as if it were asleep. This sensation may pass away in a few moments or a few hours. Pain, tenderness, anesthesia, trophic and vaso-motor symptoms may be present in greater or less degrees. Atrophy

and changes in the electrical reactions occur when the paralysis is of long standing. Loss of power for elevation of the arm, and for flexion and extension of the forearm is usually present. In the shoulder and upper arm there is involvement of the deltoid, biceps, brachialis anticus, and supinator longus muscles. In the lower arm and hand the triceps, flexors of wrist, pronators, flexors and extensors of the fingers and the hand muscles are affected.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE POSTERIOR THORACIC NERVE?

This nerve supplies the serratus magnus muscle. There is difficulty in raising the arm above the horizontal position. The arm hangs helpless by the side. The inferior angle of the scapula is nearer the vertebral column than normal, and the posterior border projects.

WHAT ARE THE CAUSES OF PARALYSIS OF THE CIRCUMFLEX NERVE?

This nerve supplies the deltoid and teres minor muscles. There is inability to raise the arm, the shoulder becomes flattened, the ligaments of the shoulder-joint become relaxed, and there is a concavity formed underneath the acromion process. Wasting of the deltoid takes place.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE MUSCULO-CUTANEOUS NERVE?

This nerve supplies the biceps and brachialis anticus muscles. There is an inability to flex the elbow and supinate the forearm if it is pronated.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE MUSCULO-SPIRAL NERVE?

This nerve supplies most of the muscles on the back of the forearm and its paralysis produces "wrist-drop," and also paralysis of the last phalanges.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE ULNAR NERVE?

This nerve supplies the elbow and wrist joints, a number of muscles, the palmar and dorsal integument of the little

finger, and one-half of the ring finger. There is inability to close the hand tightly, and weakness of the little and ring fingers. There is a drawing back of the first phalanges, and the second and third phalanges are flexed. With atrophy of the interossei and lumbricales the so-called "claw-hand" is produced.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE MEDIAN NERVE?

This nerve supplies the pronators, flexors, the radial side of the palm, two lumbricales, the integument of the thumb, and two and a half fingers on the radial side. There is inability to fully pronate the arm, the grip is weakened, and flexion and abduction of the thumb, with flexion of the first and second fingers, are impaired.

BRACHIAL SPASM.

WHAT ARE THE SYMPTOMS OF SPASM OF THE BRANCHES OF THE BRACHIAL PLEXUS?

Spasm is the opposite of paralysis, and irritation of these nerves would produce spasm in the muscles which they supply. The distribution of these nerves has been given under paralysis.

WHAT IS THE PROGNOSIS OF THESE CONDITIONS?

The prognosis of these spasms and paralysis depends upon the causes which produce them.

WHAT IS THE TREATMENT?

GENERAL.—Mild electrical currents, galvanic and faradic, are of use to tone up the muscles paralyzed. Massage is also of use. The spasms are only controlled by the use of homeopathic remedies.

The remedies for paralysis or spasm of these nerves are the same as those for paralysis and spasm in other parts of the body—always being governed by the individual case.

BRACHIAL NEURITIS.

WHAT ARE THE CAUSES OF BRACHIAL NEURITIS?

It occurs more frequently in women than in men, and during the second half of life. It usually comes on in per-

sons who are suffering from perverted nutrition and a general lowered tone of the system. There may be a history of gout or muscular rheumatism, and they may also have been sufferers from sciatica or lumbago.

WHAT IS THE PATHOLOGICAL ANATOMY?

It is the same as in any form of neuritis; usually an inflammation of the nerve sheaths, a perineuritis.

WHAT ARE THE SYMPTOMS?

Pain.—It is greater in this form of neuritis than in any other. It is usually the first symptom and lasts for a long time, even after the inflammation has passed away. When taken in connection with the general condition of the patient, it is an extremely serious symptom. The pain may be located in the region of the scapula beneath the bone, or in the wrist or back of the forearm. In some cases the pain may first be in the plexus itself above the clavicle, or in the axilla. It is unusually sudden in onset, and severe from the first. Later there is a dull, heavy sensation in the whole arm. Sometimes the pain is acute, lancinating, stabbing, or burning, and extends along the course of the nerves. The paroxysms are induced by movements or may occur spontaneously.

Hyperesthesia.—Hyperesthesia of the skin where the pain has been is commonly met with.

Flabbiness of the Muscles.—It does not amount to real atrophy, such as occurs in an ordinary neuritis.

Arthritic Changes.—Anchylosis of the joint may take place as the result of the severity of the pain.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The severity of the pain, its location, its occurrence in persons of advanced age, with absence of marked atrophy of the muscles, lead us to a diagnosis.

WHAT IS THE PROGNOSIS?

Except in slight cases it is usually a tedious malady, and it may last for months or even a year or more. Relapses are common. Recovery is never quite complete.

WHAT IS THE TREATMENT?

The same as for the other forms of neuritis.

DORSAL NERVES.

WHAT DISEASES AFFECT THE DORSAL NERVES?

They are mainly of a sensory character, intercostal neuralgia, mammary neuralgia, and herpes zoster.

INTERCOSTAL NEURALGIA.

WHAT ARE THE CAUSES OF INTERCOSTAL NEURALGIA?

This occurs more often in women than in men, and it is very frequently due to pressure from the corset. It occurs more frequently between twenty and thirty-five years of age, and during the winter season. Patients suffering from this condition are generally anemic, neurasthenic, or debilitated by child-bearing. Lead poisoning, malaria, and dyspepsia occasionally produce it.

WHAT IS THE PATHOLOGICAL ANATOMY?

In some cases there may be a neuritis, but generally there is a poisoned blood state which produces irritation of the nerves.

WHAT ARE THE SYMPTOMS?

The disease comes on suddenly. There are sharp, stabbing pains along the course of the nerves, with tender spots here and there.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The character and location of the pain will make the diagnosis easy if pleurisy and rheumatism can be excluded, and the presence of the tender spots will enable us to exclude these diseases.

WHAT IS THE PROGNOSIS?

Generally good in most cases. The disease usually lasts from two to six weeks, but may last for several months.

WHAT IS THE TREATMENT?

The same as that for other forms of neuralgia which will be given under the head of Neuralgia.

MAMMARY NEURALGIA OR MASTODYNIA.

WHAT ARE THE CAUSES OF MAMMARY NEURALGIA?

It may be produced by local tumors of the breast, or it may occur in anemic subjects. Injury, pressure from the corsets, and large and heavy breasts may produce it. It is also common as the result of hysteria.

WHAT ARE THE SYMPTOMS?

Pain.—It is of a neuralgic character and usually in one breast.

WHAT IS THE TREATMENT?

This depends upon the cause. The remedies will be given under the head of Neuralgia.

HERPES ZOSTER OR SHINGLES.

WHAT IS HERPES ZOSTER?

It is an acute dermatitis secondary to an intercostal neuritis.

WHAT ARE ITS CAUSES?

Exposure to cold; injury; the medicinal use of arsenic; the result of rheumatic and syphilitic poisons, and lowered vitality.

WHAT ARE THE SYMPTOMS?

Pain.—It is usually one-sided and comes on gradually along the course of the intercostal nerves, neuralgic in character, acute, lancinating and severe, accompanied by tenderness of the skin. In young persons it may last but a little while and then pass away. In older persons it is persistent and is often extremely intractable.

Herpetic Eruption.—After the pain has been in existence for a little while vesicles develop along the course of the nerve, extending from the spine around one side of the body to the anterior median line. These vesicles reach their

height in about ten days. During the time the eruption is most profuse the pain is less. After it passes away the pain is usually more severe.

WHAT IS THE PROGNOSIS?

This affection lasts for a few weeks in most cases; but the length of time depends upon the age of the patient; it passes away quickly in young persons but is very persistent in older persons.

WHAT IS THE TREATMENT?

GENERAL.—Warm flannel placed over the seat of the trouble will sometimes give great relief.

REMEDIAL.—*Arsenicum.*—Confluent herpetic eruptions with intense burning and blisters; dry and parchment-like skin; nausea and marked prostration; worse after midnight and from cold of any kind; better from warmth.

Graphites.—Herpes zoster, especially on the left side; large blisters, burning when touched; herpes, exuding a sticky matter.

Iris versicolor.—Herpes zoster, especially on right side; tearing, shooting, rapidly shifting pains along the nerves: fine eruption, showing black points after scratching; great itching at night.

Mezereum.—Herpes zoster with sharp, stitching, lightning-like pains, sometimes boring, which leave the parts numb; worse in bed and from motion; vesicles form a brownish scab; neuralgic pains continue for some time after the disappearance of herpes.

Rhus toxicodendron.—Right side especially affected with incessant itching; burning, tingling, alternating with pains in the chest and dysenteric stools; worse in winter and in rheumatic subjects.

LUMBAR PLEXUS AND ITS BRANCHES.

WHAT DISEASES AFFECT THE LUMBAR PLEXUS OR ITS BRANCHES?

Paralysis, neuralgia and inflammation.

WHAT ARE THE CAUSES?

The plexus itself may be damaged by abdominal tumors, ovarian tumors, tuberculosis of the abdominal lymph nodes,

psoas abscess, and dislocation of the hip-joint. The nerve roots from which the plexus arises may be damaged by pressure in caries of the vertebra, in cancerous diseases of the bone, or tumors of the meninges, and inflammations. The plexus itself is not often the seat of primary inflammatory processes.

ANTERIOR CRURAL NERVE—PARALYSIS—NEURALGIA.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE ANTERIOR CRURAL NERVE?

There is a loss of power and wasting in the extensor muscles of the knee, and lost knee-jerk. If the nerve is damaged within the pelvis, the iliac muscle is involved, and there is inability to flex the hip or extend the knee properly.

ANESTHESIA—Involves the entire thigh, with the exception of a strip along the back of the thigh and the under side of the leg and foot, produced by paralysis of this nerve.

NEURALGIA.

WHAT ARE THE CAUSES OF NEURALGIA OF THE ANTERIOR CRURAL NERVE?

Morbid growths of the spine, or the growth within the abdominal cavity, pressing upon the nerve: also inflammation extending from the sciatic nerve through the lumbo-sacral cord.

WHAT ARE THE SYMPTOMS?

Pain.—Along the course of the nerve the pain is more or less severe, involving often the entire thigh with the exception of a small strip along the back part of the thigh and the inner side of the leg and foot.

OBTURATOR NERVE—PARALYSIS.

WHAT ARE THE CAUSES OF PARALYSIS OF THE OBTURATOR NERVE?

Most cases depend upon damage to the lumbar plexus. When it occurs alone it is due to the result of pressure during labor.

WHAT ARE THE SYMPTOMS?

The chief symptom is the loss of power of adducting the thigh. The limb affected cannot be crossed over the other.

INTERNAL POPLITEAL NERVE PARALYSIS.

WHAT ARE THE SYMPTOMS OF PARALYSIS OF THE INTERNAL POPLITEAL NERVE?

Loss of Power.—This occurs in the posterior tibial muscles, the long flexors of the toe, and the muscles of the sole of the foot. There is an inability to extend the ankle-joint or to rotate the leg inward when it is flexed.

Anesthesia.—Of the outer and posterior part of the leg and sole of the foot.

WHAT IS THE PROGNOSIS OF DISEASES OF THE SPINAL NERVES?

Many things are to be considered in making a prognosis in diseases of the nerves. The first to be considered is the cause; whether it may be removed or not, the amount of damage which has been done to the nerve, and the duration of the disease. Where the trouble has been due to growths of various kinds their prognosis will be the prognosis of the nerve trouble. Where the troubles are due to a neuritis, the amount of degeneration of the nerve must be considered in making a prognosis.

WHAT IS THE TREATMENT OF DISEASES OF THE SPINAL NERVES?

This depends upon their cause. Sometimes surgical measures, such as the removal of growths and the treatment of wounds, will be the treatment for the nerve trouble. In the various kinds of paralysis electricity may be of use over the paralyzed nerve. The faradic current is the one most frequently used.

SCIATICA.

WHAT IS SCIATICA?

It has generally been considered a neuralgia of the sciatic nerve, but of late, on account of investigations into its pathology, it has been proven to be, at least in the majority of cases, a true perineuritis.

WHAT ARE ITS CAUSES?

It is a disease which occurs mainly in middle adult life, and is more common in males than in females. There is usually a disturbance of the general nutrition, and some of

the patients are subject to gout and muscular rheumatism. There is undoubtedly an excessive excretion of uric acid. The most frequent exciting cause is exposure to cold, such as sitting on the wet grass, standing in water, or sitting in a draughty water-closet. Pressure on the nerve by sitting on the edge of some hard substance, and violent muscular contraction may produce it.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is an inflammation of the sheath of the nerve which often extends into the interstitial tissues. During the acute stages there are redness and swelling of the sheath, and sometimes minute hemorrhages.

WHAT ARE THE SYMPTOMS?

Pain.—Along the course of the nerve trunk pain is the pathognomonic symptom. It is first experienced only upon motion, particularly when the nerve is put on the stretch. As the disease continues the pain becomes more constant until finally it is continuous. There is usually a dull, heavy ache throughout the whole limb, with acute, agonizing pain which may be sharp and lancinating, or dull along the nerve trunk. Sometimes it is of a burning character, usually worse at night. After a time there is tenderness of the nerve trunk to pressure. The pain is usually most intense above the hip-joint, at the sciatic notch, about the middle of the thigh, behind the knee, below the head of the fibula, behind the external malleolus, and on the back of the foot.

Abnormal Sensations.—They may be felt over the area supplied by the affected nerve, such as numbness, tingling, formication, paresthesia and anesthesia.

Atrophy.—Atrophy of the muscles supplied by the nerve, with paresis, is present to a marked degree in severe, long-continued cases.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis rests upon the position of the pain along the nerve trunk, but the main difficulty is to decide whether the sciatica be primary or secondary. If secondary it is

usually due to disease of the hip-joint or to disease in the pelvis. These, however, will generally manifest themselves in their own peculiar way, and will usually be easily determined. In every case of sciatica great care should be used in the examination of the patient to determine the cause of the trouble.

WHAT IS THE PROGNOSIS?

This is usually favorable, but most cases run a very slow and tedious course, the duration depending largely upon the severity of the symptoms. Ordinary attacks last for a week or two to several weeks, but some cases may last for a year or two, during which time a considerable degree of wasting may take place.

WHAT IS THE TREATMENT?

GENERAL.—The patient should avoid all exposure to cold or wet, and in severe cases the limb should be kept absolutely quiet, as in other forms of neuritis. Many measures are used in the treatment of this disease, but with the exception of general rest of the parts nothing will relieve the trouble so quickly and permanently as homeopathic remedies. Even the severest forms of the disease may be speedily relieved by the properly indicated remedy.

DIETETIC.—All sweets should be prohibited in some of these cases, especially those due to rheumatic origin. This is extremely important, because many cases are made decidedly worse, attacks being even brought on by over-indulgence in sweets. With the exception of this, any nutritious food may be given.

REMEDIAL.—*Aconite.*— Inflammatory irritation of the nerve sheath, with darting, burning, benumbing pain as if the part were going to sleep, worse during night and movement, especially in early cases.

Ammonium muriaticum.—Severe and long-continued sciatica; pain in left side as if the tendons of the hip were too short: limps on walking; entirely relieved when lying down; sense of contraction of the leg.

Arnica.—From over-exertion: burning, stinging, tearing pains; numb and bruised feeling; changes position constantly as if everything on which the limb rested felt too hard.

Arsenicum.—Typical regularity of the pains: worse at night; unbearable toward midnight; burning, tearing pains with great restlessness; great weakness and prostration.

Belladonna.—Pain in the hip-joint, especially at night; sensitiveness to touch, even of the clothing; the least concussion, and even the stepping of other persons in the room aggravates; worse by the least draught of air.

Bryonia.—Pain in lumbar region extending to the thigh; worse by sitting up, by moving, and late in the evening; atrophy and emaciation of the effected limb.

Colchicum.—Sciatica of the right side; sharp, shooting pains in the sacral region extending down to the knee; must keep perfectly quiet; pain sets in suddenly, is constant and intolerable.

Eupatorium.—Severe shooting pains along the course of the left sciatic nerve, producing a palsied sensation, especially after motion.

Gnaphalium.—Intense, dull, darting, cutting, or burning pain along the nerve, with feeling of numbness, rendering exercise very fatiguing; worse from lying down, from motion and stepping; better when sitting in a chair.

Rhus toxicodendron.—Especially when caused by exposure to wet, or straining in lifting; stinging, burning, tearing pain with a sensation of coldness, numbness, formication, and paralytic stiffness of the limb, increasing during rest and when beginning to move; relieved only for a short time by motion.

Silica.—Pains shoot through the extremity at the moment when the foot is raised, as when ascending; twitching of limbs day and night; limbs go to sleep easily.

MULTIPLE NEURITIS OR POLYNEURITIS.

WHAT IS MULTIPLE NEURITIS?

It is an inflammation of many nerves, and its most characteristic features are its multiplicity, its symmetry, and its peripheral distribution.

WHAT ARE ITS CAUSES?

It is a disease of adult life, and occurs between twenty and fifty years of age. It is more common in females than

in males. Exposure to cold and insufficient nourishment may help toward the production of the disease. It undoubtedly is due to a widespread systemic poisoning. There are several forms of multiple neuritis according to their causation: toxic, due to the presence of lead, arsenic, silver, or alcohol within the blood; toxemic, due to some virus within the blood the nature of which is not wholly known, and it may primarily produce a polyneuritis such as occurs in leprous neuritis, or it produces some definite disease which is followed by multiple neuritis, such as diphtheria, small-pox, typhoid fever, tuberculosis, and syphilis: endemic, due to local organisms such as malarial neuritis and beri-beri: rheumatic, in which the multiple neuritis follows exposure to cold; cachectic, when due to some general malnutrition.

WHAT IS THE PATHOLOGICAL ANATOMY?

This corresponds to that described as occurring in simple neuritis, except that the nerve fibres suffer more than the connective tissue.

WHAT ARE THE SYMPTOMS?

According to the symptoms there may be three forms of the disease: motor, in which there is loss of power with no sensory symptoms: sensory in which there is no marked muscular weakness or inco-ordination: ataxic, in which there may or may not be any sensory symptoms, but inco-ordination predominates. The symptoms may come on suddenly or may be some little time in developing.

Motor Weakness.—It may involve either the upper or lower extremities or both, but always corresponding limbs on both sides and their extremities, the hand and foot first, and to the greatest extent. There is difficulty in balancing on account of the weakness of the feet, and also difficulty in performing the finer movements with the hands, especially in extending the wrist, so that wrist-drop is a most conspicuous symptom. There is also an inability to raise the toes from the ground in walking, owing to the weakness which produces foot-drop.

Sensory Symptoms.—Tenderness of the muscles and hyperesthesia of the skin, involving especially the soles of the feet, the palms of the hands and tips of the fingers are

common features. There is also tenderness along the nerve trunk affected. Numbness, tingling and paresthesia, and a dull, burning pain deep in the limbs are also present.

Inco-ordination.—This is present in the arms and legs, but affects the latter most frequently. It is not so severe as in locomotor ataxia, but prevents the patient from performing fine motions with his hands, and from walking as steadily as he should.

Atrophy.—This may become extreme in some cases and affects only the muscles to which the diseased nerves are distributed.

Reflex Action.—Whenever there is weakness in the legs, the knee-jerk is lost; and the superficial reflexes are usually lost where the cutaneous nerves are affected.

Electrical Irritability.—It is usually lost.

Trophic Changes.—Similar to those in simple neuritis. There is glossy skin, adhesions of the joints, thickening of the skin, nails becoming brittle and falling off, hair becoming coarse and falling out.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis depends mainly upon the combination of motor and sensory symptoms described, their symmetrical distribution in opposite limbs, particularly in their extremities, and the tenderness of the skin, nerve trunks, and muscles. Bilateral wrist-drop and foot-drop also suggest the disease.

From locomotor ataxia it may be differentiated by the presence of the foot-drop when the patient walks, and the absence of fulgurating pains.

From progressive muscular atrophy by the wasting following only the course of the nerves affected, and not extending to neighboring muscles.

From polio-myelitis anterior by the symmetrical localization of the weakness, and the presence of tenderness over the inflamed nerve trunks.

WHAT IS THE PROGNOSIS?

Very acute and severe cases may terminate fatally. The prognosis depends entirely upon the severity and suddenness

of the onset. Some cases come on gradually and last for many months or even years. When electrical irritability begins to return the prognosis is more favorable. The disease may last from three to eighteen months and gradually end in recovery. Sometimes permanent contractures occur.

WHAT IS THE TREATMENT?

GENERAL.—It is necessary as early as possible to determine the cause of the disease and remove it. Rest is of the utmost importance, and except in very mild cases the patient should be in bed. Warm fomentations may be applied over the tender nerves, and the tender limbs may be wrapped in cotton wool. Electricity should never be used during the early stages of the disease, but may be of use after the inflammatory condition has passed away and only the atrophy is left.

DIETETIC.—The diet must consist of plain, nourishing food without stimulants.

REMEDIAL.—The remedies for simple neuritis will be of use here.

BERI-BERI OR KAKKE.

WHAT IS BERI-BERI?

It is an endemic multiple neuritis which is very prevalent in China, Japan, and in the islands of the Pacific Ocean.

WHAT ARE ITS CAUSES?

It depends upon a specific organism present in the blood, which produces the disease. It is an infectious malady, but repeated opportunities of infection are necessary. Insufficient food, or food lacking in albumin, is considered a cause, and it is owing to this latter fact that in the countries where rice is a staple article of diet it is most common. It occurs very frequently in sailors to whom a variety of food has been denied. It is undoubtedly propagated in water and conveyed in that way. It affects young males between eighteen and twenty-five almost exclusively.

WHAT IS THE PATHOLOGICAL ANATOMY?

The same as in other forms of multiple neuritis.

WHAT ARE THE SYMPTOMS?

The disease comes on gradually, and may be mild or severe. The symptoms are those of multiple neuritis with the addition of edema and effusion into the serous cavities, with great liability to cardiac disturbances.

Weakness of the Legs.—It is usually the first symptom noticed, and prevents the patient from walking as much as usual.

Sensory Symptoms.—Numbness and pain come on soon after the weakness.

Palpitation.—Palpitation of the heart, with shortness of breath, epigastric oppression and loss of appetite, soon follows.

Pulse.—The pulse may be irregular and dicrotic.

Edema.—There is edema of the extremities, and also in severe cases of the whole body, which may be very great in degree, preventing the patient from lying down, and causing rupture of the skin.

There are three forms of severe cases: the atrophic or dry, in which the symptoms come on slowly, but soon increase with great rapidity until there is paralysis of all the muscles of the body, with excessive muscular wasting, pain, and paresthesia; the hydropic or wet, in which the body is enormously swollen, with subcutaneous effusion, as well as effusion into the serous cavities; the pernicious, which is a combination of the other forms but occurs with extreme intensity.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis can only be of difficulty in the very beginning of the disease, or if the patient is not known to have been exposed to infection. Later in the disease the symptoms are so well marked that a mistake cannot readily be made.

WHAT IS THE PROGNOSIS?

This varies according to the severity of the disease. Mild cases usually recover. Severe cases die within two weeks of the onset of the disease.

WHAT IS THE TREATMENT?

GENERAL.—Remove the patient from the infected district as early as possible and he will then usually begin to recover.

DIETETIC.—Change of diet is of the utmost importance. A greater variety of food containing plenty of albumin is absolutely necessary.

REMEDIAL.—*Apocynum*, *Arsenicum*, *China*, *Digitalis*, and *Strophantus*, are the remedies that will be most frequently called for.

LEPROUS NEURITIS, OR LEPROSY.

WHAT IS LEPROSY?

It is a chronic infectious disease of parasitic origin, characterized by the production of new cell formations in the cutaneous surfaces, and in the connective tissues of the nerves.

WHAT ARE ITS CAUSES?

It depends upon the presence of living organisms within the blood. It may occur at any time of life, but most frequently during adult life. There are two varieties, called the tubercular or tegumentary; and the anesthetic or trophoneurotic type. Climate, race, soil, food, bad hygiene, and malaria are causative factors. It is prevalent in India, China, Turkey, the West Indies, Portugal, Russia, and the Hawaiian Islands. Sporadic cases occur in some parts of the United States: in Minnesota, Iowa, Wisconsin, and along the Pacific Coast.

WHAT IS THE PATHOLOGICAL ANATOMY?

The bacilli lepræ are found in diffuse and nodular infiltrations on the skin and mucous membranes, and in the interstitial connective tissue of the peripheral nerves. The appearance of a lepra tubercle is much like that of tuberculosis. The bloodvessels are dilated and their walls are thickened. The nerve sheaths are increased in thickness, and the nerve fibres themselves undergo slow wasting.

WHAT ARE THE SYMPTOMS OF TUBERCULAR LEPROSY?

Prodromal.—There may be a condition of general debility which manifests itself by weariness after slight exertion,

with a sense of depression, heaviness and tendency to sleep. Febrile symptoms, preceded by sensations of chilliness or even rigor, are quite common in this stage. The fever is usually of an intermittent type. These prodromal symptoms may extend over a period of many months and are followed by the so-called eruptive stage.

Cutaneous Manifestations.—There is first a heightening of the color of the skin over the malar prominences, and it has a shiny, glossy appearance. There is moreover an increase of the secretions of the skin, it has a greasy feel and appearance, and sweat is abundant. Sometimes there may be suppression of secretions, with falling of the hair, especially of eyebrows and lashes. The redness of the skin may appear and disappear several times before tuberculosis is established. The eruptions are most common on the face, hands and feet, but may appear on any portion of the trunk or limbs. The macules are oval in shape, of a reddish-brown or coppery color, fading into a dirty yellow. The skin of the face is more or less thickened and swollen, and presents an edematous appearance. The tubercles may be about the size of a pea or of a walnut, and several of them may coalesce and form elevated patches of considerable size. The regions most affected are the forehead, nose, lips, chin, ears, hands and forearms, especially the extensor surfaces.

Subjective Sensations.—Itching and burning of the skin, rheumatic pains, cramps in the lower limbs, various forms of neuralgia with a sensation of numbness or deadness, especially in the lower limbs.

Mucous Surfaces.—These are also the seat of infiltrations which break down and form ulcers, causing more or less destruction of tissue. The nose, mouth and pharynx are most commonly involved. One of the early symptoms of leprosy is a snuffling nasal respiration, due to ulceration of the nasal septum.

WHAT ARE THE SYMPTOMS OF ANESTHETIC LEPROSY?

The symptoms of this form are more of a neurotic character. There is extreme itching and burning of the skin, hyperesthesia and pains of a lancinating, boring character in the deeper structures, followed by sensations of numbness or deadness, and later by complete anesthesia in spots,

especially in the regions supplied by the ulnar and peroneal nerves.

Cutaneous Changes.—Dryness and scaliness of the skin with formation of pemphigoid blebs upon the fingers and toes, which rupture after a few hours, leaving excoriations like superficial burns, which upon healing leave pigmented stains. The eruption may develop upon one portion of the body after another, and after a time become a reddish-brown color in patches. The centre of the patch becomes thin, white, atrophic and wrinkled, and there is usually a denudation of the hair. The blanching may affect a large portion of the body. These atrophic patches are usually without sensation.

Figure 35.
Anesthetic leprosy.

Paralysis.—Paralysis of certain nerves, with atrophy of the muscular tissue which they supply, is common in this disease.

Mutilation of the Hands and Feet.—The bones of the fingers and toes may be lost without ulceration, by a process called osseous absorption. It affects one finger after another, the tissues retracting and the nail retreating, until it caps the first phalanx: or the fingers and toes may become the seat of gangrene, and spontaneous amputation may result without pain.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis depends upon the presence of irregular anesthetic patches associated with pigmentation and pallor of the skin, with muscular atrophy, in a person who has

been exposed to the infection of leprosy. The infiltration of the skin of the face, producing the characteristic leonine expression, is pathognomonic of the disease.

WHAT IS THE PROGNOSIS?

Grave in all cases. It is usually from three to twenty years in developing, and when it manifests itself upon the cutaneous surfaces it is usually after considerable nerve degeneration has taken place.

WHAT IS THE TREATMENT?

The same as in other forms of multiple neuritis.

PART VIII.

FUNCTIONAL NERVOUS DISEASES.

HYSTERIA.

WHAT IS HYSTERIA?

It is a condition characterized by mental and physical ailments which may simulate almost any other disease of the mind or body.

WHAT ARE ITS CAUSES?

It occurs most frequently in women, about twenty times as frequently as in men, and most frequently in the second decade of life, the larger number of cases beginning between fifteen and twenty years of age. An hereditary tendency to neurotic conditions is present in most cases. Physical or mental influences directly determine the development of the disease in the individual. A sudden fright, deep emotion, unhappy love affair, and sometimes great intellectual exertion may produce it. Disorders of the generative organs, either functional or organic, sexual excesses, masturbation, secondary stages of syphilis, and tuberculosis are sometimes causes. Organic diseases of the nervous system may sometimes develop true hysteria.

WHAT IS THE PATHOLOGICAL ANATOMY?

The pathological changes in this disease are negative except when it is complicated with some organic nervous trouble.

WHAT ARE THE SYMPTOMS?

These may be divided into two classes: continuous and paroxysmal.

CONTINUOUS SYMPTOMS.—*Mental Symptoms.*—These are the most prominent: defective will power, loss of self-control, inability to resist inclinations, irritable temper, physical and mental depression. Self-consciousness controls the patient's thoughts and actions. The patient is gratified by sympathy, which when given helps on the trouble. Excessive emotion, which is manifested in laughter and tears alternating with each other, on the most trivial occasion occurs in mild cases.

Globus Hystericus.—A sensation of something suddenly closing the throat, or of a ball rising from the stomach to the throat is a very common symptom.

Hyperesthesia.—There is hyperesthesia in various parts of the body, called hysterogenic spots. They may be up and down the spine or in the ovarian region. When these spots are irritated by pressure, a true hysterical spasm may be induced. They may also be on the breasts or on the trunk, and sometimes on the vertex.

Anesthesia.—This may occur in various forms; the most common are hemianesthesia, anesthetic spots, and anesthesia of one limb. Hemianesthesia may be extremely profound from the top of the head to the sole of the foot, associated with vaso-motor changes; the prick of a pin will not cause bleeding. It may change from one side to the other.

Hyperalgesia—Is present as a severe neuralgic pain in the breast, or as mastodynia.

Amaurosis.—This is rather a rare condition, coming on suddenly and disappearing suddenly. It may, however, last for some time; months or even years.

Amblyopia, Achromatopsia, Dyschromatopsia.—Any one of them may be present.

Deafness.—It is common and may come on suddenly and be complete. It may occur with hemianesthesia or alone.

Anosmia.—Presents in most cases of hemianesthesia.

Paralysis.—It may exist in various forms. There may be a monoplegia, paraplegia or hemiplegia, or it may be confined to a few muscles, such as are affected in some forms of facial paralysis. Paralysis of the muscles of the tongue, larynx, pharynx, and esophagus may also be present. There may be only a slight loss of power or complete palsy. It may come on suddenly or develop gradually and gradually

grow worse. It may last for only a short time or even for several years. The paralyzed parts become blue and mottled, and there may be hyperesthesia of the paralyzed limb.

Contractures.—They are present in some cases, and they may come on suddenly or gradually; most often suddenly. They do not relax during sleep, but may relax under chloroform. There may also be present anesthetic or hyperesthetic areas in the contracted limb.

Tremor.—It is a most important symptom, and is generally caused by trauma or some toxic agent, such as alcohol or lead. It may be persistent and last for a year or two. The oscillations may be rapid, medium or slow.

Inco-ordination.—Hysterical ataxia has been known as astasia-abasia, which is a loss of the power of standing and of walking. When the patient is lying or sitting there is full muscular power in the legs, or he may be able to walk upon all fours, but is not able to walk properly.

Atrophy.—It may occur in limbs which are paralyzed and contracted or anesthetic.

Vomiting—Is sometimes extremely frequent, but is usually dominated by the mental state.

Anorexia.—Anorexia may persist for months, giving us examples of the so-called "fasting girls."

Heart.—The action of the heart may be slow, quick, or irregular, unassociated with dyspnea.

Fever.—It may be continuous, intermittent, or remittent.

Cough.—It may be a persistent and troublesome symptom.

Aphonia.—Aphonia may be present and last for years, the voice being completely lost.

Phantom Tumors.—Phantom tumors of the abdomen occasionally occur. They may simulate pregnancy, and even be accompanied by enlargement of the breasts. They may appear with great rapidity and disappear gradually.

Anurea.—This occasionally occurs but is not a common symptom.

Paroxsmal Symptoms.—*Convulsions.*—These are usually preceded by a period of unrest and ill-feeling, with mental symptoms similar to those described above, for some time before the attack. Tears and laughter are excited readily,

hallucinations and delusions are common, or there may be disorders of the digestive apparatus. The fit is ushered in by an aura, such as globus hystericus, or circumscribed pain in the head of very limited extent, with a feeling as if a nail were driven into the head: or there may be extreme sensitiveness of one of the hysterogenic spots. The convulsion may be divided into four periods:

The epileptoid, in which the arms and legs are usually extended, hands clinched, trunk bent, usually opisthotonos, eyes crossed, and the teeth set; breath is arrested, pulse accelerated, and consciousness lost. When falling, the patient does not usually hurt herself and the tongue is not bitten. Tonic spasm is followed by a clonic stage which lasts for a little while and then gradually subsides.

The period of grand movements or clownism, which is characterized by violent and extravagant muscular movements. The patient's body may assume the so-called arc of a circle, which is a position of complete opisthotonos; or there may be rapid flexing and extending of the limbs in various ways. There is not usually loss of consciousness during this second period.

The passionate period is a period of passionate attitudes which are simply expressions of the mental condition. There may be different attitudes assumed, such as that of defense, menace, appeal, ecstasy, scorn or lamentation.

The period of delirum, which is really a period of emotional disturbance, continuous with that of the first period but not characterized by active motions. The delirium may merge into obstinate silence. This hysterical convulsion may last from the first to the fourth stage, from one-quarter to one-half an hour, but the fourth stage may last for a day or two. The attack may pass off with a profuse flow of limpid urine, and the patient after a time be herself again.

Lacunæ of memory or periods of loss of memory are frequent after the paroxysms, or may take the place of the real hysterical convulsion. During these lapses of memory, the patient is not able to realize her surroundings or just who she is, and after the lapses are over she cannot recall what has occurred during these times.

Somnambulism.—This is a common symptom in hysterical subjects.

Catalepsy.—This is a condition of both mental and motor inertia. The thoughts as well as the limbs remain in any position in which they are placed. The limbs may remain rigid for a long period of time.

Lethargy.—The patient lies in a sort of stupor with closed eyes. There may be fibrillary tremors of the eyelids. This condition may last for hours.

Trance.—A higher degree of lethargy. It is a condition of suspended cerebration, and may last for days.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis depends mainly upon the changeability of the symptoms, and, as the definition implies, it is the fact of simulation of so many diseases that enables us to make the diagnosis. The main point to be determined is the presence or absence of organic changes, and when that has been determined the diagnosis is easy. The symptoms of hysteria coming on after great mental excitement would lead us to a diagnosis.

WHAT IS THE PROGNOSIS?

The prognosis as to recovery depends upon the cause and surroundings of the individual in each particular case. Some cases get well very soon, and others may persist for years, there being absolutely no way to determine the outcome. It does not usually produce death, but organic changes may supervene as the result of hysteria and prove fatal.

WHAT IS THE TREATMENT?

GENERAL.—Avoid those causes which may produce the disease, such as great mental emotion, late hours, or anything which is likely to draw heavily upon the vital forces; but more than all, a firm yet kind and sympathetic hand should control the case. In no disease is firmness on the part of the physician and persons who come in contact with the patient so necessary as in this. Extremely severe measures have to be used sometimes, such as confining the patient to her room, or even threatening with severe meas-

ures, such as cauterization of the back. The object of these measures is to stimulate the patient to such a degree that she will endeavor to get control of herself and prevent the disease from manifesting itself. During the convulsion simply watch the patient to see that she does not harm anyone. Have the clothing loosened so that there may be free action of the respiratory muscles.

REMEDIAL.—*Agnus castus.*—Hysteria with maniacal lasciviousness; despairing sadness; peevish; inclined to be angry; nervous weakness; lethargy; frenzy; sleeplessness, or starting up frightened in her sleep.

Anacardium.—Restlessness; must be in constant motion; feels as if she had two wills, one commanding to do what the other forbids; great forgetfulness; constant desire to urinate; urine clear as water.

Asafetida.—Where the hysteria is the direct result of the checking of habitual discharges; globus hystericus; flatus accumulates in abdomen, and pressing up against the lungs causes oppression of the chest; sensation as if a ball started in the stomach and came up into the throat.

Cactus.—Sadness, crying without reason, consolation aggravates, love of solitude, fear of death, whole body feels as if caged in wires.

Gelsemium.—Hysterical convulsions with spasms of the glottis; hysterical epilepsy; excessive irritability of mind and body with vascular excitement; semi-stupor with languor and prostration.

Ignatia.—Perversion of the co-ordination of functions; disposition to grieve, to brood in melancholic sadness over real or imaginary sorrows; mental symptoms change often; cheerfulness, then great despondency.

Moschus.—Hysterical paroxysms with insensibility; cries one moment and bursts into uncontrollable laughter the next; palpitation of the heart; tremulousness; fainting spells, especially as soon as the eyes are closed, with pale face and coldness; rush of blood to head, with staring eyes; suffocative constriction in chest; copious, pale urination.

Platina.—Demonstrative self-exaltation and contempt for others; cramp pain in the forehead as if between screws; great alternation of sadness and cheerfulness; apprehension of death with disposition to weep.

Pulsatilla.—Constriction in throat; feels something in throat impeding speech, especially at night in bed; constant change in her feelings and in her symptoms; profuse, watery urine; tendency to weep; craves sympathy.

EPILEPSY.

WHAT IS EPILEPSY?

It is a disease characterized by convulsions or sudden loss, or impairment, of consciousness, in which the convulsions are not due to organic disease or reflex irritation, or abnormal blood states.

Figure 36.

Facial expression in case of epilepsy of six years' duration. Age, twenty-one years.

WHAT ARE THE CAUSES OF EPILEPSY?

After one attack has occurred without any discoverable cause other attacks come on very readily. Females suffer more frequently than males. An inherited tendency is sometimes found. Defective development of the brain may produce a tendency toward the disease. Three-fourths of the cases begin under twenty years of age, and about one-half of them between the ages of ten and twenty. After thirty years of age males suffer more frequently than females. As to immediate causes, great mental emotion, fright, excitement and anxiety are potent causes. Blows or falls upon the head, exposure to the heat of the sun, acute specific diseases, intestinal worms, masturbation, delayed menstruation at the time of puberty, and syphilis may all produce the disease.

WHAT IS THE PATHOLOGICAL ANATOMY?

In mild cases there is nothing to be seen upon a careful examination. In chronic cases there may be some thickening and opacity of the membranes of the brain, or there may be signs of meningitis. Where the patient has died during a fit there is generally intense venous congestion, but there are no organic changes.

WHAT ARE THE SYMPTOMS?

There are two classes of symptoms: major or severe, and minor or slight; or, as they are called by some authors, grand mal and petit mal.

Aura.—They are usually present just before the attack comes on. They may consist of numbness in one of the fingers or in one part of the body. General tremor or shivering is an occasional warning, or there may be sensations of pain in the epigastrium associated with nausea, or sensations of giddiness, also associated with nausea. Fear, or a vague, dreamy state, indescribable smells, sour or bitter taste, sudden loss of hearing, sudden loss of sight, or the appearance of objects before the eyes are various forms of aura which are sometimes present.

Figure 37.
Same patient four years before.

Epileptic Cry.—The so-called epileptic cry sometimes occurs at the onset of the attack. It is a weird kind of scream which is a sort of prolonged groan, not very loud, but unmistakable when it has been heard once. Some patients may commence running just before an attack, or will suddenly turn around when walking and go the other way. Palpitation and pain in the region of the heart, or sudden dyspnea may occur in others.

Grand Mal.—At the onset of the convulsion tonic spasm commences, with rigid, violent muscular contractions of the limbs, the face is distorted, and while the color may be unchanged at first, it quickly becomes pale, then flushed, and later livid, as the movements of respiration are interfered with. The eyes may be opened or closed, and the pupils dilated, as the cyanosis comes on. After a moment or two the spasms become clonic in character, and the limbs jerk violently. After the spasm is at an end, the patient lies unconscious and sleeps heavily for a time, and later can be aroused. The convulsions may begin in one part of the body, as the face or arm, and then spread to the other parts on the same side, finally involving the whole body. The patient froths at the mouth and bites the tongue. Urine and feces are occasionally passed during the convulsion. The attacks of grand mal are sometimes followed by vomiting.

Petit Mal.—The patient suddenly stops his occupation, stares vacantly about him for a moment, and then goes on with his work as if nothing had happened. He does not fall; has no convulsions; simply a loss of consciousness for a few seconds.

Dual Consciousness.—It may occur in cases suffering from grand mal or petit mal. It is a condition in which a patient seems to pass from one existence into another. When in the abnormal condition he knows nothing of his previous normal condition; and when in the normal condition knows nothing of the abnormal condition. These periods of abnormal states may last for hours, days, or weeks.

Hystero-Epilepsy.—This is a combination of hysteria and epilepsy, but it is a very rare condition. It usually commences with a true tonic convulsion, followed by clownishness instead of the sleep which is natural in epilepsy.

Frequency of Attacks.—In the beginning the patient may have severe attacks only once a year, and then in six months, gradually increasing in frequency until they may come every day or two, or three times a day.

Condition of Patient between the Attacks.—He may feel perfectly well after an attack and until within a few days of another attack, when symptoms of approaching illness

may manifest themselves, to be dispelled again by the attack.

Mental State.—In severe cases there is a gradual deterioration of the mental faculties, slight in some, but grave in others. Loss of memory, irritablility, inability to concentrate the mind, fits of anger amounting, sometimes almost to true mania, and loss of moral sense take place in these cases.

Physical Conditions.—The patient is usually physically deteriorated, unable to undergo much exertion, feels more inclined to keep quiet and do nothing, becomes pale and anemic, and loses appetite and flesh.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The aura, the epileptic cry, sudden loss of consciousness, tonic convulsions, biting of the tongue and evacuation of the bladder make the diagnosis easy.

In hysteria there is usually some emotional cause for the convulsion, and there is never evacuation of the bladder, and no biting of the tongue.

WHAT IS THE PROGNOSIS?

The disease undoubtedly shortens life to some extent, but patients may live to the age of forty or fifty. About ten per cent. suffer from dementia in the terminal stage. Attacks of grand mal may occur so quickly, one after another, that the patient passes into a condition called "status epilepticus" which usually ends fatally. Epileptics very frequently suffer from phthisis. The prognosis is better if all the fits occur in the night, nocturnal epilepsy; or if all of them occur during the day; but at the best it is a disease which is difficult to cure.

WHAT IS THE TREATMENT?

General.—Any of the causes which may have produced the disease, such as masturbation, great mental emotion, excitement, or anxiety, should be removed as far as possible. Moderate exercise, both of mind and body, is good. The patient should have plenty of sleep and avoid what may be a source of drain upon the vital forces.

SURGICAL.—If the seizures always commence in one extremity, passing to the others later, the epilepsy may be due to irritation over the motor area of the brain which controls the part in which the spasm first commences. Under such conditions surgical measures may be used to advantage, such as trephining and removing some irritating growth or depressed bone, or trephining alone may relieve.

DIETETIC.—These patients should be well fed, but must eat only digestible food and have their meals at regular intervals, the principal one in the middle of the day. Children will sometimes improve rapidly upon only a vegetable diet, or a diet of which milk, bread and butter, rice pudding, corn starch, and Indian meal pudding form the basis.

REMEDIAL.—*Amyl nitrite.*—Muscular twitching in legs, arms and face, followed by unconsciousness; haunted many times a day by an indescribable dread and sensation of the coming fit: profound and repeated yawning during unconsciousness: succession of fits with increasing frequency: before one fit ceases another begins.

Artemisia vulgaris.—Vexed, irritable, depressed during the day before a fit at night; fits brought on by violent emotions, especially by fright: paroxysm usually followed by sleep; mental powers gradually become extinct: petit mal: patient is unconscious only for a few seconds or minutes, and then continues his occupation unconscious of anything unusual having happened.

Belladonna.—Fresh cases of epilepsy, with decided brain symptoms; aura as if a mouse were running over an extremity, or illusions of sight or hearing; convulsion comes in upper extremities and extends to the mouth, face, and eyes.

Bufo.—Aura starts from sexual organs or from solar plexus; epilepsy from onanism; longs for solitude to give himself up to his vice; epileptic aura from uterus to stomach.

Calcarea carbonica.—Epilepsy at the age of puberty or from irregular menstruation: irritation of the sexual organs; child masturbates; enfeebled memory; stupid; peevish in the intervals between the attacks.

Cuprum metallicum.—Nocturnal epilepsy, or when fits return at regular intervals, beginning with sudden scream:

convulsions commencing in the fingers or toes, or in the arms, with coldness of the hands and feet; clinching the thumbs; suffocative paroxysms; frequent emissions of urine: during dentition or from a retrocession of exanthema.

Hydrocyanic acid.—Recent cases; sudden complete loss of consciousness; extreme coma for several hours; jaws clinched; teeth firmly set; frothing at the mouth; inability to swallow; hands clinched; stiffness of legs.

Nux vomica.—Epilepsy from indigestion; aura starts in epigastrium and spreads upwards; sensation of ants crawling over the face; involuntary evacuation of bladder and bowels.

Silica.—Nocturnal epilepsy; spasm occurring about the time of the new moon; feeling of coldness before the attack; violent screaming and groaning; warm perspiration and sleep after the spasm.

Stramonium.—Jerking the head continually to the right; continued rotary motion of the left arm; deep, snoring sleep; pale face; afraid of being alone; convulsions affecting the upper extremities more than the lower; sudden loss of consciousness while reading.

CHOREA, ST. VITUS' DANCE.

WHAT IS CHOREA?

It is a condition characterized by irregular jerking and inco-ordinate movements.

HOW MANY KINDS OF CHOREA ARE THERE?

Five. Common chorea or Sydenham's chorea; hereditary chorea or Huntington's chorea; habit chorea; saltatoric spasm; and electrical chorea.

WHAT ARE THE CAUSES OF COMMON CHOREA?

It occurs most frequently between the ages of five and fifteen, but also occurs in adult life. It may occur in all climates and in all seasons of the year. Fright or some great emotional disturbance, mental worry, acute rheumatism, infectious fevers, over-study, intestinal irritations, such as worms, anemia and mal-nutrition may be exciting and predisposing causes of the disease.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is usually an intense hyperemia, with dilatation of the vessels, minute hemorrhages and softened areas within the cortex of the brain. In long-continued cases there are fibrinous deposits in the walls of the heart. The pyramidal tracts, the lenticular nuclei, and even the spinal cord may take on changes which occur in the cortex.

WHAT ARE THE SYMPTOMS?

It may begin suddenly or come on gradually.

Choreic Movements.—There is first an irregular twitching of the hand or face on one side. There are also winking of the eye and twitching of the mouth. The child drops things from the hands. A little later the child stumbles in walking. In three or four weeks the disease may reach its height, and irregular movements of the hands and feet, or even of all the upper and lower extremities, and perhaps the trunk itself, may be involved in continuous and irregular movements. Walking is extremely awkward and difficult. The patient cannot dress himself on account of inability to control the movements of the hands. Speech becomes indistinct because of the inco-ordination of the muscles of the tongue. Respiration also becomes irregular. These choreic movements occur both when the muscles are at rest and during action, but cease when the patient is asleep. The child is usually worse in the morning, but improves as the day advances. Excitement and physical exertion aggravate the movements.

Muscular Weakness.—The limbs become weak in a little while, but are never completely paralyzed. Nocturnal enuresis is usually present.

Reflex Action.—It is diminished, and in many cases lost.

Electrical Irritability.—Electrical irritability of the muscles is increased in most cases.

Mental Conditions.—The mind is usually dulled, the patient becomes irritable, peevish, and hard to please or manage. Excessive mental excitement and even delirium may occur in severe cases, and sometimes constitutes the so-called maniacal chorea, or chorea insaniens.

General Symptoms.—Loss of appetite, constipation, loss of flesh, and general anemia take place after the disease has continued for a little while.

WHAT IS KNOWN OF HEREDITARY CHOREA?

It occurs between thirty and fifty years of age without any known cause, beginning first with twitchings in the face which gradually extend to the arms and legs. There is a slowly progressing mental deterioration with melancholia, and finally dementia. It is slow in its progress, lasting ten or twenty years. It is usually directly hereditary from either father or mother, more often the latter.

WHAT IS HABIT CHOREA?

It is some special movement or habit which a patient has acquired, perhaps early in life, and which continues through life. It may be only a shrug of the shoulder or a sniff, or the twitching of the eyes, or a sudden bending back of the body. It never becomes excessive and causes but little trouble.

WHAT IS SALTATORIC SPASM?

It is a condition in which there is rapid and violent contractions of the flexors and extensors, or the muscles of the entire leg when the patient attempts to stand, so that there is a jumping and springing upward. It only occurs when the patient attempts to stand, and the patient may be thrown to the floor. The disease may last only for a short time or for many years. It has sometimes occurred in epidemics in Russia, Canada and Java, and has been called the "jumping sickness."

WHAT IS ELECTRICAL CHOREA?

It is a disease in which muscular movements are sudden, like a shock produced by a sudden current of electricity, and have the peculiar shock-like contractions so produced. These movements usually commence in the arm, spread to the leg on the same side, and later involve the other side of the body. They gradually increase in severity, and after a time the limbs become weak and atrophied, until finally paralysis of the whole body comes on. The patient usually dies from the paralysis.

WHAT IS THE DIFFERENTIAL DIAGNOSIS OF CHOREA?

The disease is easily diagnosed by the irregular twitching movements which occur while the muscles are at rest or

in action, but cease when the patient is asleep. There is usually no difficulty in recognizing the disease.

WHAT IS THE PROGNOSIS?

Attacks may come on and last for a few weeks or for several months. Most cases get well after a while, but there may be frequently recurring attacks which gradually diminish in frequency and severity. In adults it sometimes lasts many years. It does not seem to shorten life to any extent unless the irregular movements become so severe that the patient is unable to eat properly or to rest, which causes extreme inanition.

WHAT IS THE TREATMENT?

GENERAL.—Mental and physical exertion and emotional excitement should be avoided as far as possible. These are most important conditions to observe, because any of them may produce and keep up the disease if allowed to continue. If the disease be severe the patient should be kept in bed for a long time, as complete rest is absolutely necessary. The mental condition of the patient should be carefully looked after, for the depression is sometimes so great that the beneficial effect of rest in bed is neutralized by it; and therefore a certain amount of mental diversion should be indulged in, such as being read to or listening to music. Care should be taken that the patient does not harm himself by hitting his hands or any part of the body against hard substances, or by falling against anything. It may be necessary sometimes to keep the patient in a padded room. Severe cases should never be allowed to feed themselves, because they may do great harm with the fork or spoon with which they break their food.

DIETETIC.—Plain, nutritious food and in sufficient quantity should be given.

REMEDIAL.—*Agaricus.*—Twitching and spasm of the eye-balls and eyelids; spasmodic motions, as jerks of single muscles or of an upright and lower left extremity; jerking of head and neck, with difficulty of swallowing; weakness and coldness of limbs and unsteady walk.

Belladonna.—Reflex chorea from dentition or pregnancy; motions of body are generally backwards, or to and fro, bor-

ing the head in the pillow; grinding of teeth; after fright or mental excitement; dull, heavy, drowsy and stupid.

Calcarea carbonica.—Fright followed by trembling motions of upper and lower limbs; patient low-spirited and peevish; cannot speak as he bites his tongue when trying to speak; great weakness.

Crotalus.—Chorea, especially when it can be traced to a septic or toxemic cause, or when occurring in rheumatic or albuminuric subjects; starting, jerking, trembling and unsteadiness of the limbs; irritable, cross, infuriated by least annoyance.

Ignatia.—Emotional chorea, especially from grief or fright, with sighing and sobbing; vacillating gait; stumbles and falls over small objects.

Laurocerasus.—Emotional chorea after fright; fearful contortions and jactitations when awake; restless sleep; violent and destructive motions; can neither sit, stand, nor lie down on account of incessant motions; speech indistinct; tears clothing; strikes at everything.

Mygale.—Arms and legs in constant motion; unable to dress without assistance; mouth and eyes open and shut in rapid succession; when attempting to control the movements he loses his breath.

Natrum muriaticum.—Chronic cases when due to fright or suppression of eruptions on the face; paroxysms of jumping high up, or mere jerking of the right side and head.

Stramonium.—Saltatoric spasm; movements characterized by great violence, affecting the whole body and producing the most grotesque leaps; motions and gestures, rotates the arms and clasps the hands over the head; full of fear.

Tarentula.—Nocturnal chorea; movements do not cease even at night; continual motion and trembling of the whole body; cannot speak, swallow, sit, stand or walk; fear of impending death; is made better by diversion and by music.

PARALYSIS AGITANS—SHAKING PALSY.

WHAT IS PARALYSIS AGITANS?

It is a condition characterized by muscular weakness and tremor.

WHAT ARE ITS CAUSES?

It is more frequent in men than in women. It generally commences after forty years of age, and most of the cases begin between fifty and sixty. The most frequent causes are great mental emotions, physical injury, acute diseases, prolonged anxiety and sudden alarm. Physical injury with sudden alarm is the most frequent cause.

WHAT IS THE PATHOLOGICAL ANATOMY?

Enlargement of the nerve cells within the pons, thickening of the bloodvessels, induration of the pons, medulla and cord, and increase of the connective tissue in the motor tracts and nerves.

WHAT ARE THE SYMPTOMS?

Tremor.—Usually commences in the hands, sometimes in the forefinger and thumb, or it may be in the arm or shoulder. It slowly spreads from the part in which it commences to neighboring parts until one side of the body is affected, and then it spreads to the other side. It is an alternating contraction of opposing muscles, causing rythmical motions of the parts to which they are attached. The muscles of the trunk, especially those of the back, are sometimes involved, but the head usually escapes. The tremor is the same whether the muscles are at rest or not. It may sometimes be controlled slightly by the voluntary action of the patient. The muscles of the tongue are sometimes affected and the jaws later.

Muscular Weakness and Rigidity.—They come on together and are as much symptoms of the disease as tremor. The loss of power at first is only slight, but gradually increases until the patient is unable to grasp anything in his hand or to rise from his seat, though paralysis is never complete. When the patient attempts to make a motion of any kind, it is not only weak but it is slow, and it takes some time to perform even simple movements. The limbs gradually become rigid and assume certain positions, the fingers are slightly flexed at all the joints, the arms are flexed at the elbows, the body is bent forward, and there is slight flexion of the hip and knee-joints. The head is usually carried forward in the same direction as the body.

Gait.—The patient when starting to walk stands first with his body bent forward, his knees flexed; then puts one foot a little in advance of the other and steps forward slowly at first, but after a few steps goes faster and then faster until there is a decided running gait manifested, which is called festination. There is also a tendency for the body to fall forward when walking, which is called propulsion. The patient seems to be running in order to keep his centre of gravity. He cannot step suddenly, for if he did he would be likely to fall. The tendency to walk rapidly backward is called retropulsion.

Figure 38.
Attitude in paralysis agitans.

Myotatic Irritability.— This is usually normal. The knee-jerk is also normal in the majority of cases, but sometimes is increased.

Sensory Symptoms.— Sensibility of the skin is not affected, but there may be an aching of a rheumatic character in the limbs in those muscles which afterwards are affected by the tremor, particularly in the early stages. Great sense of fatigue is often felt, and also a great sense of heat, which may be in the interior of the body or confined to one limb.

Mental Condition.—The mind is usually clear, but the patient is irritable on account of the physical restlessness and the mental depression.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The tremor, with the muscular weakness and rigidity, usually makes the diagnosis easy.

It may be differentiated from insular sclerosis by the fact of the tremor being present even while the patient is at rest; while in insular sclerosis it occurs only when the

patient attempts to make voluntary movement, and it is more jerky and irregular in character.

WHAT IS THE PROGNOSIS?

It is usually unfavorable as far as recovery is concerned. The patient may be made better, or the progress of the disease may be checked for a long time. There is usually no danger of life and the disease may last for many years.

WHAT IS THE TREATMENT?

GENERAL.—Freedom from mental or physical exhaustion should be had as far as possible. A quiet, regular life and freedom from care are necessary.

REMEDIAL.—*Baryta carbonica.*—Nervousness: excessive irritation of all the nerves: twitches and jerks of the body during the day; general weakness of nerves of the body; increased weakness: can scarcely put out his arm: general paresis and palsy of all the muscles of the body.

Gelsemium.—Weakness and trembling through the whole system: complete relaxation and prostration of the whole muscular system: numbness and coldness of the extremities: becomes easily tired and exhausted: neuralgic pains along the tracts of the nerves: great distress and apprehension: jactitation of muscles; tremulous, with profuse urination.

Hyoscyamus.—Not a single part of the whole body nor a solitary muscle in a quiet state for a moment: trembling of the limbs with weakness; staggering gait: painful numbness of the hands.

Mercurius.—Trembling of the hands and tongue; tremor of hands so that he cannot lift anything, eat nor write: marked tremor of neck and lower extremities: great weakness and trembling from least exertion: limbs stiff, cannot be easily moved: weariness, especially while sitting, as if all of his limbs would fall from him: depression of spirits.

Phosphorus.—Trembling, especially of hands while writing; trembling all over the body or in single limbs with nervous debility: motions involuntary and uncertain: general relaxation of muscular power: great languor and disinclination to move: inexpressible heaviness of the whole body.

Plumbum.—General debility; restlessness and uneasiness; tremor with neuralgic pains in the trunk and limbs; rapid walk, bending forward; weakness of all the limbs, with trembling or numbness.

Tarentula.—Trembling of the body and limbs; great restlessness and agitation; has to change position frequently; constantly moving hands, feet and head; least excitement irritates; paralysis caused by great mental distress, with pain and continual itching in the arms and trembling in the legs.

TETANUS.

WHAT IS TETANUS?

It is an infectious disease of the nervous system characterized by tonic spasm of the muscles with marked exacerbations.

WHAT ARE ITS CAUSES?

Trauma, which produces the so-called traumatic tetanus; exposure to cold, producing idiopathic or rheumatic tetanus; when occurring in newly-born children it is termed tetanus neonatorum; and when occurring after childbirth or abortion it is called puerperal tetanus. Tetanus may occur at any time during life and is more common in dark-skinned races and in temperate regions. The immediate cause of the disease is a specific bacillus which produces a toxic material within the system that induces the disease.

WHAT IS THE PATHOLOGICAL ANATOMY?

The lungs are usually found congested, and there may be a hypostatic pneumonia or emphysema. The muscles contain small extravasations of blood. There may also be rupture of individual fibres. In traumatic cases the wound may be in a healthy or unhealthy state, or it may have perfectly healed. In the brain and spinal cord there is distension of vessels and minute hemorrhages, undoubtedly due to the severity of the convulsions.

WHAT ARE THE SYMPTOMS?

Prodromal.—In traumatic tetanus in from five to fourteen days after the infliction of the wound, but in severe

cases from twelve to forty-eight hours, there are noticed vague pains in the head and epigastrium, with a general feeling of unrest and depression. Sensations of numbness, paresthesia, or pain at the seat of the wound will first be noticed.

Trismus or Lock-jaw.—After the prodromal stage has lasted for a short time a slight stiffness of the jaws is noticed, with some difficulty in swallowing and stiffness of the tongue, gradually increasing until the patient is unable to separate his jaws.

Rigidity of the Neck.—This may come on with the trismus or precede it. It is noticed first as a slight stiffness which the patient thinks is due to having sat in a draught, and in the idiopathic cases may be due to this cause. The head is slightly bent backward. The stiffness passes down the spinal muscles, involving later the muscles of the lower extremities. The rigidity is usually tonic, and continues with greater or less severity, with exacerbations.

Opisthotonos.—Opisthotonos is produced by the severity of the muscular contraction of the back, and the patient may rest only on his head and heels.

Pain.—At first the stiffness and rigidity are unaccompanied by pain, but as the spasms increase in frequency and severity a cramp-like pain comes on which is intense, causing the most agonizing suffering.

Spasm of Respiratory Muscles.—Fixation of the chest is present in some cases and so severe as to suddenly produce death from asphyxiation. The muscles of the larynx are also involved, complete closure being sometimes produced.

Spasm of Extremities.—The legs become extended and rigid but as a rule the arms are but little affected.

Temperature.—The body heat is usually somewhat raised and in occasional cases may be very high.

Pulse.—The pulse increases in frequency and is usually small

Mental Condition.—The mind is perfectly clear in all cases.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The symptoms of this disease are so peculiar that it is not possible to make a mistake in diagnosis except in the

very early stages, when rigidity of the muscles of the neck may be thought to be due to exposure to cold. But when trismus comes on there is no further doubt as to the diagnosis.

WHAT IS THE PROGNOSIS?

The death rate is very high. When due to trauma about ninety per cent. die. In idiopathic cases about fifty per cent. are fatal. Fatal cases die in less than a fortnight. If the patient survives for two weeks the chances of recovery are good. The cause of death is usually asphyxia or heart failure.

WHAT IS THE TREATMENT?

GENERAL.—The patient should be kept absolutely quiet, in a darkened room, and free from noise or worry. Care should be taken that he does not hurt himself during the intensity of the spasms, and that he does not bite his tongue.

DIETETIC.—Liquid food should be given in as large quantities as the stomach can bear. If the jaws are set the patient may sometimes be fed between the teeth, when they are wide apart or when one is missing. It is not well to extract a tooth in order to feed the patient because the irritation may induce increase of spasm. Nutritive enemata of milk and eggs may be given if there is no way of feeding by the stomach.

REMEDIAL.—The use of chloroform is a dangerous procedure. While it may relieve for the time being, yet when the patient recovers from its effects the spasm is generally increased. The best results can only be had by the use of homeopathic remedies.

Aconite.—Spasm of the eyes; clinched jaws; body becomes rigid and bends backward; limbs distorted with spasm; excessive restlessness and tossing about.

Hydrocyanic acid.—Jaws firmly fixed; lies on bed with head fixed and thrown backward; legs fixed and rigid; spasm of respiratory muscles; paroxysms come on without any apparent cause, dreads their approach, and cannot sleep for fear of the attacks which come on just when he is dropping off to sleep; cyanotic appearance; tetanic grin.

Nux vomica.—Jaws rigidly closed: head constantly thrown back; violent painful paroxysms in which the limbs are extended: body bent like a bow; muscles of chest rigid, impeding respiration to such a degree that the face is purple at intervals of one-half an hour to an hour; mind perfectly clear; cough between the paroxysms.

Strychnia.—Pains like electric shocks flash through the limbs: tetanic rigidity of the body with opisthotonos and trismus, suspended breathing and, finally, paralysis; all the senses are acute.

TETANY.

WHAT IS TETANY?

It is a spasmodic disorder characterized by attacks of tonic spasm of the limbs with excessive sensitiveness of the motor and sensory nerves.

WHAT ARE ITS CAUSES?

Exhausting influences, such as diarrhea, fatigue, the results of mental shock and worry, exposure to cold, alcoholism, and irritation from intestinal worms. It occurs most frequently under three years of age and, again, about the time of puberty.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is usually a hyperemic condition of the gray matter of the spinal cord, and, in children, of the meninges of the brain.

WHAT ARE THE SYMPTOMS?

It generally begins suddenly with symmetrical tonic contractions of the hands, and sensations of numbness and tingling in the extremities. At first it is confined to the upper extremities, involving the flexors of the forearm, hand and fingers, with contraction of the muscles of the back and face and of the lower extremities later. These spasms may last for a few minutes to hours or days and may occur during day or night, sometimes awakening the patient from sleep. There is increase of irritability of the motor nerves, and if the motor point of a muscle is struck there is excessive muscular action. Electrical irritability is also increased.

WHAT IS THE DIAGNOSIS?

The disease may be easily diagnosed by the character and symmetrical nature of the spasm, with excessive excitability of muscles and nerves.

WHAT IS THE PROGNOSIS?

Most cases recover, but the disease may continue at intervals for years. Sometimes only one or two attacks occur during the patient's lifetime.

WHAT IS THE TREATMENT?

The remedies may be used that are applicable for tetanus.

HYDROPHOBIA—RABIES.

WHAT IS HYDROPHOBIA?

It is an acute infectious disease of animals, dependent upon an unknown specific poison and may be communicated to man by inoculation.

WHAT ARE ITS CAUSES?

As the definition implies, it is due to inoculation of a specific virus. It is in most instances communicated to man by the bite of a dog; but wolves, cats, horses and other animals may be affected and cause it in man.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is usually considerable congestion of the brain and spinal cord, with dilatation of small vessels and an accumulation of the leucocyte-like corpuscles around them and in the tissues. There may also be clots within the small vessels and minute hemorrhages. These changes are most common in the cerebral cortex and in the medulla. Myelitis is sometimes present, even in acute cases.

WHAT ARE THE SYMPTOMS?

Period of Inoculation.—This may vary. In children it is much shorter than in adults. The average period is from six weeks to two months or even three months, and some-

times a year elapses from the time of the bite until the onset of the first symptoms.

PREMONITORY SYMPTOMS.—There is first noticed a numbness or pain about the parts bitten, with loss of appetite, depression of spirits, headache, irritability, sleeplessness, a sensation of impending danger, and hyperesthesia of all the senses.

Stiffness of the Muscles of the Throat.—Throat rigidity with slight difficulty in swallowing may be the first symptom noticed. The voice is husky.

STAGE OF EXCITEMENT.—During this stage the active symptoms of the disease manifest themselves. There are great restlessness and hyperesthesia. The least sound or draught of air may produce a violent reflex spasm. There is great dread of water and the sight of it will produce a spasm.

Spasm.—This effects particularly the muscles of the larynx and mouth, is extremely painful, and accompanied by an intense sense of dyspnea. If the patient attempts to take water there is a painful spasm of the muscles of the pharynx, larynx, and elevators of the hyoid bone. With these spasms there may be delirum, hallucinations and delusions, and the patient may attempt to injure those about him, and yet will try to avoid doing such injury, seeming to be alive to the situation. This is the most painful period of the disease. The spasms may last for a half a day to a day or two, and the mind may be clear when they are present. The spasms may involve all of the muscles of the body.

Paralytic Stage.—Paralysis usually follows the preceding stage. The patient becomes quiet and unconsciousness gradually comes on. The action of the heart becomes weaker and death occurs from heart failure.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

There is usually no difficulty in distinguishing hydrophobia from other nervous diseases.

It is differentiated from tetanus by the length of time which occurs after the inception of the bite, and the fear of water.

WHAT IS THE PROGNOSIS?

Usually grave. The inoculations of Pasteur have, however, materially changed the prognosis. When possible they should be availed of.

WHAT IS THE TREATMENT?

PREVENTIVE.—When it is known that a wound has been caused by the bite of a dog suffering from rabies it should be thoroughly cauterized and made aseptic. Sometimes the sucking of the wound, expectorating the saliva, will materially decrease the quantity of virus taken into the system.

GENERAL.—When the disease is fully developed the patient should be kept in a darkened room and perfectly quiet, and anything which may produce the spasm, such as a draught of air or any mental excitement, should be guarded against.

REMEDIAL.—*Belladonna.*—Repeated convulsions and horrible spasms with screams and loss of consciousness; difficult deglutition; dilated pupils; red or livid countenance; throwing the body from one side to the other; starts in a fright just as he is going to sleep.

Cantharides.—Convulsions and hydrophobic symptoms; any bright light, drinking, or sound of falling water causes a spasm; tetanic spasms followed by coma.

Hyoscyamus.—Constant state of erethism; not a single part of the whole body quiet for a moment; uninterrupted, irregular motions full of impetuosity; whole body twisted and turned continually; unceasing spitting; frothing at the mouth; eyes staring and distorted; suffocative spells.

Stramonium.—Fainting fits every day; suddenly falls as if dead with pale face and almost imperceptible breathing; if disturbed during paroxysm rolls about the floor, becomes enraged, and bites those around him; convulsions, alternating with rage; paralysis after convulsions.

NEURASTHENIA.

WHAT IS NEURASTHENIA?

It is a diminution of nervous energy with increased reaction, both mental and physical, to external impressions.

WHAT ARE ITS CAUSES?

Heredity is the most important cause, the parents having suffered from irritability of the nervous system, from some organic nervous disease, or from alcoholism. The school is responsible for many cases. The child is not properly disciplined at home and is over-worked at school. In the adult the causes are great mental anxiety, over-work, excitement, worry, sexual excesses, alcoholic excesses, trauma, and the results of typhoid fever, malaria and influenza.

WHAT IS THE PATHOLOGICAL ANATOMY?

Changes due to nerve exhaustion involve the nucleus, the cell-protoplasm, and even the cell-capsule when it is present. There is a marked decrease in the size of the nucleus and it becomes smooth and rounded. There is also a shrinkage in the size of the cell-protoplasm with vacuolation for the spinal ganglia. There is usually cerebral hyperemia and also hyperemia of the gray matter of the cord. The nerve cells easily break down under slight irritation, and send out feeble impulses.

WHAT ARE THE SYMPTOMS?

Motor.—Weakness of the legs and back is complained of more frequently than weakness of the upper extremities. Tremor and exaggerated reflexes are also present.

Sensory.—There is a vague feeling of distress all over the body. The patient feels tired all of the time and becomes easily fatigued. There is also a sensation of lightness or emptiness of the head, and a feeling of uncertainty in making voluntary movements. Dizziness, with staggering and inco-ordination of movement, is sometimes met. Pain in the head, back and limbs, of a dull, diffused character. The headache may be severe and last for days at a time. In other cases it may be mild and pass away after the patient stops working. Pain in the small of the back with extreme fatigue, and aching of the limbs upon slight exertion.

Spinal Tenderness.—The patient flinches when the sensitive spinous processes are pressed upon, even slightly. There is also a burning sensation down the spine. The sensitive spots are not continuous the whole length of the spine, but

occur in three or four places. The seventh vertebra, upper dorsal and lower dorsal are the points most usually affected. Cutaneous hyperesthesia over the back, on the extremities, the scalp, face, nipples or testicles may be present. Formication and prickling sensations often occur.

Disorders of Sight.—The patient cannot use his eyes for reading but a short time without suffering fatigue of eyes and head. With this vision becomes difficult and the eyes become sensitive to light.

Disorders of Hearing.—Hyperesthesia of the sense of hearing is common. Roaring, buzzing, whistling, ringing, throbbing or ticking sounds may be brought on by fatigue either mental or physical.

Disorders of Smell.—Excessive sensitiveness to odors is a common and uncomfortable symptom. Complete loss of smell may be present in some cases.

Disorders of Taste.—Loss of taste, or food tastes like sawdust; or there may be a bitter, salty, or acid taste.

Psychic Disturbances.—There is a diminished capacity for sustained intellectual efforts. Slight use of the brain will bring on a sense of exhaustion, or headache, giddiness or lightness about the head. It is difficult for the patient to concentrate his mind upon any one subject, and there is also a dislike for mental work which formerly pleased him. Loss of memory; irritability; trifles exciting him to great anger; mental depression; lessening of affections for those nearest to him; insomnia; fear of darkness, of crowds, of being alone, in close places, and of special localities; the handwriting becomes jerky and irregular; stammering or scanning speech; inability to enunciate words properly, but when he attempts to make the effort to speak clearly he can do so. Patients are liable to run their syllables, words and even phrases together. There is huskiness of the voice after becoming fatigued.

General Physical Disturbances.—There may be indigestion, palpitation of the heart, increased or diminished perspiration, increased or diminished quantity of urine, nocturnal seminal emissions, with weakness and irritability of the sexual organs.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis is usually easy. The symptoms of weakness which have been enumerated, without the association of any organic lesion, will make the case clear. It may be differentiated from hysteria by the slowness of onset of the symptoms, by the absence of convulsions or anesthesia and globus hystericus, and the fact that the symptoms are about the same from day to day and do not change from day to day as in hysteria.

WHAT IS THE PROGNOSIS?

This is usually favorable as far as recovery is concerned, but some of the cases are long-lasting. If the cause which produces the trouble can be removed the recovery is usually prompt and rapid. Some patients are confined to bed for years on account of the general exhaustion and exacerbations of pains when moving about.

WHAT IS THE TREATMENT?

GENERAL.—Sometimes a long vacation will stop the trouble. At any rate, the patient must decrease his hours of work and be relieved from mental anxiety as far as possible. Change of scene and travel may sometimes relieve the sleeplessness and enable the patient to recuperate.

REMEDIAL.—*Agaricus.*—Spinal column sensitive to touch; aching along back and limbs; great sexual desire; extremities go to sleep easily; sensation as if a cool current of air were passing from the spine all over the body.

Anacardium.—Sadness; weakness of memory; sensation of band around the head; trembling from every motion; anxiety when walking as if someone were pursuing him; full of suspicion.

Calcarea carbonica.—Great exhaustion in the morning; unable to go up stairs on account of exhaustion and shortness of breath; he may feel well, but every exertion or excitement produces exhaustion.

Gelsemium.—Muscles feel bruised and will not obey the will; loss of muscular control; sleeplessness from nervous exhaustion; brain-fag; excessive irritability of mind and body; dropping of eye-lids.

Phosphorus.—Nervous sensations and weakness; burning in small spots along the spine; stumbling gait from weakness; involuntary urination from weakness of the sphincters; palpitation of the heart.

Picric acid.— Patient dreads any mental or physical work, but improves when warmed up to it; legs tremble with great nervousness; feeling of loss of power.

Zincum.—Neurasthenia with hyperesthesia; burning between shoulder-blades and along the whole spine; stiffness and tension of the neck; frequent jerks in the limbs.

HEADACHE.

WHAT ARE THE CAUSES OF HEADACHE?

This is a common nervous symptom and may be due to many causes. It occurs during all periods of life and in all kinds of persons. Women suffer more frequently than men, and it is more common in persons living in cities than in the country. Those following literary and sedentary pursuits suffer more frequently than those following manual occupations. Trauma producing concussion of the brain, trauma of the brain, or laceration of brain substance; passive congestions due to pressure about the neck, or tumors pressing upon veins of the neck; active congestion from prolonged mental or physical strain; the first stages of meningitis; the result of the action of certain drugs, as nitrite of amyl, alcohol and glonoine; anemia from loss of blood or from prolonged mental exertion; toxemia, such as occurs in acute specific fevers, uremia, diabetes, gout, rheumatism, lithemia, gastric disturbances; syphilis and its results upon the vessels of the brain; organic diseases, as tumors, abscesses, aneurisms; caries of the cranial bones; reflexes from diseases of the eyes, nose and throat, ears, intestinal tract, sexual organs; various nervous diseases such as hysteria, epilepsy and neurasthenia; and toxic causes, as lead, tobacco, opium and chloral, are all responsible for headache.

WHAT IS THE PATHOLOGICAL ANATOMY?

In many cases there are no changes evident in the brain. When due to organic diseases certain changes are manifest.

It is difficult to determine what is the actual condition which produces the pain. Undoubtedly it is an irritation of the meningeal nerves which causes it. It is the membranes and not the brain itself which are chiefly concerned in the production of headache.

WHAT ARE THE PECULIAR CHARACTERISTICS OF HEADACHES?

It may be pulsating when due to circulatory disturbances; dull and heavy when produced by toxemia; binding or constrictive when occurring in neuropathic conditions, such as hysteria and neurasthenia; burning or sore when resulting from rheumatism or from gout. The headache may be frontal when due to constipation, errors of eye refraction, or gastric dyspepsia; on the vertex when due to anemia, uterine troubles, or disease of the bladder; temporal when caused by decayed teeth or inflammation of the middle ear; occipital when due to spinal irritation or displacement of the uterus.

WHAT IS THE PROGNOSIS?

The pains may be constant for a day, week, or even longer. Some persons suffer continuously for years with severe headaches. When due to organic brain disease the prognosis of the primary condition will be the prognosis for the headache.

WHAT IS THE TREATMENT?

GENERAL.—When headache is due to gastro-intestinal disturbances care in regard to the diet is of the utmost importance. Prohibition of sweets, indigestible substances and stimulants of all kinds should be insisted upon. The removal of the cause of the headache when it can be removed is absolutely demanded. During the attack the patient will use his own inclination as to whether it is better to keep quiet or move around in the open air.

REMEDIAL.—*Antimonium crudum.*—Headache from bathing or wetting the head or after smoking; rush of blood to the head; bursting pains; disordered stomach.

Arsenicum.—Periodical headaches; brain seems to be loose on moving the head; general confusion and heaviness in the house; better in the open air; hair falls out; nausea.

Baryta carbonica.—Headaches of aged; aggravation after walking, eating, or from a warm stove; pressing headache just over the eyes; feeling of tightness in occiput; vertigo with nausea on stooping.

Belladonna.—Right-sided headache; throbbing pains; cerebral congestion with intolerance of light and noise; hot head, cold feet; headache relieved by sitting propped up.

Bryonia.—Headache begins in occiput or else in forehead, extending into the face or neck; worse from the least motion, even from moving the eyeballs or stooping; gets sick and faint on sitting up; sour, bitter vomiting; vertigo and sensation of fullness in head, worse in the morning.

Carbolic acid.—Congestive headache; sensation of a band around head; extremely sensitive to odors; pain in right side of head.

Carbo vegetabilis.—Frontal headache worse in the morning when waking from sleep, having spent the best part of the night carousing; occipital headache with bilious symptoms; humming or buzzing in the head as though a hornet's nest were located there; worse in warm room.

Iris versicolor.—Sick-headache periodically every Sunday; school-teacher's headache; pains intense, throbbing, and preceded by blurred vision, often causing temporary blindness; supraorbital headache; vomiting frequent when the headache is worst.

Lachesis.—Left-sided headache, aggravated or caused by the heat of the sun; headache at the climacteric period or with cold in the head; pains relieved as soon as any suppressed discharge appears.

Lycopodium.—Tearing pains back and forth in forehead; worse from mental exertion and in the evening; rush of blood to the head.

Nux vomica.—Occipital pains with bilious attack; begins in the morning and increases all day until night; sour or bitter taste in the mouth; nausea and vomiting; ineffectual retching; accumulation of flatus.

Platina.—Squeezing headache as if a board were pressed against the forehead; pain increases and decreases gradually; crampy pains as if squeezed at the root of the nose.

Sanguinaria.—Violent pains in occiput, extending over head and settling over the right eye; cannot bear sound or

odors, or anyone walking across the floor, as the slightest jar annoys; must remain in a quiet, dark room; pains so violent that she is apt to go out of her head; nausea and vomiting.

Spigelia.—Pains come from nape of neck and settle over the left eye; sick-headache: sensation as if the head were open all along the vertex: headache reaches its acme at noon.

MIGRAINE—SICK HEADACHE—HEMICRANIA.

WHAT IS MIGRAINE?

It is a severe, paroxysmal, periodical pain in the head, usually unilateral, and generally associated with nausea, vomiting, and disorders of the vision.

WHAT ARE ITS CAUSES?

Some cases begin in childhood and are produced by overwork at school. Injury, shock, or exhausting diseases may produce the trouble. It may occur frequently in several members of a neurotic family.

WHAT ARE THE SYMPTOMS?

There may be a sense of malaise and depression several days before the attack, or these symptoms may last only a few hours before the pain comes on. It usually commences in the morning and gradually increases in frequency until the patient has to stop his occupation and lie down. The pain commences on one side of the head, either in the forehead or occiput, and increases until it finally involves the whole head. It is of a throbbing character, increased by the slightest noise, light, or jar. There is usually dimness of vision or flashes of light before the eyes. Confusion of ideas and dizziness are common. At the climax there are nausea and vomiting, which sometimes relieve. The face is usually pale, the pulse hard and small. These attacks may last six, twelve, or twenty-four hours, and even for several days. When the attacks begin to lessen the patient falls asleep, and when he awakens he feels much better than before the attack. The intervals between the attacks vary considerably. Sometimes they occur every week, every fortnight, or once a month.

WHAT IS THE DIAGNOSIS?

The periodicity, the method of beginning, and evolution of the attack, culminating in nausea and vomiting, make the diagnosis easy.

WHAT IS THE PROGNOSIS?

Some cases may continue for years with great regularity. As a rule when the attacks become once established they persist at varying intervals throughout the patient's life. They have no tendency to shorten life.

WHAT IS THE TREATMENT?

The same as that for headache in general.

NEURALGIA.

WHAT IS NEURALGIA?

It is a functional disease of sensory nerve fibres and characterized by pain. It may be idiopathic or symptomatic.

The most common form of neuralgia is that affecting the fifth nerve, of which there are two forms; symptomatic, trigeminal neuralgia and tic douloureux or prosopalgia.

WHAT IS THE PATHOLOGICAL ANATOMY OF NEURALGIA?

There is usually a low grade of neuritis, but the nerve does not appear to be changed.

WHAT ARE THE CAUSES OF SYMPTOMATIC TRIGEMINAL NEURALGIA?

It occurs most frequently in women and more often upon the left side. Caries of teeth, malarial poisoning, anemia, exposures to cold; frequent pregnancies, eye-strain, diseases of the nares, gout, syphilis, diabetes, trauma, rheumatism, and the great neuroses, hysteria and epilepsy, may all be causes of the disease.

WHAT ARE THE SYMPTOMS?

The pains are sharp, shooting, lancinating and intense, with periods of exacerbation and remission. There are often tender points along the course of the nerve, and over

the parietal eminence and vertex. The pain may extend all over the head from the occiput to the frontal region, involving the ear and orbit.

WHAT IS THE COURSE OF SYMPTOMATIC TRIGEMINAL NEURALGIA?

It is relieved when the disease which produces it has passed away.

WHAT ARE THE CAUSES OF TIC DOULOUREUX?

It occurs most frequently in persons over forty years of age, and is usually brought on by exposure, over-work, depressed mental condition and diseases of the teeth and jaws.

WHAT ARE THE SYMPTOMS?

There is intense darting pain, which usually starts on the upper lip and on the side of the nose and radiates into the eye, to the temple and side of the head, and through to the teeth. It is usually confined to one side, and during the pain the face is flushed, there is increased flow of water from the eyes and nose, and the expression of the patient is one of great agony. These paroxysms may last for a few moments and then diminish in severity, but do not pass entirely away. The least draught of air, speaking or eating, will bring on a spasm. The pains are usually worse during cold weather and cease during summer.

WHAT IS THE DIFFERENTIAL DIAGNOSIS OF NEURALGIA?

A sharp, shooting, lancinating pain along the course of a nerve is characteristic of neuralgia in any part of the body.

It may be differentiated from rheumatism by its occurrence only along the course of a nerve.

WHAT IS THE PROGNOSIS?

The prognosis depends upon the cause; it is usually favorable under homeopathic treatment.

WHAT IS THE TREATMENT?

GENERAL.—Rest of the part involved is most important. Local applications of heat are usually of great benefit.

REMEDIAL.—*Aconite.*—If occurring from exposure to dry, and cold winds; violent congestion of face, which is hot, red swollen; neuralgic pains in any part drives the patient to despair; worse at night; pains burning, lancinating, pulsating, tingling and benumbing.

Arsenicum.—Malarial neuralgia: recurs periodically, mostly in the face; little fine burning needles about face, following the course of the nerves; burning, tearing pains especially at night; great anguish; excessive weakness; affected parts feel cold; worse after prolonged exercise.

Belladonna.—Right side; paroxysms after gradually increasing to an intolerable acuteness cease suddenly; lancinating, burning pains, worse by motion, light, shock, or contact; worse from lying down; better by sitting up.

Bryonia.—Neuralgic pains left side of face and head; pressing, tearing, shooting pains; better by hard pressure and from cold applications.

Chininum arsenicosum.—Violent neuralgic pains in left mammary region as if it were torn out with a red-hot tongs; worse by motion; pains come and go quickly; restless.

Cimicifuga.—Neuralgic pains in any part of the body when reflex from uterine or ovarian disease; sensation of heat on top of the head; feeling as if top of head would fly off; sharp, lancinating pains over the eyes; great anxiety and nervousness.

Croton tiglium.—Brachialgia; shooting, tearing pains extending the whole length of the limb; inability to move or lie down; the least attempt to stir causes pain; arm becomes paralyzed and feels like a heavy weight.

Gelsemium.—Intense pain in the upper portion of the spinal cord and brain, commencing in the occiput, passing through the brain, and ending in the forehead and eyeballs; better by bending head backward.

Ledum.—Intercostal neuralgia, especially in axillary region; worse from motion; constant chilliness; patient morose; painful pressure in both shoulder-joints.

Spigelia.—Neuralgia begins in back of head and comes forward; left prosopalgia with severe burning, sticking pains; neuralgia comes and goes with the sun; intense excitement and great intolerance of the pain.

Sulphur.—Malarial neuralgia occurring mostly in the

face and resisting other remedies; worse every day at noon or at midnight; gradually increasing to its height and then gradually decreasing.

Terebinthina.—Brachial neuralgia mostly evenings and during the night in bed until morning; subscapular and supra-orbital neuralgia; sudden twitchings of the limbs as from electrical shocks; intense pain along the larger nerves; numbness of limbs; motion difficult, as it starts or increases the pain.

VERTIGO.

WHAT IS VERTIGO?

It is a sensation of movement on the part of surrounding objects which are really at rest, objective vertigo; or on the part of the person himself, subjective vertigo.

WHAT ARE ITS CAUSES?

Cerebral anemia or hyperemia, irritation of the auditory nerve, causing true auditory vertigo, occurring as a symptom in disease of the labyrinth, toxemia as the result of gastro-hepatic derangements, valvular disease of the heart, neurasthenia, epilepsy, eye-strain, organic disease of the brain, and such mechanical causes as swinging rapidly, rotating motions, or the rolling of a ship at sea.

WHAT IS THE PATHOLOGICAL ANATOMY?

Except in aural vertigo or organic brain diseases there are usually no pathological changes.

WHAT ARE THE SYMPTOMS?

Dizziness, with partial blindness or wave-like sensations before the eyes. In severe cases it is accompanied by nausea and vomiting. In mild cases it lasts but a moment or two and then passes away.

WHAT IS THE TREATMENT?

GENERAL.—Remove the cause if possible.

REMEDIAL.—*Esculus.*—Vertigo with sensation of balancing in the head; sensation as if intoxicated; dull, stupefying headache.

Agaricus.—Strong sunshine causes momentary vertigo with staggering gait and imperfect vision either for near or distant objects; vertigo from mental exertion; tendency to fall forward.

Aloes.—Revolving vertigo aggravated by turning quickly; insecurity in walking or standing; vertigo after dinner.

Argentum nitricum.—Morning dizziness with headache; complete but transitory blindness with nausea and confusion of the senses; buzzing in the ears and general debility of the limbs.

Arsenicum.—Vertigo with reeling during a walk in the open air, and stupid feeling in the forehead as if intoxicated; obscuration of sight when raising the head; nausea when in the recumbent position.

Belladonna.—Vertigo with nausea when waking from sleep in the morning after a night of revelry; dizziness relieved in the open air.

Bryonia.—Vertigo when rising from a chair, disappearing after walking, with weakness of the limbs.

Causticum.—Dizziness at stool and after it, with nausea; violent dizziness in the morning on waking, with painful dullness of the head.

Cocculus.—Vertigo with flushed, hot face; confused feeling in head after eating and drinking.

Cyclamen.—When leaning against something feels as if the brain were in motion or as if he were riding in a carriage with his eyes closed; despondent and irritable.

Hydrocyanic acid.—Insufficiency of arterial contraction with frequent headaches; stupefaction and falling down; sees through a gauze; can scarcely keep his feet when raising the head after stooping.

Moschus.—Sensation as if he were turned about so rapidly that he perceives a current of air produced by the motion; sensation as if falling from a height; vertigo on moving the eyelids and on stooping, passing away on rising.

WRY-NECK—TORTICOLLIS.

WHAT IS WRY-NECK?

An unnatural position of the head due to contraction of the muscles of the neck. It may be a permanent shortening of a muscle or simple spasm.

WHAT ARE ITS CAUSES?

It may be congenital, due to atrophy of the sterno-cleido-mastoid muscle while within the uterus. Delivery by instruments may also produce it by causing extreme traction of the neck. These causes produce permanent contraction of the muscle. It may also be due to spasm of the sterno-cleido-mastoid.

The simple spasm is caused by exposure to cold; falling, when the patient strikes upon the side of the neck; mental or physical shocks; a strain in the muscles of the neck may also produce it. It occurs much oftener in women than in men, and during early adult and middle life. The spasmodic form never occurs in children. There is usually a neuropathic disposition present in the individual.

Figure 39.
Wry-Neck.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is evident irritation of the spinal accessory nerve, but where that irritation is located it may be difficult to determine. It may be in the cortex or along the course of the nerve itself.

WHAT ARE THE SYMPTOMS?

Pain in the sterno-cleido-mastoid muscle like that of an ordinary stiff neck may be the first symptom, this being soon followed by a spasm of the muscle. The head, when one muscle is involved, is inclined toward the affected side, the chin is raised, and the head rotated to the opposite side. The upper fibres of the trapezius muscle are usually affected, together with the sterno-cleido-mastoid, and when both trapezii are involved the head is pulled backward.

WHAT IS THE PROGNOSIS?

It does not shorten life but usually progresses to a certain stage and then becomes chronic. Sometimes it is cured, though rarely.

WHAT IS THE TREATMENT?

GENERAL.—Electricity applied to the affected muscles may sometimes help to relax the spasm. In the congenital form surgical measures may have to be resorted to, such as tenotomy and re-section of the nerve. In the spasmodic form surgical measures should never be resorted to.

REMEDIAL.—*Aconite.*—Drawing in the muscles of the neck with tearing pain, worse by moving the neck; pains extend down to the shoulder in cases due to cold.

Arsenicum.—Neck stiff as if bruised or sprained; neuralgic pains in left side of neck.

Belladonna.—Painful swelling and stiffness of neck, worse by bending the head backwards; feeling as if the head would break when coughing.

Lachnanthes.—Pain and stiffness in neck, going over the whole head when turning the head or bending it backwards; head drawn to one side.

Rhus toxicodendron.—Stiff neck, with painful tension when moving, caused by sleeping on damp ground.

EXOPHTHALMIC GOITRE—BASEDOW'S OR GRAVES' DISEASE.

WHAT IS EXOPHTHALMIC GOITRE?

It is a disease characterized by enlargement of the thyroid gland, protrusion of the eyes and peculiar symptoms about the heart.

WHAT ARE ITS CAUSES?

It occurs more frequently in women during early adult life, between fifteen and thirty-five. Anemia or general exhaustion of the system in a person of a neuropathic tendency may produce it. Great physical exertion or depressing emotions may also be the cause.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is a relaxation of the walls of the bloodvessels, with cutaneous hyperemia, enlargement of the thyroid gland, and deposit of fat within the orbit. Hemorrhages and hyperemia of the medulla have sometimes been found. There is usually hypertrophy and dilatation of the heart.

WHAT ARE THE SYMPTOMS?

Palpitation of the Heart.—Heart palpitation with pain about the heart and shortness of breath may be the first symptoms noticed. Upon examination of the heart nothing can be determined except its rapid action, the pulse reaching from one hundred and twenty to one hundred and sixty per minute.

Exophthalmus and Enlargement of the Thyroid Gland.—These two symptoms usually come on together a little while after the palpitation of the heart has been observed. Exophthalmus may not be present in all cases, but enlargement of the thyroid is. A thrill is felt in the enlarged gland with the pulsations of the heart, and a venous hum is sometimes noticed in the neck. As the exophthalmus increases the patient is unable to close the lids and the dry conjuctiva becomes inflamed. Opacity and ulceration of the cornea may ensue. When the patient attempts to look downward the upper lid follows the ball.

Tremor.—This is an almost constant symptom. It is usually slight and fine, but may be coarse. The hands only may be affected, or the tongue may be involved.

General Condition.—The patient is usually irritable, depressed, hysterical, or neurasthenic; insomia is present, as well as anemia and emaciation. Intermittent albuminuria is often noticed in these conditions. Increase in the quantity of urine is frequently observed. Profuse sweating upon the least exertion often occurs.

WHAT IS THE DIAGNOSIS?

In well-developed cases a diagnosis is easily made. If the exophthalmus be not present the increased action of the heart, with tremor, sweating, and general nervousness, will enable us to diagnose the disease in the early stage.

WHAT IS THE PROGNOSIS?

A large number of cases improve materially and a few get entirely well; others are not benefited at all. The disease may increase rapidly and cause death.

WHAT IS THE TREATMENT?

GENERAL.—Rest is of the utmost importance, both physical and mental. In severe cases the patient should be put to bed and kept there for a month or two or even longer. Under any conditions over-exertion, mental excitement and worry should be avoided as far as possible.

REMEDIAL.—*Aurum.*—Prominent, protruding, staring eyes; tensive pressure in the eyeballs; tremulousness; frequent urination; restlessness and palpitation.

Belladonna.—Protruding, staring, half-opened eyes; eyes shining; violent palpitations, reverberating in head; pressure in cardiac region.

Bromium.—Protrusion of eyes; anxious feeling about the heart; goitre; sweat from least exertion; emaciation.

Calcarea carbonica.—Stiffness of eyeballs; inability to move the eyes without unpleasant sensations; excessive palpitation, with irregular pulse.

Gelsemium.—Excessive action of the heart; pulse frequent, soft and weak; great nervous excitement; excessive irritability of mind and body; sweating upon least exertion; sleeplessness; excessive nervous hyperesthesia and vascular excitement.

DIPHTHERITIC PARALYSIS.

WHAT IS DIPHTHERITIC PARALYSIS?

A form of paralysis beginning in the second or third week after the disappearance of the throat symptoms in a case of diphtheria.

WHAT ARE ITS CAUSES?

It occurs most frequently in adults, but may occur in children, and is due to a toxemic condition produced by the diphtheritic poison and affecting the nerves.

WHAT IS THE PATHOLOGICAL ANATOMY?

The muscles are usually found normal, but there may be granular and fatty degeneration of the fibres. There may also be degeneration of the nerves supplying the paralyzed parts, either in the extremities of the nerves or along their whole course. It is a simple degeneration, often called parenchymatous neuritis. The motor nerve cells of the anterior cornua of gray matter of the spinal cord are sometimes found swollen and unduly homogeneous and vitreous in aspect, smaller than normal, and with shrunken processes.

WHAT ARE THE SYMPTOMS?

Paralysis of the Palate.—This feature is manifested by regurgitation of liquids through the nose when attempting to swallow; nasal tone of the voice due to shutting off the cavity of the nose during phonation; inability to gargle the throat; when the patient utters the sound "ah" the palate is not raised.

Paralysis of the Pharynx.—This symptom is not common but sometimes may occur; when present to a great degree prevents swallowing. It is a serious sign when it does occur. The voice is hoarse, food gets into the larynx when swallowing, producing coughing and strangling.

Eye Symptoms.—There is loss of power of accommodation of the eye, due to paralysis of the ciliary muscles. Vision for distant objects is normal, but is impaired for near objects. Both eyes are always affected. Pupils respond sluggishly to light. Divergent squint is often present.

Paralysis of the Limbs.—Soon after the affection of the palate a gradually increasing weakness of the lower extremities is observed. The muscles become flabby and without tone, the knee-jerk is lost, the legs are moved as if they were heavy and limp. These symptoms gradually increase until the patient is unable to walk. The arms are also affected to a greater or less degree after the lower limbs have become involved.

Sensory Symptoms.—Sensations of numbness, tingling, pins and needles, and hyperesthesia may last for a time and be followed by diminished sensibility, or even complete anesthesia of different parts may be present.

Inco-ordination.—Both upper and lower extremities may be affected; a condition resembling locomotor ataxia is often present.

The Trunk Muscles.—These are not usually affected, but may become so weak that the patient will not be able to sit up or turn over in bed.

Convulsions.—Spasms are sometimes present in severe cases.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

Gradually increasing paralysis of the palate and lower extremities, coming on two or three weeks after an attack of diphtheria with flaccidity of the muscles, will make the diagnosis easy.

WHAT IS THE PROGNOSIS?

It is usually good unless the symptoms commence with great severity, when the disease may prove fatal on account of heart-failure. Generally improvement begins in a few weeks and in two or three months the patient will have entirely recovered.

WHAT IS THE TREATMENT?

GENERAL.—The patient should be kept quiet in bed and not allowed to make any physical exertion. It is of the utmost importance to keep up the patient's strength, and in order to do this liquid food may have to be given by means of a stomach-tube, or it may be given by injections into the rectum.

REMEDIAL.—*Argentum nitricum.*—Paralytic heaviness and weakness of the legs with sick feeling, drowsiness, chilliness and sickly appearance; limbs, especially the knees, start up at night, awakening the patient; legs feel as if made of wood.

Baryta carbonica.—Dragging the thighs, particularly when going up stairs on account of paralyzed feeling in the middle of the thigh; knees totter when attempting to walk; coldness of the feet; burning soreness in the bends of the knees; sudden attacks of momentary pains in the limbs, with chilliness.

Causticum.—Hands and feet go to sleep; formication in limbs; paralytic weakness and trembling of limbs; intolerable uneasiness of limbs in the evening.

Cuprum.—Paralysis of lower limbs; frequent involuntary doubling up of the knees in walking; tingling in the extremities; weakness and weariness of limbs; coldness and bluish appearance of extremities.

Phosphorus.—Paralysis and formication in the extremities, with nervous debility; loss of power over all the limbs, especially in the joints as if paralyzed; hands and feet numb and clumsy; limbs tremble from every exertion; when walking makes missteps from weakness.

Zincum.—Weakness and weariness of the limbs, with trembling upon the least exertion; formication and coldness of feet; excessive nervous feeling in feet with constant attempts to move them; edema of the legs.

OCCUPATION NEUROSES.

WHAT ARE OCCUPATION NEUROSES?

They are certain conditions produced by attempts to perform some oft-repeated muscular action, usually one that is involved in the occupation of the sufferer. The most frequent symptom is spasm in the part, which prevents the intended action. The various kinds of spasm are writer's cramp, piano-player's cramp, telegrapher's cramp, and gold-beater's cramp. The most common form is writer's cramp.

WHAT ARE THE CAUSES OF WRITER'S CRAMP?

It is more common in males because fewer women are engaged in writing than men. It occurs during the active adult period of life. There is usually a neuropathic tendency, either hereditary or acquired. Excessive mental worry, intemperance, and anything which lowers the vital forces may be predisposing causes. Excessive writing is the exciting cause.

WHAT IS THE PATHOLOGICAL ANATOMY?

In some forms neuritis is undoubtedly present. In others some organic lesion of the brain may be present.

WHAT ARE THE SYMPTOMS?

There may be:

(1). A simple weakness of the muscles of the hand and

forearm, which allows them to become easily fatigued after writing a little while. This fatigue will gradually increase until it becomes so great that upon continuing the attempt to write the arm will fall powerless to the side. This is commonly known as pen paralysis or pen palsy.

(2). The pen may be held in the proper manner and the patient feel confident that he will be able to write, but the moment he attempts to do so there is a violent trembling of the hand, and the letters are so shaky that it is impossible to decipher them. He grasps the pen more tightly, and perhaps uses all the muscular power he has to steady his hand, but without avail, and finally gives it up from pure exhaustion. This is the tremulous variety, and is rarely manifested unaccompanied by spasm.

(3). After writing for some time a painful sense of fatigue is felt in the muscles, accompanied by a dull, heavy ache which commences in the fingers, extending to the hand, wrist, forearm, and finally passing to the shoulder. If writing is persisted in the pain becomes almost unendurable. It is most severe along the course of the median and musculo-spiral nerves, with here and there tender points exceedingly sensitive to pressure. This is the neuralgic form.

(4). The patient notices first that after writing a little while he unconsciously grasps his pen more firmly than usual. At the same time it does not move along as freely as it should. The letters become irregular; some are too high and others are too low, and the down strokes are shaky. The index finger frequently slips from the pen, which then falls between it and the second finger. After a while the hand, wrist and arm become painfully tired, and spasm, tonic in character, supervenes. The is true writer's cramp or graphospasm.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The symptoms are usually clear-cut and the disease cannot be mistaken for any other.

WHAT IS THE PROGNOSIS?

If taken in the early stage it may be cured, but if allowed to continue for any length of time it becomes incurable.

WHAT IS THE TREATMENT?

GENERAL.—The patient must at once stop writing, for if persisted in the trouble will increase steadily. All kinds of devices for holding the pen have been invented, but they only relieve the trouble for a short time.

REMEDIAL.—*Belladonna.*—Heaviness and paralytic feeling in the arm; spasmodic closing of the fingers; painful drawing of the middle joint of the right index finger; twitching of the arm.

Causticum.—Paralytic feeling in the right hand; pain in right wrist as if sprained; sensation of coldness in hand when grasping anything; painful numbness of thumb and index finger, especially when touching something; fingers half closed, cannot move them excepting with the other hand.

Cyclamen.—Cramp-like slow contractions of right thumb and index finger; tips approach each other and can only be extended by force; numbness in right hand; cannot open thumb and index finger.

Gelsemium.—Tired sensation in the arm after writing, steadily increasing; vague pains from tips of fingers to scapulæ; trembling of hand when attempting to write.

Nux vomica.—Muscles of fingers and thumb firmly and painfully contracted and pressed against the palm so that he is unable to open the hand; any attempt to open the hand causes severe pain.

Silica.—Tonic spasm of the hand while writing; cramp-like pain and lameness of hand after slight exertion; tearing pain in wrist and ball of hand, followed by paralysis of the hand.

Stannum.—Cramping of fingers on attempting to pick up the pen, or when they have been used a long time fingers become suddenly rigid, distorted, spread out, or contracted; the spasm can often be ended by opening fingers with unaffected hand.

TROPHO-NEUROSES.

RAYNAUD'S DISEASE.

WHAT IS RAYNAUD'S DISEASE?

A variety of dry gangrene described by Raynaud in 1862, and characterized as a neuroses dependent on an exaggeration of the excito-motor nerves.

WHAT ARE ITS CAUSES?

It usually occurs between twenty and forty years of age, and twice as frequently in women as in men. Children also sometimes suffer. A neuropathic tendency is present in most cases. Anemia, chlorosis, malaria, sexual excesses, syphilis, menstrual disorders, and exposure to cold may produce it. Laundresses often have it, due to the constant emersion of their hands in water during wet weather.

WHAT ARE THE SYMPTOMS?

Local Syncope.—There is first noticed a frequent occurrence of coldness, blanching or local syncope, and numbness of one or more fingers of the hand or hands which lasts for a few minutes, passes away, and is followed by redness and burning in the parts first affected. This condition occurs with increasing frequency and lasts longer until decided changes in the nutrition of the fingers take place. This condition is called digiti-mortui, or dead fingers. The fingers feel cold to the touch and are pulseless. If they be pricked with a needle no blood flows.

Local Asphyxia.—The tips of the fingers and toes, or the parts affected by previous local syncope, assume a bluish-black appearance. The nails look as if they had been in ink, the fingers and toes become blue and swollen, and there may be burning sensations and severe neuralgic pains.

Local Gangrene.—In this stage small blisters appear on the ends of the fingers and toes which fill with blood and serum, then dry up, and underneath the scab which forms, ulceration takes place. This may heal slowly and a slight scar be left. If the gangrenous process be very severe, the

skin may become black, dry and shrivelled at the ends of the fingers and toes. The line of demarcation between this and the healthy-tissue is very plain. Sometimes spontaneous amputation may result from extensive gangrene. Other parts of the body may be affected, such as the cheeks, lips, deltoid muscles, and heels.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

It has to be differentiated from senile gangrene and from frost-bite. The age of the patient will differentiate it in the first case, and the frequent recurrence of the local syncope will determine it in the second case.

WHAT IS THE PROGNOSIS?

There is a frequent recurrence of the disease after it has once existed in the patient, and the prospect of complete recovery is not very good. Sometimes the hand or foot may be amputated by the gangrenous process.

WHAT IS THE TREATMENT?

REMEDIAL.—*Arsenicum.*—Gangrene of extremities; the affected part is hot and painful; stinging and tearing around the old scars.

Carbo vegetabilis.—Humid gangrene in cachectic persons whose vital powers are exhausted; great prostration.

Lachesis.—Coldness of the part as if ice were in contact with it, followed by tingling, heat and numbness; skin cracked and deep rhagades; bluish or black looking blisters.

Secale.—Dry gangrene of the extremities; parts are dry, cold, hard, and insensible; the limbs become pale, cold, and shrivelled, losing all sensibility.

ANGIONEUROTIC EDEMA.

WHAT IS ANGIONEUROTIC EDEMA?

A disease characterized by the rapid appearance of circumscribed swellings on different parts of the body, but mainly upon the face.

WHAT ARE ITS CAUSES?

It occurs oftener in men than in women, and is known to run in families, showing an hereditary tendency in some cases. It occurs oftener in winter and in the early morning. Exhaustion, sudden exposure to cold, slight blows upon the part, great mental anxiety, grief, and the ingestion of some kinds of food, such as apples, fish, or strawberries may produce it.

WHAT ARE THE SYMPTOMS?

The condition comes on suddenly, without premonitory symptoms. In a few minutes or hours there may be developed a circumscribed edematous swelling upon the face, lips, tongue, hands, or genital organs. The parts feel stiff, with scalding, burning, and sometimes an itching sensation. The affected part is usually pale, but may be slightly reddened. The swelling may cover a space an inch or two in diameter, though it may be four inches in diameter, and it may be multiple or several swellings may coalesce. They may last a few hours or a few days and then pass away, to return after a few weeks or three or four months. When the disease attacks the throat dyspnea may be produced which is quite serious.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The symptoms of the disease are so peculiar that it is not easily mistaken for anything else. The sudden appearance of the edema at intervals, without evidences of inflammation is the pathognomonic condition for the diagnosis.

WHAT IS THE PROGNOSIS?

The attacks may recur frequently and some cases do not seem to recover completely.

WHAT IS THE TREATMENT?

GENERAL.—Articles of food which are likely to produce the trouble should be avoided.

REMEDIAL.—*Apis.*—Stinging, burning, prickling, smarting or itching sensation of the skin, with edematous swell-

ing; edema of the lips and face: face stiff and sensitive on pressure; hands swollen, white and glossy.

Arsenicum.—Face and lips swollen and edematous, with drawing and stinging here and there: face cold and pale; swelling and dryness of parts affected: burning, needle-like pains.

Belladonna.—Swelling of face, with slight redness and heat: tissues appear thickened; swelling lasts a day or two and then passes away; recurs frequently: vomiting may sometimes accompany the attacks.

Calcarea carbonica.—Circumscribed swelling in different parts of the body with itching: skin pale; bloated appearance of the skin: chronic form of edema: round swellings which appear in different places.

Rhus toxicodendron.—Smooth, shining swelling of the skin with itching: one side of the face swollen with hardness and thickness of the skin; edema after getting wet.

ACROMEGALY—MARIE'S DISEASE.

WHAT IS ACROMEGALY?

It is a disease characterized by a progressive enlargement of the extremities, thorax and bones of the face.

WHAT ARE ITS CAUSES?

It affects men about as frequently as women, and usually occurs between eighteen and thirty years of age. No special exciting causes are known.

WHAT IS THE PATHOLOGICAL ANATOMY?

Enlargement of the pituitary body has been found in some cases, which suggests that the disease may be due to some disorder of its function. As the function of this body is not well known nothing certain can be determined about it. Enlargement of the thymus gland has been found. Hypertrophy of the thyroid gland has been present in some cases while its atrophy has occurred in others.

WHAT ARE THE SYMPTOMS?

There is a gradual enlargement of the hands, feet and head. In women there is suppression of the menses, and in

men impotency. There is a general feeling of weakness and apathy, associated with frontal headache, dragging pains and anesthesia in the extremities. There is also increase of desire for food and drink, and increase in weight. The patient is not able to think as quickly as formerly. Excessive perspiration and increase of urine are noticed. The patient may stand severe cold without feeling it. When standing the neck is bent forward and the head is tilted backward. In order to bring his eyes upon a level the shoulders are bent forward. The gait is heavy and non-elastic. The nose, lips and tongue are sometimes enormously swollen, the ears become greatly increased in size, and the eye-lids grow thick. There is also enlargement of the superior maxillary bones, causing a lengthening of the face. The inferior maxillary bone increases in size in all directions. The cheeks are flattened and the zygoma stands out prominently. The eyes are dull and the patient has a sad expression. The extremities are greatly enlarged and also the trunk. The wrists are enlarged and thickened, and the feet are flat and enlarged. Kyphosis is present, as is also scoliosis.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

It has to be differentiated from myxedema. In the latter condition there is no enlargement of the bones, the face is round, skin pale, waxy and shiny, and the fingers clubbed at the ends.

From elephantiasis it may be differentiated by its absence of thickening and induration of the cutaneous tissue and of wasting of the muscles.

WHAT IS THE PROGNOSIS?

Acromegaly is incurable, but it may be sometimes arrested by treating the disease symptomatically.

WHAT IS THE TREATMENT?

GENERAL.—The patient's strength should be kept up by good nutritious food, and he should exert himself as little as possible while in a weakened condition.

REMEDIAL.—The remedies will have to be prescribed for the general symptoms, such as frontal headache, general

weakness, pains in the extremities and paresthesia, and for the mental condition. *Belladonna*, *Baryta carbonica*, *Calcarea carbonica*, *Phosphorus*, *Silica*, *Selenium* and *Sulphur*.

ERYTHROMELALGIA.

WHAT IS ERYTHROMELALGIA?

It is a disease mainly affecting the feet, and characterized by burning pains and congestion of the parts, with redness and swelling.

WHAT ARE ITS CAUSES?

It occurs usually in men and during middle life. It may follow severe physical exertion while on the feet. It may also follow long-continued low fevers when the physical condition is below par.

WHAT IS THE PATHOLOGICAL ANATOMY?

There is sometimes a plantar neuritis, and occasionally disease of the spinal cord.

WHAT ARE THE SYMPTOMS?

There are congestion and swelling of the feet, with burning pain. Sometimes the parts become intensely red. Usually both feet are involved, though one alone may be affected. The flushing of the painful part is the most characteristic symptom. The veins stand out as if a cord were tied around the leg. There is profuse perspiration of the parts. In severe cases the limbs become cold and pale when the patient is at rest. The feet become so painful and tender that standing or walking is impossible. The hands may be effected in some cases.

WHAT IS THE DIFFERENTIAL DIAGNOSIS?

The diagnosis must be made from pedalgia and alcoholic neuritis.

In pedalgia there is no swelling or redness. In alcoholic neuritis there is no redness.

WHAT IS THE TREATMENT?

GENERAL.—The patient should keep his feet elevated as much as possible, and perhaps remain in bed for a period of time.

REMEDIAL.—*Belladonna.*—Feet hot, swollen, burning, throbbing and painful; worse when putting the foot down; tension in soles of the feet; heaviness and tired feeling in the limbs.

Causticum.—Feet hot, tense and swollen, with perspiration; foot feels contracted, with tension on putting it to the ground; feet go to sleep.

Nitric acid.—Swelling and pain in the feet with itching; they inflame from the slightest degree of cold; great redness of the parts with intense heat; paralytic pain in the leg with excessive heaviness and lassitude.

MORTON'S NEURALGIA—METATARSAL NEURALGIA.

WHAT IS MORTON'S NEURALGIA?

It is a pain located at the back of the fourth toe, which usually extends up the leg. It is dull, throbbing, lancinating in character and usually comes on in spasms. The pain may be so severe at times that it prevents walking for a few minutes. It is increased by lateral pressure on the foot. It is supposed to be caused by squeezing the foot in too small a shoe, which produces a bruising of the nerve by the fifth bone. It is most common in women. Ordinary cases are relieved by changing the shoe so that it does not press upon the bone.

TARSALGIA—POLICEMAN'S DISEASE.

WHAT IS TARSALGIA?

It is a neuralgic affection generally due to flattening of the feet and stretching of the plantar ligaments; or to a contusion of the covering of the os calcis. It occurs in persons who are on their feet a great deal when not being accustomed to it. Raw recruits suffer frequently from this condition when going on long marches.

The treatment consists in resting and being off the feet as much as possible.

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