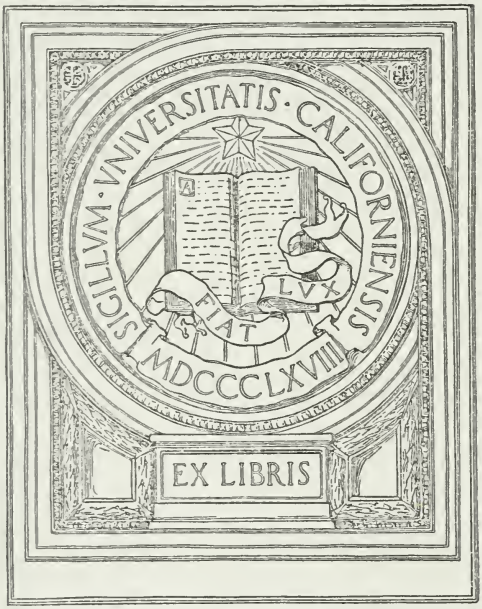

MANUAL

OF

NERVOUS DISEASES

BY

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PREFACE

TO THE SECOND EDITION

ALTHOUGH the second edition of this MANUAL has been thoroughly re-written and enlarged, yet the author has not departed from his original design to give a concise description of the essential facts in relation to the study and treatment of nervous diseases. It is hoped that the additions to the preliminary chapters on the anatomy of the nervous system and the general symptomatology and therapeutics of nervous diseases will be found acceptable. Experience in teaching has induced the author to adopt the arrangement of the subject matter followed in this MANUAL.

A. B. ARNOLD.

SAN FRANCISCO, 1890.

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CHAPTER I.

OUTLINES OF THE ANATOMY OF THE NERVOUS SYSTEM.

In the following Sketch of the Anatomy of the Nervous System it is intended to give prominence to those parts that are of special importance in relation to Neuropathology.

FISSURES AND CONVOLUTIONS OF THE LATERAL SURFACE OF THE HEMISPHERE.

Each hemisphere is marked by furrows running in different directions, apparently without any fixed order, but on comparing the brains of the higher mammalia fissures or sulci analogous to those in the human subject are recognized that follow a typical arrangement. Fissures divide the surface of the hemisphere into convolutions, and serve to mark the boundaries of the cerebral lobes.

The Fissure of Sylvius. This fissure is already visible in the third month of embryonic life. It commences on the under surface of the hemisphere and divides into two branches—a short vertical branch and a long horizontal branch. The bifurcation of the two branches forms a fossa, which lodges the island of Reil.

The Central Fissure or the Fissure of Rolando. This fissure runs in nearly a vertical direction from the upper margin of the hemisphere toward the posterior branch of the fissure of Sylvius.

The Parieto-Occipital Fissure. The lateral portion of this fissure is short, and sometimes not recognizable. Its median portion is long and deep, and separates the parietal from the occipital lobe.

The Interparietal Fissure. This fissure begins at a short distance behind the central fissure, and runs in a horizontal direction toward the occipital lobe.

The Paracentral Fissure runs in front and parallel to the central fissure.

The Superior and Inferior Frontal Sulci divide the convolutions in front of the paracentral fissure.

The termination of the *Calloso-Marginal Fissure* is represented by a notch in the superior aspect of the hemisphere between the central and interparietal fissures.

The sulcus occipitalis transversus and *longitudinalis inferior* divide convolutions on the lateral aspect of the occipital lobe.

The first and second temporal fissures divide the respective convolutions of the tempero-sphenoidal lobe on the lateral aspect of this lobe.

The Frontal Lobe and Its Convolutions.

The frontal lobe is bounded on its superior aspect by the longitudinal fissure, and forms the frontal extremity of the hemisphere. Its posterior side borders on the fissure of Rolando, and is separated inferiorly from the temporal lobe by the short branch of the Sylvian fissure.

The Ascending Frontal Convolution. The convolution running upward in front of the fissure of Rolando and uniting at both ends with the convolution at the opposite side of this fissure is named the ascending or anterior frontal convolution.

The First or Superior Frontal Convolution. This convolution begins at the upper extremity of the ascending frontal, where it is bounded by the longitudinal fissure. It turns downward and backward, and forms a part of the median and inferior surface of the hemisphere.

The Second or Middle Frontal Convolution. This convolution is separated from the foregoing by the superior frontal fissure, and from the anterior ascending convolution by the paracentral fissure. It extends forward to the extremity of the frontal lobe, and, bending downward, forms a part of the under surface of the hemisphere.

The Third or Inferior Frontal Convolution. This convolution is a prolongation of the lower portion of the ascending frontal. It winds around the vertical branch of the Sylvian fissure, and, running forward, forms the lateral aspect of the frontal lobe. The last three frontal convolutions constitute the so-called *prefrontal lobe.*

Convolutions of the Parietal Lobe.

The longitudinal fissure forms the upper boundary of the parietal lobe. It is separated from the frontal lobe by the fissure of Rolando, from the temporal lobe by the fissure of Sylvius, and from the occipital lobe by the parieto-occipital fissure.

The Ascending Parietal or Posterior Central Convolution

borders on the fissure of Rolando and runs parallel with the opposite ascending frontal convolution. These two convolutions, which form the *Rolandic region* of the hemisphere, are also spoken of as the *Central Convolutions.*

The Upper or First Parietal Convolution is the longitudinal continuation of the ascending parietal, and has the interparietal fissure for its inferior boundary. This convolution consists of a number of secondary folds, and is often mentioned as the *Parietal Lobule.*

The Second Parietal Convolution is situated below the interparietal fissure. That portion of it which is a continuation of the ascending parietal curves around the end of the posterior branch of the Sylvian fissure, where it bends downward to unite with the temporal lobe. This portion is also called the *Supramarginal Convolution.*

A fold of the same convolution, which, bordering upon the interparietal fissure below, makes an angular bend downward and then runs forward to unite with the second temporal convolution, is called the *Angular Gyrus.*

The Temporal Lobe.

The temporal lobe is separated by the fissure of Sylvius from the whole of the frontal lobe. Its inferior portion fills the sphenoidal fossa, and it is for this reason often named the *Tempero-Sphenoidal Lobe.*

The First Temporal Convolution runs parallel with the fissure of Sylvius. Its posterior portion, directly below this fissure, is continuous with the supramarginal convolution.

The Second Temporal Convolution is a continuation of the angular gyrus, and unites with the occipital lobe.

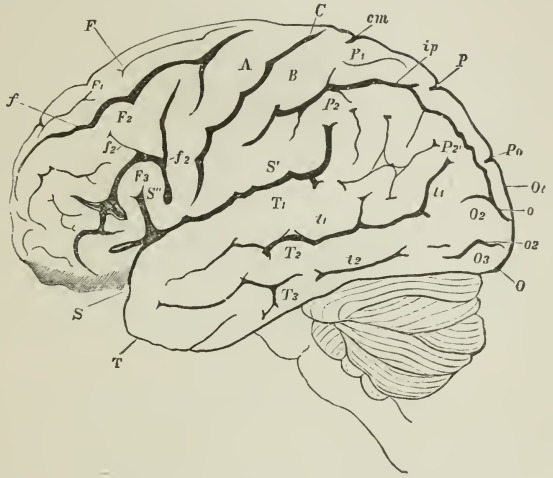

Fig. 1.—Side View of the Human Brain. (Ecker.)

F Frontal lobe.
P Parietal lobe.
O Occipital lobe.
T Temporal lobe.
S Fissure of Sylvius.
S' Horizontal.
S'' Ascending branch.
C Central fissure (Fissure of Rolando.)
A Ascending frontal convolution.
B Ascending parietal convolution.
F_1, F_2, F_3, Superior, middle, inferior frontal convolution.
f_1, f_2, f_3, Superior, inferior, vertical frontal fissure (precentral *f*).
P_1 Superior parietal convolution.
P_2 Inferior parietal convolution (Gyrus supramarginalis).
P_2' Gyrus angularis.
ip Interparietal sulcus.
cm Extremity of the Sulcus calloso marginalis.
O_1, O_2, O_3, First, second, third Occipital convolution.
Po Fissura parieto-occipitalis.
o Transverse occipital fissure.
o_2 Sulcus occipitalis longitudinalis inferior.
T_1, T_2, T_3, First, second, third temporal convolution.
t_1, t_2, First, second temporal fissure.

The Third Temporal Convolution forms the inferior

portion of the temporal lobe. Like the foregoing, it runs backward and connects with the occipital lobe.

The Occipital Lobe.

This lobe forms the posterior part of the hemisphere. It is separated from the parietal lobe at its extremity by the parieto-occipital fissure. There is no other visible division between these lobes on the lateral aspect of the hemisphere. The occipital lobe is divided by short and indistinct folds, which radiate upward and forward where they unite with the parietal and temporal convolutions.

The first occipital convolution borders above on the longitudinal fissure, and joins the superior parietal convolution.

The second or middle occipital convolution joins the angular gyrus.

The *Third or Inferior Occipital Convolution* is continuous with the third temporal convolution. The two lower folds of the Occipital lobe are separated by the *transverse fissure* and unite at the extremity of the lobe.

Fissures and Convolutions of the Median Surface of the Hemisphere.

Section of the corpus callosum in the line of the longitudinal fissure exposes the median surfaces of the hemispheres.

The calloso-marginal fissure runs parallel with the corpus callosum. It incloses a part of the way the *gyrus fornicatus.* This convolution begins by a narrow fold beneath the corpus callosum, curves around the whole length of this commissure, unites with the occipital lobe and then

bending downwards and forwards it forms in that region the *gyrus hippocampi*. The hook-like extremity of this convolution is called the *uncus* (unciatus or unciform process).

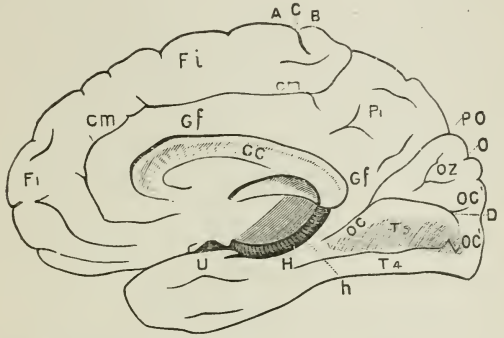

FIG. 2.—MEDIAN ASPECT OF THE HEMISPHERE. (AFTER ECKER.)

C, *C* Corpus callosum.
G, *F* Gyrus fornicatus.
H Gyrus hippocampi.
h Sulcus hippocampi.
U Gyrus unciatus.
C, *M* Sulcus calloso-marginalis.
F Median aspect of the first frontal convolution.
C Extremity of the central fissure.
A Frontal and B posterior central convolution. (These inner portions of the central convolutions constitute the paracentral lobule.)
P Precunial convolution (lobus quadratus).
O, *Z* Cuneus.
po Parieto-occipital fissure.
O Occipital transverse fissure.
O, *C* Fissura calcaria.
O, C_1 Superior and *O*, C_2 inferior limb of the calcarian fissure.
T_4 Lobulus fusiformis. (Gyrus occipito-temporalis.)
T_5 Lobulus lingualis (Gyrus occipito-medialis.

That part of the median surface of the hemisphere which lies above the fissura calloso-marginalis, corresponds with the superior inner portions of the two central convolutions, and is called the *paracentral lobule*.

The lobus quadratus or precunial convolution is that part of the median surface of the hemisphere which is situated between the ascending portion of the fissura calloso-marginalis and the parieto-occipital fissure.

The calcarian fissure begins at the posterior extremity of the occipital lobe, and deeply penetrates it in a vertico-transverse direction. Below the gyrus fornicatus it crosses the parieto-occipital fissure at an acute angle, by which a triangular portion of the occipital lobe is inclosed. This part of the lobe is called the *cuneus* or cuneiform convolution.

The lobulus lingualis is a longitudinal fold on the median aspect of the temporal lobe. It unites by a narrow strip with the gyrus hippocampi.

The lobulus fusiformis is separated from the former by a curvelinear fissure. It forms the inferior border of the temporal lobe, and runs backwards to join the occipital lobe.

THE INFERIOR SURFACE OF THE BRAIN.

The frontal inferior surface of the hemisphere consists of portions of the first, second and third convolutions. The first frontal convolution in this region is represented by a very small fold called the *gyrus rectus*. It is separated from the under surface of the second convolution by the *olfactory fissure*. The *sulcus orbitalis* divides the latter from the inferior portion of the third frontal convolution, which rests on the orbital plate. It curves outward and upward and forms the lateral aspect of the frontal lobe.

The gyrus hippocampi forms the central under-surface of the hemisphere. A part of this region is also occupied by portions of the lingualis and fusiform lobules. The rest of the inferior surface of the hemisphere behind the

fissure of Sylvius belongs partly to the temporal and partly to the occipital lobes.

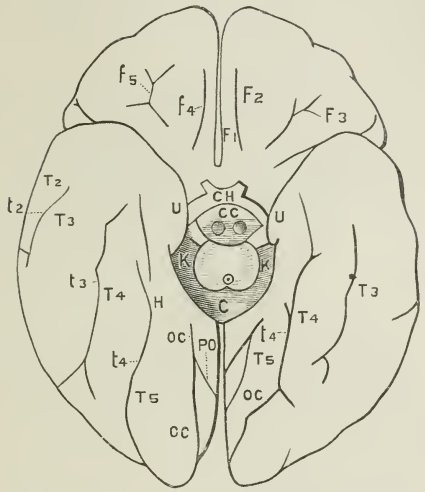

FIG. 3.—INFERIOR SURFACE OF THE HEMISPHERE. (AFTER ECKER.)

F_1 Gyrus rectus.
F_2 second and F_3 third frontal convolutions.
F_4 Sulcus olfactorius.
F_5 Sulcus orbitalis.
T_2 second or middle.
T_3 Third or inferior temporal convolution.
T_4 Lobulus fusiformis (Gyrus occipito temperalis lateralis.)
T_5 Lobulus lingualis (Gyrus occipito-temporalis medialis.
t_4 Sulcus occipito-temporalis inferior.
T_3 inferior and T_2 middle temporal fissure.
po Parieto-occipital fissure.
oc Fissura calcarina.
H Gyrus hippocampi.
U Gyrus unciatus.
Ch Chiasm.
C, *C* Corpora candicantia.
K, *K* Crus cerebri.
C Corpus callosum.

THE CORTEX.

Histologists distinguish five layers of nerve cells in the cortex. The cells are of different shapes and sizes. A

large number of them consist of "pyramidal," or "giant cells;" others are oval or irregular. They give off "branches" that connect them with each other. A fine connective tissue, called the *neuroglia* surrounds the nerve elements like a cement.

Physiology assigns to the cortex of the hemispheres the seat of *mental activity.* The following considerations point to the intimate relation existing between the cortex and the manifestations of the psychical powers: 1. Intellectual capacity is proportionate to the development of the hemispheres. This is shown by the great number and complexity of the convolutions in man, which enlarge the area of the cortical substance. 2. Smallness or atrophy of the hemispheres is observed in idiotism. 3. Injury or disease of the hemispheres gives rise to cerebral excitement, confusion of ideas, stupor and coma.

The Excitable Cortical Areas.

In speaking of the "localization of the functions of the brain" it must be understood that in our present state of knowledge, this term mainly refers to the results of the highly interesting experiments of Fritsch, Hitzig, Ferrier and their followers. When according to these experiments certain limited areas of the cortex of the brain are excited by the electric current, uniform movements are made by the animal; and conversely, when such areas are extirpated, these movements cannot be evoked, at least for a considerable length of time afterwards. Thus, the animal moves a foreleg, a hindleg, or the eye, whenever the electric stimulus excites a definite cortical area.

Ferrier experimented on the monkey. The accompanying figure gives the topography of the surface of a monkey brain of an inferior species.

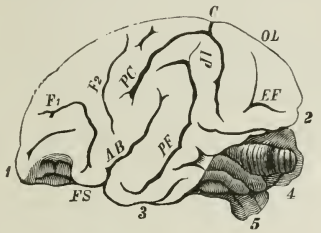

Fig. 4.—The Monkey Brain. (Meynert.)

(*Cercocebus cinomolgus.*)

1 Inferior extremity of the forehead.
2 Inferior extremity of the occiput.
3 Inferior extremity of the temple.
4 Cerebellum.
5 Medulla oblongata.
FS Fissure of Sylvius.
AB Posterior, or ascending branch.
C Central fissure (Fissure of Rolando).
F_1 Inferior frontal fissure.
F_2 Superior frontal fissure.
PC Paracentral fissure.
IP Interparietal fissure.
PF Parallel fissure.
EF External occipital fissure (monkey fissure).
OL Occipital lobe.

Ferrier mapped the cortex of the human brain into areas that correspond with the excitable areas of the monkey brain. From his figures it appears that the principal ' motor zone" is located in the region of the central convolutions (Rolandic region). It is a significant fact that cells of a large size, resembling those of the anterior cornua of the spinal cord abound, in that region. Sufficient pathological evidence has now accumulated in support of the existence of excitable motor areas in the regions indicated by the experiments.

PSYCHO-MOTOR CENTERS.

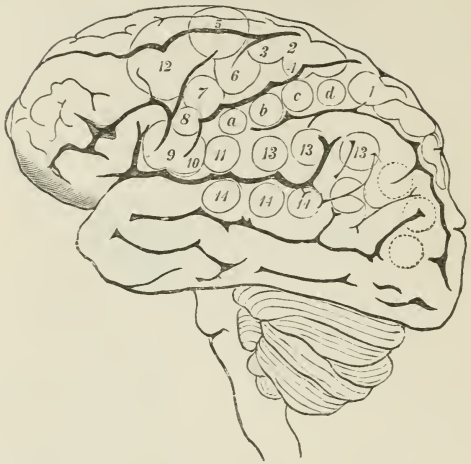

FIG. 5.—SIDE AND UPPER VIEW OF THE BRAIN OF MAN.
(*According to Ferrier.*)

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

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

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

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

6 On the upper part of the antero parietal or ascending frontal convolution (anterior central). Supination and flexion of the opposite forearm.

7 On the median portion of the same convolution. Retraction and elevation of the opposite angle of the mouth by means of the zygomatic muscles.

8 Lower down on the same convolution. Elevation of the ala nasiand upper lip.

9, 10 At the inferior extremity of the same convolution. Opening of the mouth with protrusion (9) and (10) retraction of the tongue. *Region of Aphasia.* Bilateral action.

11 Between (10) and (11) and the inferior extremity of the posterio-parietal convolution. Retraction of the opposite angle of the mouth; the head turned slightly to one side.

12 On the posterior portion of the superior and middle frontal convolution. The eyes open widely, the pupils dilate, and the head and eyes are turned toward the opposite side.

13, 13′ On the supra-marginal lobule and angular gyrus. The eyes move toward the opposite side with an upward (13) or downward (13′) deviation. The pupils generally contracted. Center of vision.

14 On the infra-marginal or superior (first) tempero-sphenoidal convolution. Pricking of the opposite ear; the head and eyes turn to the opposite side and the pupils dilate largely. Center of hearing.

Theories of the Motor Phenomena.

It should be premised in connection with the theories explanatory of the phenomena observed in the experiments, that the intervention of the cortical substance is not necessary, as the movements also occur when the excitable areas are denuded of their gray covering. The latter serves as the medium of voluntary impulses. The question arises: What is the nature of the functional defect, when, on the extirpation of certain circumscribed areas of the cortex, the movements cease to be elicited by the electric stimulation.

Hitzig attributes the defect to the loss of the muscular sense: the animal being in consequence unable to co-ordinate the movements of corresponding groups of muscles.

Ferrier ascribes the defect to a psychical inability to initiate voluntary movements.

According to Munk, there is an incapacity to recall the representations of co-ordinate movements which the animal had learned by experience.

It may be of interest to summarize the conclusions which Munk has drawn from his numerous and well-conducted experiments. He localizes the excitable areas of the cortex as follows:

1. *The region of the leg.* The upper third of the central convolutions and the upper adjacent portion of the parietal lobule.

2. *The region of the arm.* The middle third of the central convolutions and lower portion of the parietal lobule.

3. *The region of the head.* The lower third of the central convolutions and a contiguous portion of the frontal convolution bordering on the fissure of Sylvius.

4. *The center of vision.* The surface of the occipital lobe.

5. *The center of hearing.* The temporal lobe.

6. *The ocular region.* The gyrus angularis.

7. *The region of the ear.* The marginal convolution.

8. *The region of the nape of the neck.* The part of the frontal lobe adjacent to the middle third of the frontal ascending convolution.

9. *The region of the trunk.* The convex surface of the frontal lobe bordering on the precentral fissure.

The results of the recent investigations of Luciani, Horsley and Shaefer agree in the main with those of Munk.

Ferrier, since the first publication of his celebrated

experiments, has somewhat modified his views in regard to the so-called psycho-motor centers. There is now a tendency among neurologists to consider the excitable cortical areas in the light of sensory-motor centers as will be inferred from the following summary of Munk's late contributions. To the cortical zone of the Rolandic region he assigns the function of elaborating perceptive images of the tactile and muscular sensations, that are constantly conveyed to the intelligence. The feelings attending the action of muscles, tendons and ligaments in voluntary and reflex movements are registered in memory as representatives of special movements. When these mnemonic images, which consist of components of the registered sensations are destroyed by the method of extirpation, the corresponding movements can no longer be innervated by voluntary impulse or artificially excited. Decortation of the visual center produces "psychical blindness." The animal sees perfectly well, and avoids objects that are put in his way, but does not recognize the food set before him. He has lost the power of associating the perception of the food with the object of his sight. When the auditory center is destroyed the animal is unable to recall auditory images although he hears the sounds well enough. This is "psychical deafness." The animal does not recognize the voice of his master, although it hears the sounds.

Systems of Nerve-Fibres of the Brain.

Three main divisions of nerve fibres may be distinguished in the white substance of the brain.

1. *The association system* of fibres connect contiguous and remote convolutions of the hemisphere. They are arched, and present their concave side to the surface of the brain.

2. *The commissural system* of fibres connect symmetrical parts of the hemispheres. They present in their course the shape of the letter U. These fibres constitute the corpus calossum.

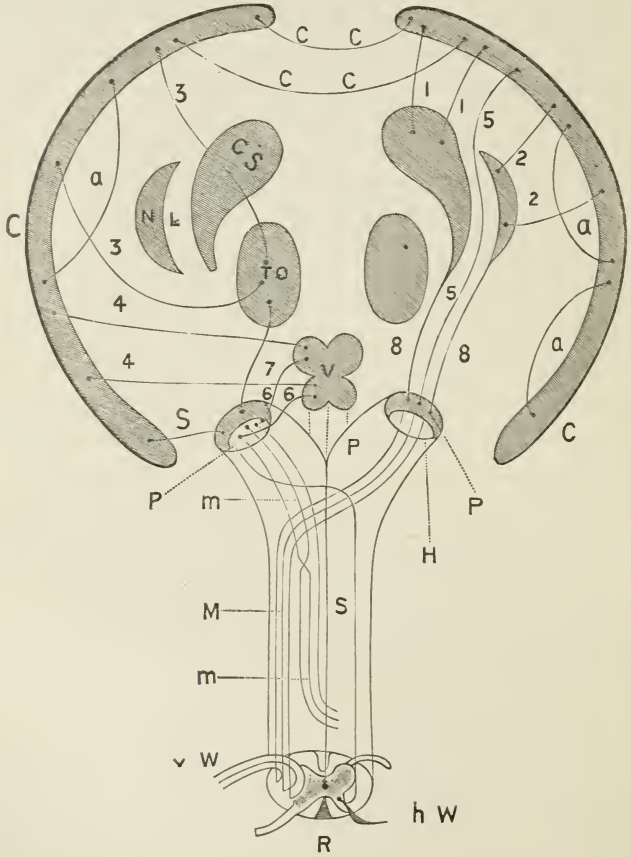

FIG. 7. SCHEMA OF THE CEREBRO-SPINAL SYSTEM OF NERVES. (LANDOIS.)

C, C	Cortex of the brain.
Cs	Corpus striatum.
N L	Lenticular nucleus.
T O	Optic thalamus.
V	Corpora quadrigemina.
P	Crura cerebri.
H	Tegementum.
p	Crusta.
1, 1	Radiate fibres of the corpus striatum.
2, 2	Those of the lenticular nucleus.
3, 3	Those of the optic thalamus.
4, 4	Those of the corpora quadrigemina and tegementum.
5, 5	The pyramidal tract.
6, 6	Fibres connecting the corpus quadrigemina and tegementum.
m	Their further course.
8, 8	Fibres connecting the corpus striatum and lenticular nucleus with the crusta.
m	Their further course.
S, S	Course of sensory fibres.
R	Transverse section of th spinal cord.
V, W	Anterior and r, W, posterior roots of nerves.
a, a	Associating fibres.
c, c	Commissural fibres.

3. *The system of radiating fibres* (corona radiata, peduncular fibres). These nerve fibres come from the hemispheres, and converge at the base of the brain, where they go to form the internal capsule.

The Internal Capsule.

The connection of the medullary substance of the hemisphere with the deep-seated parts of the brain, and finally with the spinal cord, is effected by the convergence of parts of its constituent fibres toward the large ganglia at the base of the brain, where they present a white strand of fibres called the *internal capsule*. That part of the capsule in front of the head of the caudate nucleus and behind the lenticular nucleus, constitutes the *anterior limb* of the internal capsule. As the capsule passes along the internal margin of the lenticular nucleus, it makes a bend (genu) and runs between the

front of the optic thalamus and behind the lenticular nucleus, where it forms the *posterior limb* of the internal capsule. The continuation of the tract of fibres of which the capsule consists, forms part of the peduncle of the brain; and passing on through the pons, and then to the medulla oblongata, it decussates in the anterior pyramids, from which circumstance it has been named the *pyramidal tract*. The fibres of this tract chiefly come from the two central convolutions. the paracentral lobule and the supra-marginal gyrus. In its downward course it forms a distinct fasciculus of the cord. and disappears after having connected with the successive motor cells in different regions of the cord.

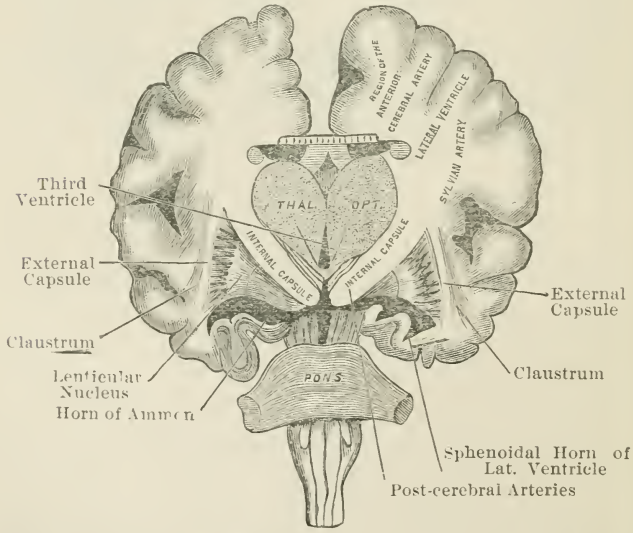

FIG. 8.—VERTICO-TRANSVERSE SECTION OF THE BRAIN, POSTERIOR TO THE TUBERCULA MAMMILLARIA, ANTERIOR TO THE PEDUNCLE. (CHARCOT.)

On comparing the number of fibres that come from the hemisphere and pass to the internal capsule, with the small size of the crus cerebri, the difference in mass is

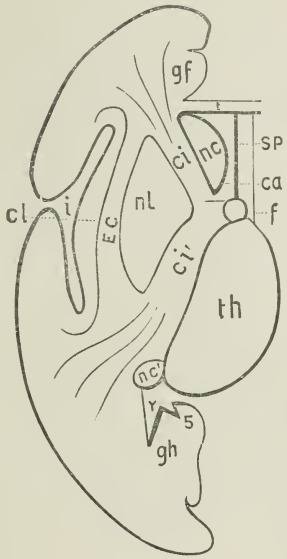

FIG. 9.—DIAGRAM OF A HORIZONTAL SECTION OF THE BRAIN BELOW THE LEVEL OF THE CORPUS CALLOSUM. (WERNIKE.)

I Island of Reil.
t h Thalamus optici.
n l Lenticular nucleus.
n c Head of the caudate nucleus.
N c' Extremity of the caudate nucleus.
C i Anterior limb of the internal capsule.
Ci' Posterior limb of the internal capsule.
G, f Gyrus fornicatus.
S P Anterior horn of the lateral ventricle.
T Corpus Calossum.
E C External capsule.
C l Claustrum.
V Ventricle.
G h Gyrus hippocampi.

apparent. Some of those fibres in their passage downwards must therefore be interrupted, or lost in parts at the base of the brain. A portion of the fibres occupy-

ing the anterior limb of the internal capsule enter the corpus striatum. This basal ganglia, the optic thalamus and the corpora quadrigemina may be considered as "intermediary stations" where the entering nerve fibres, and those that proceed from them are modified in their function. The exact functions of these basal ganglia are however not well understood. The rest of the fibers of the anterior limb of the interior capsule constitute the pyramidal tract. It occupies the middle third of the foot of the crus cerebri. The outer third of the crus is composed of fibres that come from the corpus striatum, and its inner third consists of fibres that pass to the optic thalamus, the corpora quadrigemina and posterior part of the hemisphere.

According to the investigations of Meynert, there is an anatomical distinction between the inferior part of the crus cerebri, which he calls the *crusta*, and its upper part the *tegementum*. The fibres of the crusta consist of the pyramidal tract, and those of the tegementum consist of fibres that come from the spinal cord and pass to the mesocephalon.

Nerve-tracts of the Cerebellum.

The connection of the cerebellum with adjoining parts consists of fasciculi of afferent and efferent nerves that form the three peduncles of that organ. Although little has been accurately determined in regard to the functions of the cerebellum, its influence on the cerebrum, the spinal cord and intermediate nervous structures must be considerable as shown by the numerous nerve paths that connect it with these parts.

NERVE-NUCLEI AND NERVE-FIBRES OF THE MEDULLA.

The components of the complex structure of the medulla oblongata that merit particular attention, include

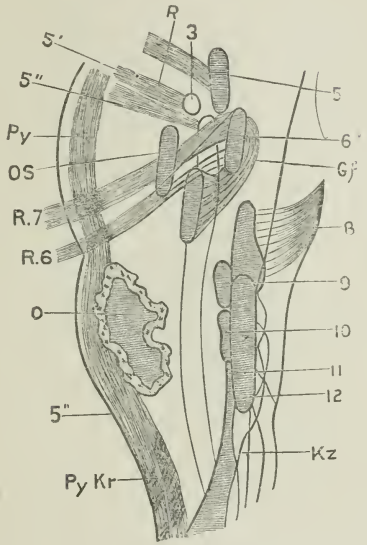

FIG. 10.—LATERAL VIEW OF THE RIGHT HALF OF THE MEDULLA, SEEN FROM THE SURFACE OF THE SECTION. (AFTER ERB.)

Py Pyramidal tract.
Py, Kr Decussation of pyramids.
O Olivary body.
Os Superior olivary body.
5 Motor,
5' Middle sensory,
5'' Inferior sensory nucleus of the trigeminus.
6 Nucleus of abducens.
G f Genu facialis.
7 Nucleus facialis.
8 Posterior median acoustic nucleus.
9 Glosso-pharyngeal nucleus.
10 Nucleus of vagus.
11 Spinal accessory nucleus.
12 Hypoglossal nucleus
Kz Nucleus of the funiculis gracilis.
R_5 Trigeminus roots, those of the R_6 Abducens and R_7 Facialis.

the collections of gray masses from which the roots of some of the cranial nerves on the floor of the fourth

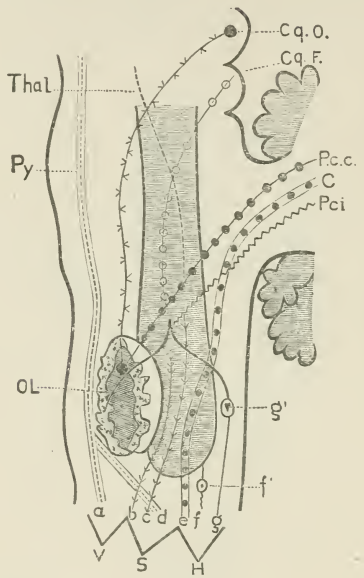

FIG. 11.—DIAGRAM OF THE CHIEF TRACTS OF THE MEDULLA. (AFTER ERB.)

Ol Olivary body.

V Anterior,

S Lateral and H Posterior spinal funiculi.

a Pyramido-anterior tract.

d Pyramido-lateral tract.

Py Pyramidal tract.

b Remainder of anterior column.

c Remainder of lateral column.

e, e Cerebello-lateraltract.

f Funiculis gracilis, and f′ nucleus of the same.

g Funiculus cuneatus, and g′ nucleus of the same.

P, c i Internal fasciculus of the peduncle cerebelli.

P, c, e external fasciculus of the same.

Cq, F Tract from corpus quad. to formato retic.

Cq, O The same to olivary body.

Thl tract from the thalamus opticus. The formatis reticularis is presented by shading in the center of the figure.

ventricle arise, and also the different nerve tracts in their relationship to the spinal cord. The annexed diagrams are intended to show the positions of the nuclei and tracts.

Cerebro-Spinal Conducting Paths.

At the lower end of the medulla, the larger portion of the pyramidal tract crosses and enters the lateral columns of the spinal cord. This part of the tract is called *the crossed pyramidal tract*. A small portion which does not cross borders upon the anterior fissure of the cord, and is called *the direct pyramidal tract* (Türk's column). Both the crossed and the direct pyramidal tracts undergo secondary descending degeneration in lesions, affecting the internal capsule. The *direct cerebellar tract* consists of fibres at the periphery of the lateral column. In its upward passage it lies on the surface of the restiform body and enters the inferior peduncle of the cerebellum. This cerebello-spinal conducting path is subject to secondary ascending degeneration. *Goll's column* borders upon the median fissure of the posterior spinal column. In passing upwards from the lumbar region of the cord it enters the medulla and connects by arcuate fibres with the inferior peduncle of the cerebellum. Loss of sensibility has not been noticed in disease of Goll's column. The outer portion of the posterior column of the spinal cord is known as *Burdach's column*. It consists of fibres that enter the cord by the posterior nerve roots. This column contains a sensory conducting tract, and joins the fibres which constitute the posterior third of the hinder limb of

the internal capsule. This region of the capsule is in the immediate vicinity of what is called the "sensory crossway," where fibres of special and common sensation meet. For the more precise information in regard to some of these distinct nerve paths, we are indebted to the admirable investigations of Flechsig. Türk had previously made the observation that lesions affecting certain parts of the brain stood in relation to secondary degeneration of definite tracts of nerve fibres. Flechsig subsequently discovered that these and other bundles of nerve fibres in the brain and spinal cord acquire their medullary sheaths at different stages of embryonic development. By this method of research he succeeded in determining the existence of distinct cerebro-spinal tracts of nerve fibres.

Additional structural peculiarities of the spinal cord remain to be mentioned. The regions of the antero-lateral depression of the cord where the anterior nerve roots are situated are called the *anterior root zones*. The *posterior root zones* are in the regions of the postero-lateral groove of the cord where the posterior nerve roots originate. The gray matter at the tips of the posterior cornua is called *the gelatinous substance of Rolando*. Near and along the junction of the anterior and posterior cornua a peculiar vesicular appearance of the central gray substance of the cord is observed which bears the name

of *Clarke's column.* The *formatio reticularis* is an interlacement of fibres between the anterior root zones.

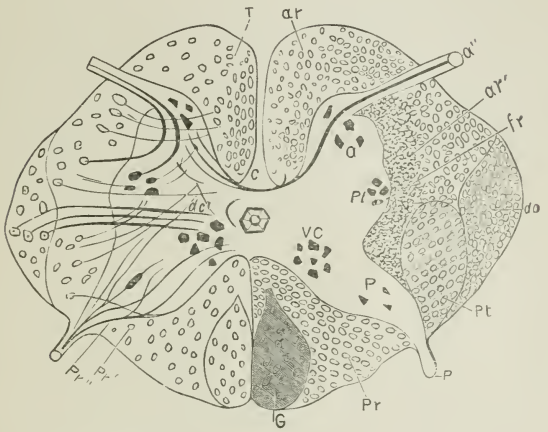

FIG. 12. TRANSVERSE SECTION OF THE SPINAL CORD IN THE UPPER HALF OF THE DORSAL REGION. (AFTER FLECHSIG.)

C, Anterior commissure.
d c' Fibres which pass from the vesicular column of Clarke (v c) to the direct cerebellar tract.
a, Anterior grey horn.
P, Posterior grey horn.
P t, Pyramidal tract of the lateral column.
T, Column of Türk.
d c, Direct cerebellar tract.
G, Goll's column.
a r, Internal portion of the Anterior root-zone.
a r', External portion of the Anterior root-zone.
v c, Columns of Clarke.
f r, Reticular formation of the spinal cord.
d r, Burdach's column.
p r', p r'', Posterior root zones

SPINAL GANGLION CELLS

Nerve cells varying in size and number occupy different regions of the central gray matter of the spinal cord. They are especially numerous in the cervical and lumbar

segments. Groups of very large cells are found in the anterior cornua. Cells of smaller size and less in number are found in the gelatinous substance and the posterior nerves. Clarke's column contains but few cellular elements. All the cells in the cord are surrounded by a fine network of nerve fibres and interstitial tissue (neuroglia).

The spinal ganglion cells perform the function of motor, sensory and reflex centers. There is also strong evidence that the cells of the anterior nerves exert a trophic influence on the muscular organs with which they are connected.

Sensory Conducting Paths of the Spinal Cord.

Our knowledge in regard to distinct sensory tracts in the cord is still very imperfect. We only know that all sensory impressions enter the posterior nerve roots and are transmitted to the posterior cornua. The sensory fibres of the cord on their entrance immediately cross over to the opposite side.

Circulation of the Brain.

It is of pathological importance to consider briefly the peculiarities of the cerebral circulation. The brain receives its blood from two sources—from the vertebral arteries and the internal carotids. Each carotid divides into two large branches—the anterior cerebral and the middle cerebral arteries. It is the distribution of the latter which is of special interest. This artery enters the fissure of Sylvius and sends branches to the lenticular nucleus and adjacent parts of the brain, including portions of the caudate body of the corpus striatum, the optic thalamus and the internal capsule. Some of the branches are also distributed to the greater part of the

central convolutions, the supramarginal and angular convolutions, and partly to the inferior frontal convolution. Although the middle and anterior cerebral arteries have a common origin, yet their ramifications do not freely anastomose. They are terminal arteries. It is due to this circumstance that in occlusion of branches of the middle cerebral artery, collateral circulation often fails to be established. Hemorrhage in the *lenticular vascular district* is very common.

CHAPTER II.

GENERAL SYMPTOMATOLOGY OF DISEASES OF THE NERVOUS SYSTEM.

SENSORY DISTURBANCES.

1. *Hyperalgia.* Pain is the most common of all the varieties of morbid sensibility. The mind locates the seat of pain with great precision, when the source of irritation affects an external part of the body, but often fails to do so when internal parenchymatous organs are involved. It is well known that extensive and destructive disease of the brain, lung and liver does not give rise to pain if the enveloping membranes of these structures are not implicated. The character of the pain is often significant of the part which is affected. For example, neuralgic pain is paroxysmal, lancinating, darting, etc. The pain in inflammation of serous membranes is sharp, piercing, stabbing. The pain in irritation of mucous membranes resembles the sensation of smarting, burning or soreness. Muscular pain consists of an indescribable feeling of restlessness and fatigue. Pain in the joints is of a deep-seated, boring, gnawing nature.

2. *Hyperæsthesia* (hyperalgesia). Morbid increase of the cutaneous sensibility is recognized by the circumstance that in this condition of the nervous system ordi-

nary exciting causes give rise to unusually painful feelings and heighten the reflex excitability. Hyperæsthesia may be limited to circumscribed areas of the skin. General hyperæsthesia is probably due to an irritable condition of the spinal cord. It is a prominent symptom in tetanus, hydrophobia, spinal meningitis, strychnia poisoning and hysteria.

3. *Anæsthesia* (analgesia). All cases of anæsthesia must of course depend on interruption, either central or peripheral, of sensory conduction. Loss of the cutaneous sensibility does not constitute the only form of anæsthesia. The different qualities of common sensation may be wholly or separately abolished. Tactile sensibility is simply tested by touching the skin with a blunt instrument. The sensation of pain is conveniently determined by a prick of the skin with a needle. The experiment to ascertain whether a patient is able to localize an impression, or to tell whether the contact has been made in one or several places at the same time, may be made with the points of a compass while the patient has his eyes closed. The appreciation of differences in temperature is tested by the application of warm and cold substances to the skin. *Polyæsthesia* is a rare form of partial anæsthesia. The patient feels two, three or five impressions, though only one impact is made.

4. *Allochirea* denotes a perversion of sensibility, the patient being uncertain which side of the body is touched, although the cutaneous sensibility is normal.

5. *Analgia* means that morbid condition of common sensibility in which no pain can be elicited. It is observed in the hypnotic state and hysteria. A tardy

transmission of sensory impression may be the only sign of partial anæsthesia.

6. Impairment of the "*muscular sense*" is ascertained to exist when the patient is unable to appreciate palpable differences of weight. To exclude the tactile sensibility during the test the weights should be suspended from the limb. An easy way of determining the condition of the muscular sense of the upper extremities is to request the patient to touch the tip of his nose with a finger; if defective the finger will miss its mark. Muscular contractility is best determined by faradization.

7. *Paræsthesia.* Certain morbid sensations which patients compare to a feeling of tingling, creeping, numbness or formication are usually called paræsthetic. The sensation as if the limb were "asleep," or stuck with "pins and needles," flushes of heat in the face, or a feeling of chilliness in some part of the body relate to the same class of sensory disorders.

8. *Paralgia.* By this term is understood an anomalous sensation that partakes more of the feeling of constriction than pain. A distressing sensation of this character, which patients locate in the region of the heart, is often called (precordial anxiety). This symptom is frequently complained of by nervous and hysterical individuals. Tobacco smokers are also troubled with it. It occurs with greatest intensity in angina pectoris.

VASO-MOTOR DISTURBANCES.

Irregularities of the vaso-motor system of nerves are often associated with disease of nerve centers. Among the two varieties of vaso-motor nerves—the vaso-constrictors and the vaso-dilators—the former is of greater

pathological importance. Experiments have determined the existence of vaso-motor centers in the medulla oblongata and the spinal cord. The vaso-motor fibres pass into the lateral columns of the spinal cord, emerge from there with the anterior nerve roots, and collect in the trunk of the sympathetic nerve and plexus.

Irritation of the vaso-motor nerves causes constriction of the blood vessels. Paralysis of these nerves causes dilatation of the vessels.

The external effects of vaso-motor disturbance are chiefly seen in the skin. Spasmodic constriction of the blood vessels produces pallor or cyanosis of the skin. It sometime causes a sensation of creeping and coldnes of the hands, with which nervous and hysterical persons are often troubled. The more severe effects of vaso-motor spasm are seen in malignant decubitus—Reynaud's disease, circumscribed gangrene of the extremities and the eruption of dark bullæ in different parts of the skin.

Paralysis of vaso-motor nerves gives rise to redness of the skin and the sensation of heat in the affected part. This condition is often observed in hysteria and neurasthenia, and also occurs as an independent affection. Weir Mitchell has described an analogous condition as *erythromelalgia*. The redness and subjective feeling of heat in this condition may be persistent or occur at irregular periods. It is attended by palpitation of the heart, strong pulsation of the arteries, anxiety, ringing in the ears and sweating. A milder affection of this kind consists of a red or mottled appearance of the palms of the hands and soles of the feet, a distressing sensation of heat in these parts, slight swelling and profuse perspiration.

Trophic disturbances. Certain nutritive changes are now supposed to be caused by an independent set of trophic nerves. Examples of such trophic disorders are the "glossy skin," the falling out of the hair, stunted nails and perforating ulcer of the foot.

Motor Disturbances

The subjects under this head include the two important groups of convulsive and paralytic affections.

convulsion, spasm. (hyperkinesis).

Convulsion denotes an involuntary muscular contraction. The synonymous term spasm is often used to express convulsive movements of a single muscle or a group of muscles. We speak of general convulsions when the head, the trunk and the extremities are simultaneously affected, as in epilepsy. A difference in the character of convulsive movements underlies the distinction of *clonic* and *tonic* spasm. In clonic spasm there is alternate contraction and relaxation of a muscle. Tonic spasm is distinguished by the rapid recurrence of the contractions, so that the affected muscle appears to be in a fixed condition. The muscle in spasm is always shortened. *Cramp* means a painful contraction. All forms of convulsive movements are caused by motor irritation in some part of the nervous system.

Contracture signifies the persistent contraction of a muscle, which fixes the limb either in a flexed or an extended position.

Tremor consists of very slight contractions of numerous bundles of muscular fibres. The movements in tremor are of a very limited sweep.

Fibrillary contractions consist of a waving motion of small fasciculi of muscular fibres.

Paralysis (Akinesis).

Any lesion or morbid influence which interrupts the conduction of motor impulses causes paralysis. Immobility of muscles from any other cause is not paralysis. The term *paresis* is used to signify incomplete paralysis.

The classification of paralysis, viewed anatomically, includes 1, cerebral; 2, spinal and 3 peripheral paralysis. The division of the different forms of paralysis in accordance with their area of distribution and combination with special morbid conditions, is of great clinical importance, and will be briefly considered in this place.

1. *Hemiplegia.* This form of paralysis is typical of cerebral disease. It occurs when any part of the motor tract above the spinal cord is interrupted. The paralysis affects the lateral half of the body, namely, the face, and the upper and lower extremity of the side opposite to that of the lesion in the cerebrum. This distribution of the paralysis necessarily results from the crossing of the motor tract in the anterior pyramids.

2. *Alternate or crossed paralysis* is a form of hemiplegia in which the arm and leg of the side of the body opposite to that of the lesion in the brain is paralyzed, while the face or an ocular muscle of the same side as the lesion is also involved.

3. *Monoplegia.* This is a form of cerebral paralysis which either involves one side of the face, or one arm, or one leg; or simultaneously, the face and tongue, or one side of the face and one arm. Such a partial hemiplegia occurs when a cerebral lesion affects a part of the motor tract in the brain, where its fibres are yet too divergent to become collectively involved.

4. *Paraplegia* is typical of tranverse disease of the spinal cord. The paralysis affects symmetrical parts of the body; most frequently the two inferior extremities.

5. *Atrophic paralysis.* The loss of motor power in this form of paralysis is associated with atrophy of the affected muscles. When the lesion is situated above the spinal cord there is hemiplegia, but rarely atrophy. Atrophic paralysis in central lesion of the nervous system occurs in disease of the ganglion cells of the anterior cornua of the spinal cord.

It has been inferred from development of atrophic paralysis in disease of the anterior cornua, that its ganglion cells exert an influence on the nutrition of the corresponding muscles with which they are connected.

6. *Spastic paralysis.* This form of paralysis is distinguished by a rigid condition of the affected muscles. The muscles are not atrophied, and offer resistance to passive motion. There is usually exaggeration of the tendon reflexes. The cause of the rigidity of the muscles is either a direct central irritation or reflex action.

7. *Functional paralysis.* By this term is understood a group of paralyses, whose pathology is obscure. It includes, reflex, hysterical and alcoholic paralysis, and also those paralytic affections that occur as sequellæ to infectious fevers.

Besides the essential points of distinction between the different forms of paralyses, there are certain accessory symptoms which assist differential diagnosis. These refer to sensory and vaso-motor disturbances, and alteration of reflex and electric reaction which will be described in their proper places.

The following explanation has been proffered to account for the exemption of certain groups of muscles, which for anatomical reasons would be expected to become also involved in hemiplegia. The muscles of the neck, the tongue, the eyes and the trunk are rarely found paralyzed. Those of the jaws, the pharynx and larynx always escape. This exemption or relative immunity is due to the circumstance that all these muscles act in pairs, from which it may be inferred that they are innervated by both sides of the brain.

The recognition of hemiplegia when the patient is unconscious is not always an easy matter. The following hints will assist the diagnosis. On observing the appearance of the face, it will be noticed that the cheek "hangs," that it bulges during expiration and flaps during inspiration. The mouth is drawn to one side and often saliva escapes from the half-opened lips. On raising a paralyzed arm and then suddenly releasing it the limb falls down like a dead weight. Sometimes the head and eyes are turned to one side. *Hemiparesis* is recognized with greater difficulty, even when the consciousness of the patient is clear. Much information may be gained by noting the position of the limbs and the range of their voluntary movements. The grasp of one of the hands is feeble in comparison with that of the other. One corner of the mouth is slightly drawn when the patient laughs or cries. In the act of walking his body leans towards one side. In place of finding the paralyzed limb in a flaccid condition and passively movable in every possible direction, which is usually the case, it sometimes happens that the arm is stiff and tightly pressed against the chest, and the hands and fingers are forcibly flexed.

Occasionally the leg maintains an extended position. This "early rigidity" is characteristic of hemiplegia, but it soon disappears.

Hemianæsthesia. This is a far less common symptom in brain lesions than hemiplegia. The special senses in severe cases may be more or less simultaneously affected. In complete loss of the cutaneous sensibility, there is often obtuseness of the contiguous mucous membranes. The lesion in central hemianæsthesia is located either in the posterior or inferior third of the internal capsule, or in the vicinity of the optic thalamus or lenticular nucleus.

Cephalic Symptoms.

This group of symptons includes headache, vertigo, delirium, insomnia, somnolence, stupor and coma. Occasionally the following adventitious symptoms are quite prominent in brain disease: gastric derangement, nausea and vomiting, disorder of the special senses and epileptiform convulsions.

1. *Headache* is a symptom attending a great variety of morbid conditions, but is usually of little diagnostic importance. Very violent and persistent pain in the head is, however, an early and constant symptom of meningitis, cerebral tumor, cerebral syphilis and cerebral abscess. Infants manifest severe headache by a peculiar abrupt cry, and boring of the head into the pillow.

2. *Vertigo*, like headache, is due to different causes. In numerous cases it merely amounts to a slight sensation of dizziness. Sometimes it is an obstinate and troublesome symptom and calls for special treatment (essential vertigo.) The patient in severe cases has the sensation of "swimming" in the head, or as if external objects con-

stantly moved around him. Anæmic persons and invalids after fever are liable to vertiginous attacks on suddenly assuming the erect position. Vertigo attended by a momentary confusion of the mind constitutes one of the minor forms of epilepsy. Gastric disorders, affections of the eye and the ear, alcoholism and masturbation frequently give rise to vertigo. Associated with deafness, nausea and vomiting, it constitutes a very prominent symptom in *Meniere's disease*. A reeling gait resembling the effects of severe vertigo is a characteristic sign of cerebellar disease. In regard to the nature of vertigo, it appears to indicate an abnormal condition of the consciousness in relation to the position of the body. There is a disturbance of the sense of equilibrium.

3. *Delirium.* The diagnostic value of delirium is not of a high order. It is a symptom arising under influences of various and opposite pathological conditions. Delirium may assume all conceivable grades of mental excitement, from transient periods of wandering to maniacal frenzy. It may present all the features of insanity; but the incoherence of language and the morbid mental condition pass off with the affection that gave rise to the disturbance. In certain nervous diseases, however, much significance attaches to the peculiarity of the psychical derangement. The hallucinations in delirium tremens are repulsive and frightful to the patient. The demented paralytic revels in delusions of grandeur and exaltation.

4. *Insomnia.* Sleep is often much disturbed in functional nervous disorders. An excited or ill-nourished brain is incompatible with the conditions of rest and quiet. Wakefulness is frequently an initial symptom of insanity, and is a very common feature of chronic alco-

holism. Insomnia, accompanied by severe headache, is often a marked symptom of cerebral syphilis. Short snatches of sleep disturbed by unpleasant dreams more commonly result from gastric or intestinal irritation than from cerebral trouble.

5. *Somnolence.* A disposition to sleep at unusual hours is observed in cerebral anæmia and chlorosis. Great drowsiness may be a premonitory symptom of uræmic convulsions. Brain disease in children is often ushered in by prolonged and deep sleep. The so-called "inward fits" in infants come on during sleep. The child grinds its teeth, turns the eyes upwards, presses the fingers tightly on the palm of the hand and flexes the toes downwards (carpo-pedal spasm). These symptoms usually pass off without any serious consequences, but sometimes the little patient awakes with a start and cries incessantly, or has an attack of convulsions.

6. *Stupor, Coma.* Consciousness may be disturbed in different degrees. A bewildered or dazed condition of the mind is often occasioned by powerful mental excitement. Coma is usually ushered in by stupor, from which the patient can still be roused. In profound coma there is a total abolition of the perceptive powers. Loss of consciousness and utter insensibility constitute the essential symptoms of cerebral apoplexy. The usual mode of death in cerebral disease is by coma.

7. *Gastric symptoms* attending brain affections are probably of a reflex character, affecting the pneumogastric nerve. "Cerebral vomiting" is recognized by the absence of a palpable cause of gastric irritation. This symptom is so frequently present in children threatened with serious brain trouble that due weight must be given

to its appearance. Brain lesions occasionally give rise to gastric disturbances of such great severity and persistence so as to mask the real cause for a considerable length of time. Abernethy relates the case of a young woman whom he treated for what he thought to be some serious stomach affection. At the autopsy he found a large abscess of the brain: the stomach was perfectly healthy.

8. *Epileptiform convulsions.* All grave diseases of the brain tend to develop spasmodic seizures. Cortical lesions are especially prone to give rise to epileptiform convulsions (Jacksonian Epilepsy.) Unilateral convulsive movements or when one side is more affected than the other are usually dependent upon gross lesion of the cerebrum. Epileptiform attacks frequently occur in tumor, abscess and syphilis of the brain, and less frequently in meningeal and cerebral hemorrhage and embolism.

9. *Infantile convulsions.* Children of a tender age are exceedingly prone to spasmodic attacks, though in the greater number of cases they take a favorable termination. The unstable condition of the nervous system in the very young offers but slight resistance to trifling exciting causes, and in consequence reacts in an excessive manner. Many children are readily thrown into spasms by difficult dentition, and gastric or intestinal derangement from the presence of indigestible food or worms. It is a matter of daily experience that acute diseases occurring in early childhood are often ushered in by general convulsions. Sometimes it is difficult to foretell whether the sudden onset of the spasms indicates the establishment of some formidable disease (meningitis,

pneumonia, scarlatina, etc.), or is merely the temporary effect of a source of reflex irritation, or perhaps the forerunner of epilepsy.

10. *Nervousness.* This vague expression much in vogue in popular pathology has not yet been admitted into medical nomenclature. It is nevertheless true that patients often complain of numerous and anamolous symptoms which cannot be fairly labeled with the names of well-known nervous affections. A fruitful source of such undefinable ailments is undoubtedly dependent on an enfeebled condition of the general nervous system, however brought on. Such patients often exhibit a constitutional tendency to become readily excited, depressed or emotional. Among the special causes may be mentioned frequent loss of small quantities of blood, debilitating discharges, excesses of every kind, prolonged mental depression and the incipient stage of many chronic diseases. Conditions of this nature often underlie nervous dyspepsia, insomnia, the hysterical or hypochondric disposition and neurasthenia.

11. *Ocular symptoms.* The clinical significance of pupillary changes can only be adequately appreciated if the reflex mechanism regulating the movements of the iris be properly understood. The size of the pupil in health is determined by the quantity of light which enters the eye, and by the act of accommodation. The pupils contract when exposed to light, and dilate in the dark. They contract when looking at near objects, they dilate when looking at distant objects. As long as there exists but the feeblest sensibility to light, the pupillary reaction occurs, whether the light falls upon one or both eyes. Contraction of the pupil is in direct proportion to the

quantity of light impinging on the retina. The stimulus of light acting upon the retina is transmitted by the optic path to its cortical center and excites on reflex action the branches of the motor oculi that connect with the iris. Derangement of any of the integral parts of this reflex mechanism is manifest by abnormal reaction of the pupils.

12. *Optic neuritis* is a symptom of great frequency in cerebral disease. The change in the optic nerve when the fundus of the eye is examined by the ophthalmoscope, is characterized at the beginning by congestion and œdema, and if the inflammation does not subside, very marked changes take place, which are implied by the term "choked disk." The arteries are reduced in size, the veins are enlarged and tortuous, and finally atrophy of the disk ensues. Vision is often unimpaired, although the alterations discovered by the ophthalmoscope show the existence of optic neuritis. The optic neuritis observed in cerebral disease is bilateral and must be distinguished from that form which results from local causes. Primary optic atrophy tending to amaurosis occurs in locomotor ataxia. The ophthalmoscopic appearance of optic neuritis bears great resemblance to that of albumenuric retinitis.

13. *Paralysis of ocular muscles.* Isolated and combined paralysis of ocular muscles occur in diseases affecting the pons, the crus and base of the brain. Paralysis of the external ocular muscles is manifested by the occurrence of strabismus, diplopia or ptosis. In paralysis of the ciliary muscles, there may be loss of accommodation, or the reflex contraction of the iris in looking at near

objects may be intact, whilst the pupils do not contract on exposure to light. This symptom is known as the "Argyle-Robertson pupil." It is sometimes observed in locomotor ataxia and cerebral syphilis. Inequality of the pupils is often an early symptom of general paresis of the insane. Pupillary changes are observed in disease of the spinal cord when the oculo-spinal center is affected. Reflex irritation of the cervical sympathetic is probably the cause of dilatation of the pupils in migraine, nephritic colic and in children troubled with worms. It should be remembered that pupillary changes and paralysis of external ocular muscles frequently depend on local causes.

Hemianopsia, which means blindness of one lateral half of the retina, is a sympton of much diagnostic importance. The most common form of hemianopsia is blindness of the nasal half of one eye, and of the temporal half of the other eye. The usual test, if a perimeter is not used, is to close one of the patient's eyes with the finger and to request him to fix the open eye upon one spot or a near object. Standing in front of the patient, the examiner passes the unemployed hand up and down and to the right and left of the object at which the patient is gazing, and asks him whether he sees the hand distinctly and simultaneously with the object he looks at. If he does not, then the retina is blind on the side opposite to that on which the sight of the hand is lost. Hemianopsia has been observed in lesion of the cuneiform convolution of the occipital lobe, and also in lesion of the cerebrum, which implicates the optic tract or chiasm. Hemianæsthesia is sometimes attended by Hemianopsia.

14. *Auditory symptoms.* Disorder of the auditory nerve is not often witnessed in cerebral disease. General anæmia is sometimes attended by a buzzing or humming noise, or sounds resembling the tinkling of bells. There is reason to infer that the temporal lobe is the central destination of the auditory nerve. Deafness of one ear has been observed when the auditory path between the medulla and the superior temporal convolution of one side was involved in a lesion.

15. Disturbance of the function of the *olfactory nerve* is rarely noticed in cerebral disease. Hemianæsthesia is sometimes accompanied by loss of smell on the affected side. Hysterical patients occasionally experience perversion or obliteration of the sense of smell.

16. It is difficult to recognize disorder of the sense of taste. The *gustatory sense* is sometimes abnormally acute or perverted in hysteria. Nothing definite is known concerning the central destination of the gustatory nerve.

17. Paralysis of the *sensory portion of the fifth nerve*, which is of rare occurrence, causes anæsthesia from the vertex to the lower jaw, and loss of sensibility of the mucous membrane of the nose, tongue and mouth on the same side. The most serious effect resulting from paralysis of the nerve is atrophy and ulceration of the cornea. Paralysis of the *motor portion of the fifth nerve* causes weakness of the masticatory muscles of one side. Eventually the temporal and zygomatic fossæ become flattened from wasting of the fronto-temporal and masseter muscles.

18. A lesion in the path of the *facial nerve* above its nucleus, between it and its cortical destination, causes paralysis of the face on the opposite side. Paralysis of

the face on the same side as the lesion happens when the facial is implicated in the vicinity of the pons varolii. It is usually observed that in hemiplegia, only the muscles of the lower half of the face are affected. This shows that the lesion has involved the central path of the facial.

19. *Circulatory disturbances.* Alterations of the pulse are not commonly observed in chronic affections of the brain. The pulse in acute meningitis is variable. It may be rapid, wiry and jerky, or abnormally slow and feeble, irregular or intermittent. The full, bounding pulse in cases of cerebral hemorrhage is often due to ventricular hypertrophy.

20. The *tache cerebral* is a test to determine the condition of the cutaneous circulation which is frequently depressed in cerebral disease. On passing the finger somewhat firmly across the upper part of the inguinal region or the inner aspect of the thigh, a red streak appears after the removal of the finger, which but slowly fades when the circulation is feeble.

21. By the term "cerebral breathing" is understood a morbid alteration of the act of respiration, which is of bad omen in brain trouble. The breathing is noisy, nasal and interrupted. An aggravated kind of cerebral breathing is known as the "Cheyne-Stokes respiration." It consists of a series of respiratory movements that attain to a great rapidity and then gradually become exceeding slow.

Spinal Symptoms.

Sensory disturbances. Pain in the region of the vertebral column is a rare symptom in chronic affections of the spinal cord; but violent rhachialgic pain coming on

spontaneously or aggravated by movement of the body, occurs in spinal meningitis and intraspinal tumor. *General hyperæsthesia* is a marked symptom of irritation of the spinal cord. It is observed in spinal meningitis, tetanus, hydrophobia and hysteria. The reflex excitability is usually heightened in this condition. *Tenderness of the vertebræ* usually limited to the cervical and upper dorsal regions independent of disease of the bones or spinal cord is probably of a hyperæsthetic character (points apophysaire). This is the essential symptoms of "spinal irritation," but it is often associated with dorso-intercostal neuralgia. A dull constricting sensation encircling the waist frequently attending disease of the spinal cord is called the "girdle" or "cincture" sensation.

Motor Symptoms.

Paraplegia is the typical form of paralysis in inflammation of the spinal cord. In the vast majority of cases the inferior extremities are affected. The paralysis varies from complete loss of the muscular movements to incomplete paralysis or *paraparesis*. In mild cases there is a shuffling gait, or the patient is able to walk only with the aid of crutches. Sometimes the paraplegia merely amounts to muscular weakness of the legs. The distribution of the paralysis varies with the seat of the disease in the cord. The lesion may affect the lumbar, the dorsal or the cervical region, though in the greater number of cases the lesion is in the lumbar cord. In severe lesion of the cervical cord all four extremities are paralyzed. Sometimes the paralysis of one lower extremity is more complete than in the other, and may simulate hemiplegia. In very exceptional cases one lateral half of the spinal cord is

involved. The motor paralysis in conformity with the anatomical arrangement of this organ is on the same side as the lesion, and the anæthesia on the other side.

Impairment of the sphincters of the bladder and rectum occurs in transverse myelitis. Weakness of the bladder from this cause is recognized by imperfect evacuation of the urine. As the disease advances, the urine, on being retained, decomposes and becomes ammoniacal, which tends to develop cystitis and pyelitis. Rectal paralysis causes obstinate constipation and finally involuntary discharges.

Weakness of the *sexual function* usually accompanies the paralysis of the sphincters. It is indicated by imperfect erections tending to complete loss of the sexual power.

Tests of reflex action. Alteration of the reflexes is a common symptom in spinal disease. Reflexes are of two kinds, the superficial and the deep. The former are excited by irritating the cutaneous surface; the latter by exciting the tendons or fascia of muscles. Tickling or pinching the skin is the usual manner of eliciting the cutaneous reflexes. A more elegant method consists in stimulating the skin with the faradic current.

1. *The plantar reflex* is excited on tickling the sole of the foot. It causes a jerking movement of the limb, through the action of the gastrocnimii muscles.

2. *The cremaster reflex* is obtained on pinching the inner side of the thigh. This causes retraction of the testicle.

3. *The gluteal reflex* is induced on pinching the skin in the region of the buttock.

4. *The epigastric reflex* is excited on irritating the skin of the chest between the fifth and sixth intercostal spaces. It causes dimpling of the epigastrium.

The most important of the deep reflexes are the patellar tendon reflex and the ankle clonus.

5. *Patellar reflex* (knee jerk). One of the methods of eliciting this reflex consists in letting the patient sit on a chair, one leg being crossed over the other so that it does not touch the floor. A light tap with the edge of the hand on the patellar ligament immediately below the knee cap causes the foot to jerk forward through the action of the rectus femoris muscle. Another method of inducing the knee jerk, only practiced on males, is to raise and support the thigh by passing one hand beneath it, just beyond the knee joint, and to grasp the knee of the other leg. The tap is then made at the proper point of the free leg. Experiments in healthy individuals show considerable differences in regard to the energy of the patellar reaction.

6. In exciting the *ankle clonus* the leg is slightly bent upon the thigh and supported by placing the hand under it near the knee joint. The toes are seized with the fingers and pulled forward, so as to stretch the tendon Achillis. On suddenly flexing the foot, while the fingers continue to press against the toes, a series of rythmical contractions of the calf muscles ensue. The ankle clonus is not as readily excited as the patellar reflex.

Inhibition. There is good reason to infer the existence of an anatomical mechanism, probably in the spinal cord, which controls reflex action independent of the will. We know, that by an effort of the will, we often succeed in suppressing a strong inclination to laugh or to cry, and sometimes we are able to restrain a fit of sneezing or coughing. When infantile convulsions are checked by a warm mustard bath we have an analogous illustration

of the controlling influence of an artificial impression. Brown-Sequard put a stop to spinal spasm by forcible flexion of the big toe. In these examples it may be said that the reflex action which produced the spasm has been inhibited. Inhibition may therefore be supposed to take place when a center, which is the medium of reflex action looses its excitability on being acted upon by a nervous influence which forms a part of the same reflex mechanism. Perhaps many an anomalous phenomena in nervous and hysterical individuals is due to defective inhibitory action.

The subject of incoördination will be more conveniently considered in connection with locomotor ataxia.

CHAPTER III.

GENERAL THERAPEUTICS OF NERVOUS DISEASES.

Rest. Whilst in surgical treatment, absolute rest is often of itself curative, the beneficial influence of relaxation from nervous strain and exhaustion is not as frequently insisted upon as it should be. The benefit derived from the tonic and bracing effects of mountain air or a sojourn at the seaside cannot be overrated. Many an overworked person, broken down in health, who constantly complains of languor, sleeplessness, dyspepsia and a host of other nervous ailments that had baffled the ordinary remedies has returned from one of these resorts completely restored. Probably the chief factor in this happy change was the release from unremitting attention to business and the worry it entails.

The treatment introduced by Weir S. Mitchell in aggravated cases of functional derangement of the nervous system in females frequently meets with brilliant success. It chiefly consists in seclusion of the patient and in securing rest and quietude. Active exercise is replaced by electricity and massage. The diet is strictly controlled by the attending physician.

Diet. Derangement of the digestive functions frequently attends nervous disorder, and in turn a reduced

tone of the nervo-muscular apparatus develops an enfeebled digestion. This vicious circle of morbid influences may cause embarrassment of diagnosis. Dyspeptic symptoms obstinate to treatment are often but the manifestations of nervous depression. It can hardly be expected that anorexia, nausea and epigastric uneasiness brought on by anxiety, disappointment and other mental trouble, perhaps bad habits, will yield to regulation of diet and reputed anti-dyspeptic remedies. A starving dietary would surely augment the mischief. On the contrary, sufficient substantial nourishment is called for, and the medical attendant will do well, who, under these circumstances, succeeds in weaning his patient from the domineering caprice of a squeamish stomach. Above all, nervous patients should be warned against yielding to the seductive fascination of alcoholic stimulants, however much they may afford temporary relief. What may be called "nervous dyspepsia" is very often but the obtrusiveness of digestive symptoms among manifold complaints that arise in general deterioration of health from whatever cause.

Opium. Among the internal remedies in the treatment of nervous diseases, none are as frequently employed as the narcotics. These substances relieve pain, promote sleep, arrest spasm, and often exert a beneficial influence on the course of many diseases. The therapeutical properties of opium, especially its chief alkaloid, morphia, are not equaled in efficacy by any other of the class of sedatives. Since the introduction of the hypodermic syringe, the desired promptness of action of morphia and its compeers can be obtained.

Codeia is a good substitute for morphia, when the latter, owing to idiosyncrasy, cannot be tolerated. It is not so likely to derange the stomach or to confine the bowels as morphia, but is less reliable in its action, and must be given in relatively larger doses. As a general rule it cannot take the place of morphia, but very strange susceptibilities are sometimes witnessed. I remember the case of a young man who suffered from chronic ulcer of the stomach. His attacks of hematemesis were frequently attended by violent cardialgia. Hypodermics of morphia invariably brought on epileptiform convulsions, while codeia gave prompt relief.

Belladonna. It must be said in favor of belladonna that it sometimes exerts a sedative effect when morphia fails. Owing to the antagonism of these powerful remedies they are often given in combination. The tincture of belladonna, or the extract, is often useful in neuralgia, gastralgia, whooping cough and spasmodic affections. This remedy still retains some reputation in the treatment of epilepsy. The parvules of atropia of different strengths afford a reliable and convenient mode of prescribing this medicine. Extreme caution is advisable in the use of this powerful remedy.

Hyosciamus. The hypnotic effects of this remedy suggest its use when opium is indicated, but it is far less certain. Hyosciamine in doses of gr. 1–150 has been found useful in tremor.

Aconite. Remarkable cures of inveterate facial neuralgia are reported from *aconitia.* This dangerous alkaloid is given in doses of gr. 1–200.

Stramonium leaves enter into the composition of cigarettes and pastiles for the relief of asthma.

Calabar bean. Its alkaloid *physostigma* has been chiefly employed in the treatment of tetanus.

Canabis Indica sometimes acts well as a hypnotic and in mild forms of neuralgia.

Conium. This remedy is little used. Alienists ascribe to it the effect of subduing maniacal excitement.

Chloral hydrate is one of the best hypnotics we possess, though it does not relieve pain like morphia. This remedy is often of signal service in acute alcoholism.

Chloroform. This valuable anæsthetic is often our last resort in severe and protracted forms of spasmodic paroxysms.

The bromides form a class of remedies of great value. They lower reflex excitability and exert a general soothing effect on the nervous system. Bromide of potassium alone or in combination with other bromides is our sheet anchor in epilepsy. Large doses of this drug promote sleep.

Alcohol in the form of whisky, brandy or wine is unsurpassed as a restorative in shock or sudden nervous prostration. These liquors cannot be replaced by any other remedies when reliable stimulants are indicated.

Gelseminum sempervirens. The tincture or fluid extract of yellow jessamine is strongly recommended in neuralgia of the dental nerve. This remedy is said to quiet the "hydrocephalic cry" in meningitis.

Antipyrin, Phenacetin, Sulfonal. These newly introduced remedies possess undoubted analgesic qualities. Their excellent effects in nervous headache is especially praised. Sulfonal does good service in insomnia.

Paraldehyde is well adapted to allay the mental excitement and worry of hysterical patients. It is also a hypnotic.

Phosphorus is now much prescribed as a nervine tonic. Its reputation has yet to be established.

Strychnia. This powerful excitant of the axial nerve center was formerly the most favorite remedy in all paralytic affections. Its use has been in a great measure superseded by electricity. Subcutaneous injections act sometimes favorably in feebleness of the bladder and rectum and impairment of the sexual function.

Iodide of potassium. Independent of the antisyphilitic action of this remedy, a considerable influence in controlling tissue changes of a low inflammatory character may be claimed for it. The good effects not infrequently witnessed from its employment in cases where no history of syphilis exists, justifies such a favorable opinion.

Musk, camphor, ether, aromatic spirit of ammonia, sweet spirits of nitre and valerian are frequently useful for the relief of nervous depression and various functional ailments.

Among the empirical remedies most frequently used with advantage in the treatment of chronic nervous diseases, belong arsenic, nitrate of silver and preparations of zinc and gold.

Medical Electricity.

The importance of electricity as an invaluable therapeutical agent in the treatment of nervous diseases demands for it a larger space than has been assigned to the consideration of other classes of remedies.

The electric currents in use in medical practice include the galvanic and faradic currents. Static electricity is as yet very little employed.

Galvanism. (voltaism, constant current, battery current.) Galvanic electricity is generated by the contact of dissimilar metals which are submitted to chemical action. The simplest arrangement consists of the combination of a plate of zinc and copper or of carbon immersed in a glass vessel containing dilute sulphuric acid. To each of the plates outside of the fluid, a wire is attached, and as soon as these wires are joined, a current of electricity is established which continues to flow until the plates are oxidized. Such an arrangement is called a cell or an element. The combination of a number of cells consisting of an alternate series of dissimilar metals constitute a battery. The terminal of the wire connected with the copper plate, the last in the series, is the *positive pole* or the *anode*. That connected with the zinc plate at the other end of the series is the *negative pole* or the *kathode*.

In explanation of the terms positive and negative, it should be understood that electricity inheres in all bodies. The tendency to develop electricity is called the *potential* of the body. The earth, being an unlimited reservoir of electricity, is taken as a standard in regard to the comparative potential of a body. Those bodies from which electricity flows to the earth we call *positive bodies*, and bodies which draw electricity from the earth we call *negative bodies*. The former are said to be of a *high potential*, the latter of a *low potential*. The terms positive and negative merely express the difference of the potential; for all bodies are relatively positive and

negative. When we say that a body is charged with positive or negative electricity, we imply the condition of positive or negative potential of a body. This may be made more clear if we compare two different metals to two tanks containing water at different levels. If the tanks be connected by a tube, the water at a high level in the one tank will flow into the second tank containing water at a lower level. If a third tank containing water at a still lower level be connected with the second tank, then the water between these two tanks will flow again. The water will cease to flow as soon as it has reached a common level in all the tanks. In a precisely analogous manner we must create a difference of potential between two points if we wish to have a flow of electricity, and likewise the current of electricity ceases when an equilibrium is established between the high and low potential. Within the galvanic cell positive electricity passes from the zinc plate to the copper or carbon plate, and outside of the battery the current passes to the zinc in completing its circuit. It is due to the latter circumstance that the terminal of the connecting wire attached to the zinc element is called the negative pole.

Faradism. Faraday's discovery that a galvano-magnetic current induces new currents in a neighboring conductor, has been utilized in the construction of an apparatus which renders such "induction currents" available for medical use. A portable faradic battery consists of one or two galvanic cells that generate the original current. A coil of wire wound around a wooden cylinder and having in its center a bundle of soft iron rods is connected at one end with the cell and at the

other end with an automatic interrupter. This interrupter is an ingenious contrivance by which the "make" and "break" of the current is effected. It consists of a spring attached by its lower end to the coil. Its free extremity has the form of a hammer which impinges upon the iron core of the coil and is in contact with a screw that connects with one of the poles. As long as the current generated in the cell is not interrupted, this mechanism is at rest, but as soon as the iron core becomes magnetized it attracts the hammer and the circuit is broken. On quickly becoming demagnetized, the hammer by force of its spring flies back to the screw, the circuit being thus again re-established. The successive closure and opening of the circuit generates the induction current in the coil. This current is called the *primary current*. A second coil consisting of thinner and longer wire surrounds the primary coil, but is not otherwise connected with the apparatus. The connecting wire (rheophore) to which the electrodes are attached, connect with the terminals of the second spiral. The current which develops in this second coil constitutes the *secondary current*. This current is the one usually preferred in medical practice. The strength of the current is regulated by sliding the upper coil over the lower to the required distance, which is indicated by a graduated scale. Faradic batteries are now made in which the coils are immovable, the strength of the current being regulated by a draw tube. The play of the hammer can be controlled by the finger or by means of a stud, which every good battery should have.

It should be remarked that the physiological effects of

both the primary and secondary currents only become manifest with the break of the original current.

Electro-motive force. The work which a definite quantity of electricity can perform is called its electro-motive force. This working capacity of a current is influenced by the resistance which the current meets in its passage. There are two such resistances—the one essential to the battery (fluid, connecting wires) called the "internal resistance," and the other, outside of the battery (the interposition of a part of the human body) called the "external resistance." Ohm laid down the following law for the determination of the strength of electric currents: *The strength of a current is always proportionate to the electro-motive force divided by the resistance.* This is mathematically represented by the formula $C = \frac{E}{R}$. C signifies the current, E the electro-motive force and R the resistance. If we suppose E = 5 and R = 100, then $C = \frac{5}{100} = \frac{1}{20}$. It is apparent that we cannot increase the strength of a current by merely multiplying the number of the elements —since for each increase of electro-motive force we get a proportionate increase of resistance. For instance, if we use two elements, our formula becomes $C = \frac{5+5}{100+100} = \frac{1}{20}$ *i. e.*, the same as for one, and obviously with a like result for any greater number of elements. We obtain a different result when an external resistance is interposed, which is always very great in comparison with the internal resistance. This occurs in practice when the human body forms a part of the circuit. In such a case, giving the same values to E and R as above, let us call R_1 the external resistance 1000; we then have the equation $C = \frac{E}{R+R_1}$ and substituting values, $C = \frac{5}{100+1000} = \frac{1}{220}$.

If we now use two elements we get $C = \frac{5+5}{100+1000+100} = \frac{1}{120}$ the strength of the current is nearly doubled, for the external resistance is the same for any number of elements as it is for one. Practically, the minute additions of the internal resistance may be entirely neglected in the calculation of the current strength when the external resistance is relatively very great.

Measurement. In medical practice we adopt the one-millionth part of an ampere as a unit of measure for the current strength, and call it a milliampere. The scale of galvanometers is now usually divided into milliamperes.

Density. The strength of a current in relation to a transverse section of its conducting medium is called the density of the current. This means that the current strength is in proportion to the quantity of electricity which, in a given moment of time, passes through a sectional area of the conducting medium. Suppose an electric current be conceived to consist of a bundle of parallel rays it is evident that the smaller the diameter of a bundle the more compact will be the rays, and the density of the rays will increase in proportion. The density of a current is therefore the strongest in the conducting wires, less strong in the electrodes, and least in the lines of distribution through the human body. Among the rays of the current in its passage, the strongest is the straight one between the points of application, for it takes the shortest route and meets with the least resistance. All the other rays from pole to pole decrease in strength in proportion to the length of their circuitous course, and the cumulative resistance they encounter. If it is

intended to excite a certain part of the body, the method of conducting the current must be such that its greatest density shall act upon the part.

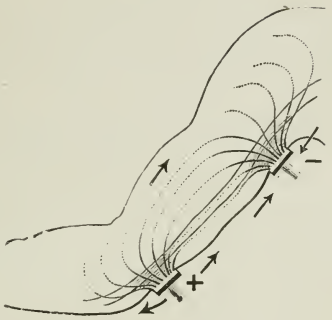

FIG. 13.

This diagram is intended to illustrate the diffusion of the current throughout the arm. It also illustrates the greatest density of the current in the immediate vicinity of both electrodes of equal size, which are placed over a nerve (ulnar). The inactive rays of the current are marked by dotted lines; the shaded parts indicate the regions of greatest density.

Polar action. A contraction is the visible effect of the current when acting upon a healthy nerve or muscle, and a contraction only develops at the moment when there is a make or a break of the current. But the energy and amplitude of contractions are also influenced by the particular pole with which the closure and opening of the circuit is effected. This peculiarity of the electrical phenomena exhibited in normal neuro-muscular organs is called the "polar action." Examination with the galvanic current in varying the experiment with currents of different strengths exhibits the normal laws of polar action:

a. With a *weak current* the first perceptible contraction occurs, when the circuit is closed with the kathode. This

kathodal closing contraction is expressed by the letters K C C (sometimes C C C or Ka S Z, according to the German notation).

b. A *medium current* excites a strong kathodal closure contraction (K C C^{1}), also a moderate anodal closure contretion (A C C), and a moderate anodal opening contraction (A O C).

c. A *strong current* excites a tetanic kathodal closure contraction (K C C^{11}), also a strong anodal closure contraction (A C C^{1}), a strong anodal opening contraction (A O C^{1}) and a perceptible kathodal opening contraction (K O C).

It will be observed that according to the normal laws of polar action the closure contractions appear earlier than the opening contractions, and that there is a regular order of polar action in relation to the appearance of the contractions. The formula of normal polar action only holds good when muscles are *indirectly* acted upon by exciting their corresponding motor nerves with the galvanic current. The *direct* stimulation of muscles is only effected by closure currents.

Electrotonus. Although when a current passes uninterruptedly through a motor nerve or muscle no visible effect is produced, experiment shows that a change occurs in the condition of the nerve during this apparent quiescence. This change consists of an altered irritability of the nerve. Such a nerve is said to be in a condition of electrotonus. The irritability of the nerve is heightened at the point of contact of the kathode and in its immediate vicinity (katelectrotonus); it is lowered at the point of contact of the anode and in its vicinity (anelectrotonus). From this circumstance it is inferred that the kathode is the stimulating pole and the anode the sedative pole.

The conducting power of organic tissues. The distribution of the current in the human body is influenced by the inequality of the conducting power of the different tissues. The muscles and nerves are the best conductors. The epidermis opposes the greatest resistance to the passage of the current, but this resistance varies in different areas of the cutaneous surface, as seen by the following table according to the investigations of Erb:

Temples	40°
Cheeks	50°
Side of the neck	35°
Shoulder-blade	30°
Anterior surface of thigh	3°
Anterior surface of the upper arm	25°
Popliteal space	26°
Palm of the hand	20°

Effects of the current on the special senses. The faradic current barely affects the special senses, but they promptly react to the galvanic. On applying a weak current to the temple or cheek the sensation of a flash of light is experienced. When a closure or opening current is applied to the ear, a whistling, ringing or hissing noise is heard. Stimulation of the tongue causes a peculiar metallic taste in the mouth. The sense of smell does not appear to be influenced. Galvanization produces a peculiar stinging or burning sensation of the skin. Faradization causes a feeling of tingling. Strong currents cause pain and spasmodic rigidity of muscles.

Electro-diagnosis. Electric examination intends to determine the existence or non-existence of quantitative and qualitative changes of the excitability of nerves and muscles. *Quantitative changes* relate to energy and amplitude of contractions. A normal electric contraction

develops at once and is brief and vigorous. Diminution of the electric reaction is indicated when a relatively strong current is required to excite a contraction, or when the contraction develops slowly and is prolonged. The strongest current fails to excite a contraction in complete loss of electric reaction. Morbid increase of the electric excitability is manifest when a weak current elicits an energetic contraction of great amplitude. *Qualitative changes* chiefly relate to the existence of abnormal polar reaction. A diseased nerve or muscle may react to a weak current, but the contractions manifest a reversal of the normal formula of polar action. Such an irregular order of the electric reaction indicates the existence of degenerative changes of motor nerves or muscles, and is therefore called the *reaction of degeneration* (R D). This condition is exhibited, for example, when the sequence of normal polar action is altered, so that A C C appears earlier than K C C, or when K O C is too promptly excited. It may also be found that the faradic contractility of a muscle is diminished or abolished, while the muscle still responds to the galvanic current for a certain time, and even with increased energy. In severe cases of atrophic paralysis exhibiting R D a gradual diminution of the galvanic muscular contractility occurs until it is finally abolished. It is remarkable that in favorable cases the voluntary power of the affected muscle is restored sooner than its electric excitability. The reason why a degenerated muscle reacts, at least feebly, to the galvanic current and not to the faradic, has not yet been explained. *A partial reaction of degeneration* is sometimes observed. The nerve in this condition retains its electric excitability and the muscular contractility is normal, but the direct galvanic excitability of the muscle

is increased and the normal sequence of polar action is altered. This form of degenerative reaction indicates anatomical changes in the muscles, but not in the nerve. It is often manifest in atrophic paralysis.

Motor points. To facilitate electric examination it is very advantageous to be familiar with those regions on the surface of the body where nerves are accessible to electrodes. Stimulation of such "motor points" excite a number of muscles to which a nerve trunk or a large branch is distributed. By this indirect method of electrization deep muscles are reached by the current. The annexed figures, showing the motor points, are reproduced from Ziemssen and Erb.

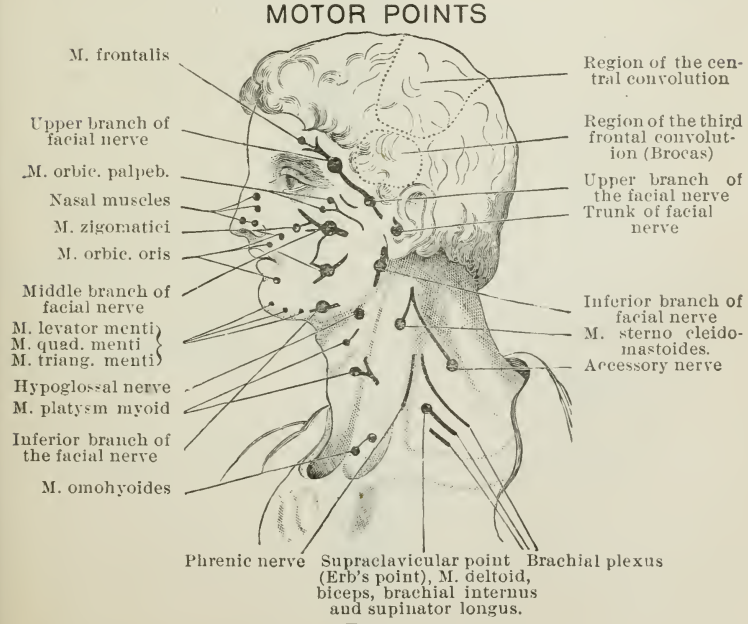

FIG. 14

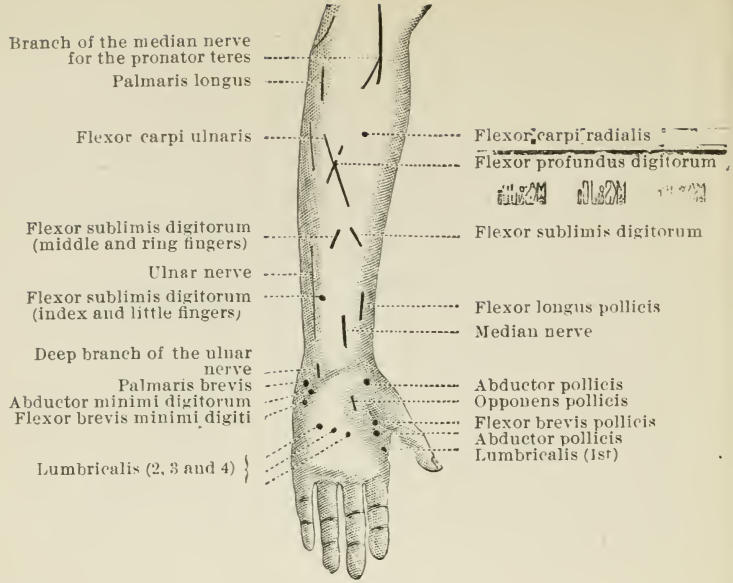
Branch of the median nerve for the pronator teres
Palmaris longus
Flexor carpi ulnaris
Flexor sublimis digitorum (middle and ring fingers)
Ulnar nerve
Flexor sublimis digitorum (index and little fingers)
Deep branch of the ulnar nerve
Palmaris brevis
Abductor minimi digitorum
Flexor brevis minimi digiti
Lumbricalis (2, 3 and 4)
Flexor carpi radialis
Flexor profundus digitorum
Flexor sublimis digitorum
Flexor longus pollicis
Median nerve
Abductor pollicis
Opponens pollicis
Flexor brevis pollicis
Abductor pollicis
Lumbricalis (1st)

FIG. 15

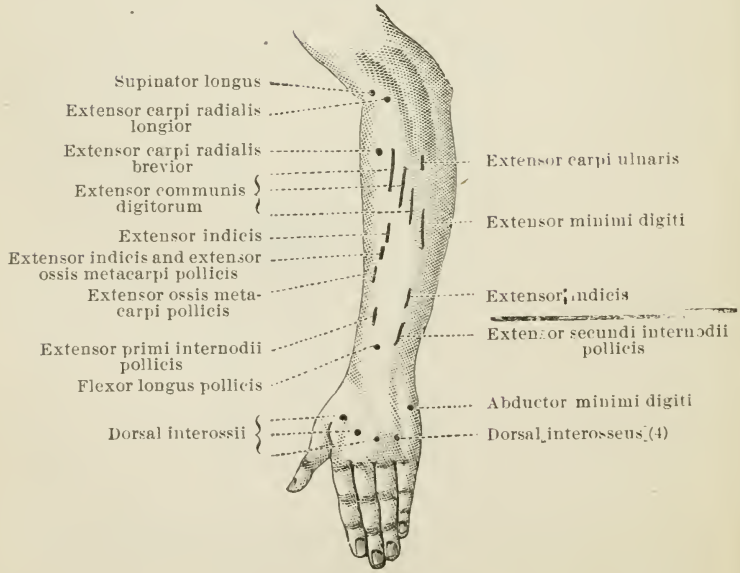
Supinator longus
Extensor carpi radialis longior
Extensor carpi radialis brevior
Extensor communis digitorum
Extensor indicis
Extensor indicis and extensor ossis metacarpi pollicis
Extensor ossis metacarpi pollicis
Extensor primi internodii pollicis
Flexor longus pollicis
Dorsal interossii
Extensor carpi ulnaris
Extensor minimi digiti
Extensor indicis
Extensor secundi internodii pollicis
Abductor minimi digiti
Dorsal interosseus (4)

FIG. 16

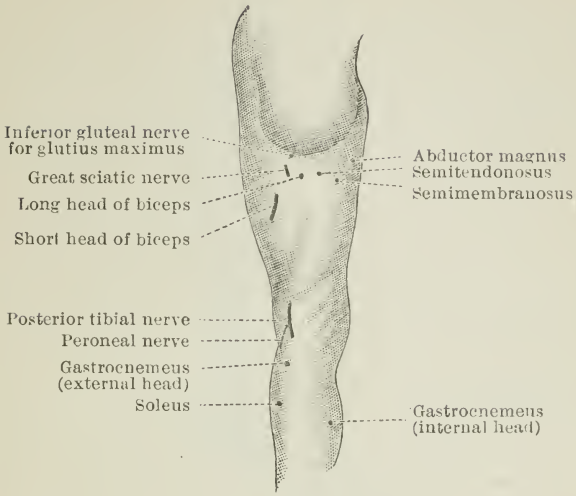
Inferior gluteal nerve for glutius maximus
Great sciatic nerve
Long head of biceps
Short head of biceps
Posterior tibial nerve
Peroneal nerve
Gastrocnemeus (external head)
Soleus
Abductor magnus
Semitendonosus
Semimembranosus
Gastrocnemeus (internal head)

FIG. 17

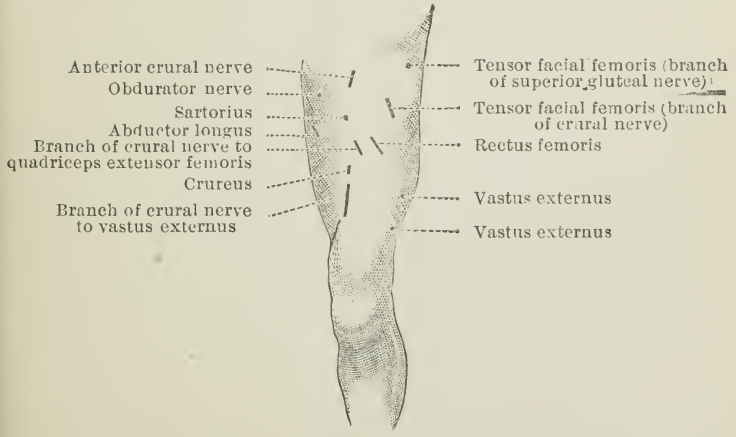
Anterior crural nerve
Obdurator nerve
Sartorius
Abductor longus
Branch of crural nerve to quadriceps extensor femoris
Crureus
Branch of crural nerve to vastus externus
Tensor facial femoris (branch of superior gluteal nerve)
Tensor facial femoris (branch of crural nerve)
Rectus femoris
Vastus externus
Vastus externus

FIG. 18

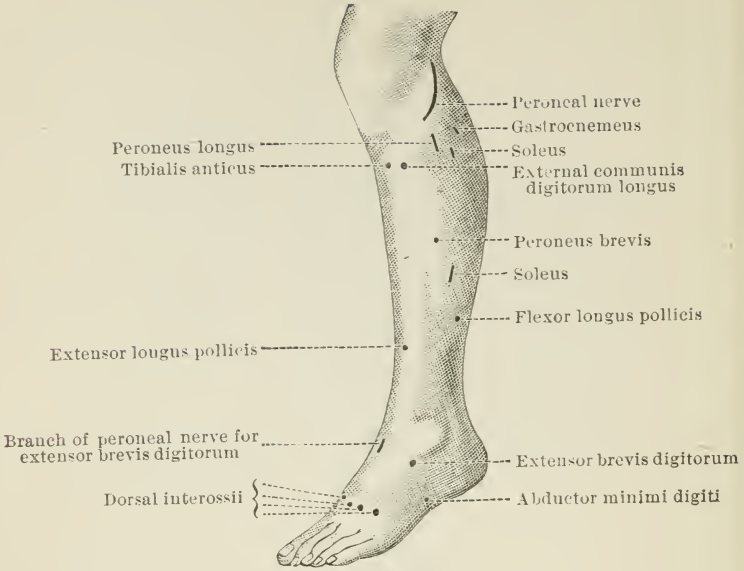
Peroneal nerve
Gastrocnemeus
Peroneus longus
Soleus
Tibialis anticus
External communis digitorum longus
Peroneus brevis
Soleus
Flexor longus pollicis
Extensor longus pollicis
Branch of peroneal nerve for extensor brevis digitorum
Extensor brevis digitorum
Dorsal interossii
Abductor minimi digiti

FIG. 19

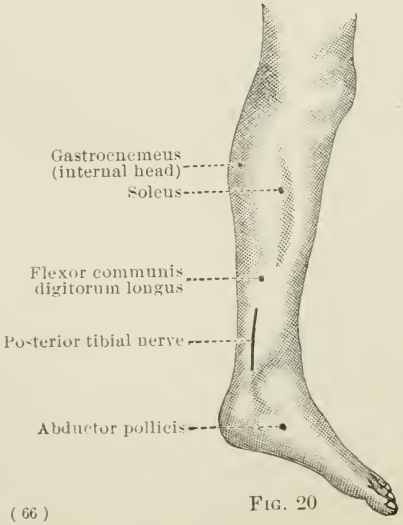
Gastrocnemeus (internal head)
Soleus
Flexor communis digitorum longus
Posterior tibial nerve
Abductor pollicis

FIG. 20

Methods of electric examination. In forming a correct judgment of the comparative reaction of symmetrical muscles it is necessary to choose a current of the same strength and electrodes of the same size. Symmetrical points of the two sides must be tested in every separate examination. The sponges should be well soaked in warm water (to which salt may be added), and the skin thoroughly moistened with the same solution. Interruptions, when the galvanic battery is used, are made with a special interruptor. It consists of an electrode handle furnished with a spring which is connected with a stud. Pressure with the finger on the stud controls the make and break of the circuit. The strength of the faradic current is measured by the sliding scale or draw-tube. The secondary current is preferred in examinations. In testing with the galvanic current the number of cells in use must be noted. The galvanometer is, however, a more exact means of measurement. Small or "fine" electrodes are selected for exciting motor points and small muscles.

In examining with the faradic current it should be noticed what minimum strength of the current is requisite to cause a contraction of each of the symmetrical muscles. The suspected muscle may be as excitable as its fellow, or require an increased current strength; or its electric contractility may be diminished in energy, or abolished. Examination with the galvanic current determines similar conditions of the muscular contractility, and especially the signs of the reaction of degeneration.

Electrotherapy. Little of a positive character can be advanced in explanation of the *modus operandi* of elec-

tricity as a therapeutical agent. Its reputation rests for the most part on the teachings of experience. We know that certain pathological conditions are modified under the influence of electricity in some undetermined manner. The peculiar stimulant effect of the current on nerve or muscle is of great value in paralytic affections. It is supposed that in cases where electricity exerts a beneficial influence on pathological changes, it is by a process of molecular or chemical action. When morbid exudations tend to be absorbed and removed through the effects of the current it may be conceived that an osmotic process has been developed among the organic cells. The palliative influence of the current in hyperæsthesia, neuralgia and spasm is perhaps to be attributed to its anelectrotonic effect. Experiments tend to show that electric excitation may act beneficially by exciting reflex action.

Methods of using the current. A *stabile* current means the application of electricity when no contraction is intended to be excited. The electrodes in this method are held immovable upon the part. By *alabile* current is understood a more active influence of the current. For this purpose one of the electrodes remains in contact with a part of the body and with the other the muscle is dabbed or stroked. Energetic contractions are caused by successive closures and openings of the circuit with the interrupter. An increase of the strength of the continuous current without adding to the number of cells is accomplished by occasional reversals of the poles during the sitting (Voltaic alternatives). Stimulation of the skin is best done with the faradic brush.

Position of the electrodes. The following rules in regard to the position and size of the electrodes are of practical importance:

1. When it is desired to concentrate a current on a large structure near the surface, both electrodes should be of a medium size and placed upon it near to each other, and be successively applied to all of its parts. This method is suited to large muscles, as the deltoid, the gluteus, vastus internus, etc.

2. When it is intended to send a current to an elongated structure, such as the spinal cord, it is advisable to select two very large electrodes, and to place them wide apart over the region of the affected organ so that the rays of the current may include a part of it.

3. Deep seated structures may be reached by the current when two large electrodes are placed opposite each other so that some rays of the current may permeate the diseased part of the organ. This method is adopted for electrizing circumscribed lesion of the brain and diseased joints.

4. If it be desired to direct the current to a particular part by the polar method a small electrode is selected which is then called the "active electrode." The other electrode of a larger size, called the "indifferent electrode," is applied to a remote part, usually the nape of the neck or the knee cap.

The direction method. As this method of electrization is advocated by eminent observers it is proper to refer to it here. A current is said to be *descending* when it passes from the center to the periphery, namely when the kathode is nearest to the muscle, and conversely, it is an *ascending* current when it passes from the periphery to

the center, the anode being then nearest to the muscle. The majority of electricians prefer the polar method, which certainly has superior advantages.

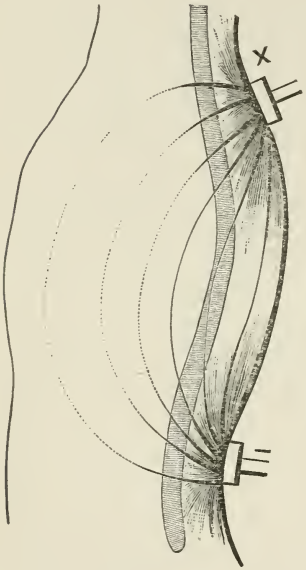

Fig. 21.

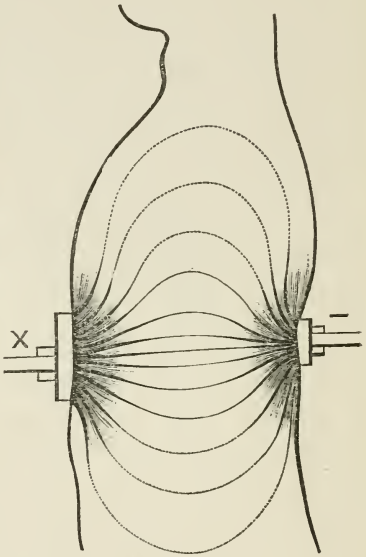

Fig. 22.

Fig. 21 is intended to show the necessity of placing the two electrodes sufficiently apart to allow the current to reach the organ (spinal cord) when it lies at a certain depth below the surface.

Fig. 22 shows the greatest current density by the full lines. The dotted lines represent the inactive currents. This figure also shows the greatest current density at the small active electrode which is indicated by the darker shading of the lines.

General galvanization is practiced for the purpose of influencing the nerve centers. The positive pole is successively passed from the vertex and forehead down the

whole length of the vertebral column. The negative pole is applied to the epigastirum. A sitting should at least occupy fifteen minutes. General galvanization is recommended in the treatment of insomnia, neurasthenia and various functional affections of the nervous system.

General faradization. The patient in this method is seated on a low chair, his feet resting on an electrode of suitable size which is connected with one of the poles of the faradic battery. With the other electrode, mounted with a large sponge, the operator electrizes the whole surface of the body from head to foot, care being taken to adjust the strength of the current to the varying sensitiveness of the different parts of the body. An application should occupy about twenty minutes. General faradization has a refreshing effect in muscular weakness.

The electric bath is a mode of general faradization. One of the poles of a strong induction current is placed in the bathtub in which the patient is immersed to above his shoulders. The operator faradizes the body with the other pole, which should be mounted with a large sponge.

The electric hand. This method is adopted for stimulating the face of children and timid persons. One sponge is held by the patient, the other by the operator. The latter applies the palm or fingers of his free hand to the well-dried skin of the patient. Dusting the face with toilet powder mitigates the unpleasantness of the application.

Practical rules. An overdose of electricity should be avoided. There is no better way of becoming familiar with the different strengths of the current than to practice on one's self. A good way of judging the intensity

of the current is to apply one of the electrodes to the ball of the thumb. To prevent the pain which is caused in using a strong galvanic current, it is advisable to switch in the desired number of elements, a few at a time, and to exclude them in the same manner before removing the sponges. A firm steady pressure on the electrodes produces less pain than holding them lightly. Dry sponges or the uncovered disks, and, better still, the electric brush should be used in anæsthesia. Wet sponges must always be used in electrizing motor nerves and muscles. Daily sittings are required where the cumulative effect of the stimulation is desired. The duration of a sitting varies according to circumstances from two to fifteen minutes. Batteries require constant care to keep them in good working order. In most instances when an induction apparatus loses its force it is due to rust which collects on the hammer or to some flaw in the conducting wires. The fluid requires frequent renewal.

Friction, massage, tapotement. The method of rubbing and kneeding the skin and subjacent muscles is much adopted in the treatment of nervous disorders. This procedure, technically called massage, takes up much time, and to carry it out effectually requires considerable expertness. Another method of practicing massage consists in hammering the muscles. For this purpose the inferior edges of the hands may be used after the manner of meat choppers. A more elegant and efficient way of practicing tapotement is to tap the part with India rubber balls or cylinders fastened to a wooden handle. The percussion hammer is well adapted for use on superficial nerves and the small muscles of the face.

CHAPTER IV.

DISEASES OF THE NERVOUS SYSTEM.

Diseases of Peripheral Nerves.

NEURALGIA.

Pathology. There are sufficient reasons to consider neuralgia an independent disease. Although pain is always due to a morbid condition of some part of the sensory apparatus, yet the pain of neuralgia cannot be referred to a known pathological change of the affected nerve. Probably the anatomical alteration is sometimes of the nature of a congestion or slight inflammation of the nerve sheath, somewhat of the character of a neuritis. In the greater number of cases we can only conceive of the existence of a peculiar morbid sensibility of certain nerves which is intensified by a source of irritation. Clinically neuralgia may be defined to imply the spontaneous occurrence of paroxysms of pain of great severity, limited to the course of a nerve and frequently associated with motor and vaso-motor symptoms.

Etiology. In many cases of neuralgia we recognize abnormal influences and conditions that act as predisposing or exciting causes. The predisposing causes include 1. *Heredity.* This is an important etiological factor as shown by the frequent occurrence of allied neu-

roses in members of the same family, such as hysteria, neurasthenia, epilepsy, etc. A neuropathic tendency disposing to the development of neuralgia may also be fairly presumed to exist in individuals free from hereditary influence. 2. *Age* exerts a marked influence. Neuralgia usually develops between the ages of twenty and forty, which includes the period of life when the general nervous system is most actively engaged. Children of a tender age and persons advanced in life rarely suffer from neuralgia, but elderly people are sometimes subject to a terrible form of this affection (epileptiform neuralgia.) 3. *Sex.* Females are more frequently attacked than men. Pregnancy and the climacteric period especially dispose women to neuralgia. 3. *Constitutional conditions.* General anæmia and the chlorotic diathesis often act as predisposing causes. The impoverished state of the blood and the consequent mal-nutrition lessen resistance to the obnoxious influences that favor the development of neuralgia.

Among the exciting causes of neuralgia are included: 1. *Cold.* Neuralgic attacks are often traceable to the direct effects of exposure to draughts of cold air or living in damp cellars. 2. *Traumatic* and *analagous causes,* including injury to superficial nerves from contusion and laceration, or irritation from a contiguous tumor, aneurism, necrosed bones and the contraction of cicatrices. 3. *Malarial influences.* The periodical type of neuralgia is usually but not always of malarial origin. 4. *Toxic causes.* The most frequent sources of neuralgia from these causes are chronic lead and arsenical poisoning. 5. *Syphilis.* The nocturnal pains in syphilis may be of a purely neuralgic character. 6. "*Reflex neuralgia.*" The

frequent connection of neuralgia with disease of remote organs, more especially with uterine and ovarian trouble, is a common observation. If this form of neuralgia as is supposed is of a reflex character, it is difficult to understand the connection. Finally neuralgia is often associated with diabetes mellitus, gout and pulmonary phthisis.

General Symptomatology of neuralgia. The essential characers of neuralgia have already been described. The onset of an attack may be sudden, but premonitory symptoms are often noticed consisting of a sensation of pricking, furriness or coldness in the area to which the affected nerve is distributed. Usually the pain begins with twinges in the course of the nerve that soon assume great intensity with variable intermissions. The pain is of lancinating, darting, tearing or burning character. The paroxysms of pain greatly vary in violence and duration. There are patients who have only one attack or several at long intervals. Others suffer for a prolonged period with numerous attacks in rapid succession, always affecting a particular nerve or one of its branches.

Painful points. The pain often affects with aggravated intensity certain points in the path of the nerve. These *puncta dolorosa* are recognized by passing the finger along the course of the nerve and exerting some pressure which causes a feeling of soreness even during the intermissions of the paroxysms. They are not always present, but usually they are found in parts where the painful nerves pass through bony canals or penetrate the fasciæ of muscles. It is not unusual for the pain to radiate to other twigs of the same nerve. Symmetrical nerves or even

nerves in remote parts of the body are sometimes simultaneously affected.

Motor irritation. Symptoms of motor irritation frequently associated with neuralgia, consist of twitching of muscles or cramps when a mixed nerve is affected as in sciatica. But more frequently the motor disturbance is of a reflex nature. This is well examplified in the "convulsive tic" of facial muscles in trigeminal neuralgia.

Vaso-motor symptoms are especially often observed in neuralgia of the face and head. There is marked pallor of the face followed by redness of the skin and of the adjacent mucous membranes. The secretions of the lachrymal and salivary glands are increased.

The trophic changes of neuralgia consist of cutaneous eruptions in the area of the affected nerve, atrophy of the skin, especially of the fingers, and alteration of the color of the hair in regions corresponding to the path of the painful nerve.

The general health in neuralgia does not seem to suffer, but in long standing cases an excitable condition of the nervous system develops which gives rise to a despondent state of the mind.

General treatment of neuralgia. It is of prime importance in all cases of neuralgia to search for its possible cause. A morbid tendency whether constitutional or acquired frequently opposes the best directed efforts toward permanent cure. To guard against the recurrence of attacks, those prophylactic and therapeutical measures are indicated that invigorate the general system and especially tend to improve the condition of the blood. Cases that admit of surgical interference promise satisfactory results. Thus foreign bodies and necrosed bones may

be removed, cicatrices split or excised and neuromatous tumors extirpated. Neuralgia occurring at regular periods, whether caused by malaria or other morbific influences are often successfully treated with quinia or arsenic. Neuralgic pain of syphilitic origin indicates specific treatment. Iron has a deserved reputation in all varieties of neuralgia occurring in anæmic individuals. Codliver oil and malt extract may also be given with advantage. Local treatment sometimes suffices in very mild cases. Good effects are often derived from the various forms of sedative and stimulant applications. The cantharidal collodion is certainly preferable when in cases of this kind a strong counter-irritant effect is desirable. This vesicant has the advantage that it can be applied with a brush to any part of the surface.

Electricity is often of great service. Different methods of using the current may be adopted. It is a good method to apply the anode of a weak galvanic current to the whole extent of the affected nerve, or to select the painful points. The strength of the current may be gradually increased. Variations of the current strength during a sitting, or interruptions should be avoided. In neuralgia of large nerve trunks the anode of a descending stable current should be applied either to the center of the nerve or to the corresponding region of the vertebral column, and the kathods to an indifferent peripheral point. Sometimes the interrupted current gives good results when applied with the faradic brush in the form of a counter-irritant. Duchenne saw good effects when the brush was applied at some distance from the painful part.

Subcutaneous injections of morphia give such instantaneous relief even in the severest forms of neuralgia that patients are apt to demand a frequent repetition of the remedy.

Nerve-stretching or neurectomy is the last resort in desperate cases. The relief is sometimes permanent.

Among the numerous empirical remedies in vogue for the cure of neuralgia, there are undoubtedly some that do not owe their reputation to mere coincidence. Arsenic is one of them and should have a fair trial in obstinate cases. Turpentine in large doses is an old remedy in neuralgia. Strychnia is sometimes useful.

THE DIFFERENT FORMS OF NEURALGIA.

Neuralgia of the Trigeminus.

Etiology. This is the most frequent of all the forms of neuralgia. On taking a glance at the distribution of this nerve it is observed that its main divisions and branches pass through many openings of the cranial bones where they are readily subject to pressure and irritation from various causes. The mild type of facial neuralgia is usually caused by atmospheric or malarial influences. The "brow ache" is said to be very common in malarial districts. Draughts of cold air give rise to the so-called "rheumatic" form of trigeminal neuralgia. Search should be made in neuralgia of the head for decayed or filled teeth or a crowded wisdom tooth. Affections of the eyes, ears and nose are well known to be often attended by facial neuralgia. In many cases of obscure origin, constitutional causes may be suspected.

Clinical History. The pain in severe cases of trigeminal neuralgia is extremely violent. Patients can hardly find words enough to express their suffering. Sometimes the slightest physical exertion, such as washing the face, talking, masticating, or a temporary mental excitement suffices to bring on an attack. Various sensations like a feeling of pulling or dragging the skin precede the pain. Some patients frantically rub the face with the hand, which seems to give them relief. The pain is not always confined to the part of the face where it commenced, but frequently shoots towards the occiput, and sometimes to the region of the shoulder and clavicle.

Motor Disturbances, in the form of reflex spasmodic movements, are especially frequent in severe trigeminal neuralgia. The twitching of the facial muscles affects the forehead, the eye and corner of the mouth and causes distortions of the face (tic doloreux). *Vaso-motor* symptoms, which are quite common in severe cases, consist of strong pulsation of the temporal arteries and an increased secretion of the lachrymal and salivary glands. Neuralgia of the opthalmic branch is sometimes complicated with herpes zoster, which, if it affect the eyeball, may have serious consequences.

The most constant *painful points* in the distribution of the opthalmic branch are found in the regions of the supra-orbital notch, on the upper eyelid, and the parietal eminence. In the superior maxillary there is a focus of pain at the infraorbital foramen and another in the region of the malar bone. The most constant painful point in

the distribution of the inferior maxillary branch is at the mental foramen.

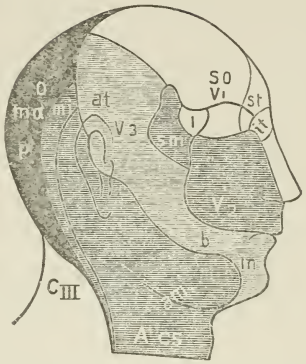

FIG. 22. V_1, V_2, V_3, FIRST, SECOND AND THIRD BRANCH OF THE TRIGEMINAL NERVE (V).

A Region of the anterior.
P Region of the posterior cervical nerve.
C iii The third cervical nerve.
So Supraorbital n.
St Supratrochlear n.
It Infratrochlear n.
1 Lacrimal n.
S m Subcuteneus malar n.
at Auriculo-temporal n.
b buccinalis.
m Mental n.
o ma and o mi Occipital major and minor nerves.
cs Cervicalis superficialis.

Diagnosis. A careless diagnosis may possibly confound trigeminal neuralgia with an affection of the skull, nose or jaw. As a point of distinction between the central and peripheral form of neuralgia of the head or face, it should be remembered that the distribution of the pain in the former is the more extensive.

Prognosis. Recent cases, especially when the pain is confined to a small nerve twig, generally get well in a short time, but curative treatment is not encouraging in inveterate cases occurring in persons advanced in life.

Treatment. A thorough examination of the possible local or constitutional cause of neuralgia should always be made. The teeth should be inspected. A crowded wisdom tooth or an old stump may require removal. Attention should also be directed to the condition of the upper nasal passages in severe pain in the region of the frontal sinus. The neuralgia, from disease of the middle ear, requires local treatment.

Large doses of quinia are indicated in the periodical type of neuralgia. The same remedy sometimes acts well in cases of irregular paroxysms. Arsenic often succeeds when quinia fails. This remedy, usually given in the form of Fowler's solution, should have a fair trial in obstinate cases. The rheumatic form of neuralgia sometimes yields promptly to a full dose of salicylate of soda. The fluid extract of gelseminum in doses from 10 to 20 drops every hour, is highly recommended in neuralgia of the dental nerve. The remedy is to be omitted if relief does not follow the third or fourth dose. Morphia, as may be expected, gives sometimes instantaneous relief in even the severest cases. Mild cases are often cured by one injection. Atropia injections are indicated in very obstinate cases. A syringe full of warm water injected in the region of the painful nerve is said to answer as well as a small dose of morphia. Phenacetine has lately established much reputation in facial neuralgia. Aconitia has been known to give surprisingly good results in cases which had baffled other remedies. The dose is from $\frac{1}{150}$ to $\frac{1}{120}$ grs.

Narcotics may sometimes be avoided in mild cases by the use of sedative or irritating embrocations. A very good application consists of a mixture of chloroform, one

part to three of water. A piece of flannel is soaked with the mixture and applied to the painful part; the flannel should be covered with a thick cloth to prevent rapid evaporation. The external application of atropia is sometimes successful. (See formula.) The veratria ointment well rubbed in until it produces a sensation of tingling is often of benefit.

In very obstinate cases one is tempted to try many remedies that have occasionally been found of service. Among these may be mentioned croton chloral, nitrate of amyl, turpentine, phosphorus, strychnia, chloride of gold.

The general treatment in weak, anæmic persons includes rest, a generous diet, and the administration of iron, cod liver oil and malt extract.

Section of the nerve has been frequently practised with success in supraorbital and infraorbital neuralgia. The risk of the operation is hardly to be considered in desperate cases.

Occipital Neuralgia.

As branches of the upper cervical nerves distributed to the lower portion of the cheek and the supraclavicular region are often simultaneously involved with those that go to the occiput, it is usual to speak of cervico-occipital neuralgia.

Etiology. A very frequent exciting cause of this form of neuralgia is exposure to drafts of cold air when the body is overheated. The neuralgia is not to be confounded with pain located in the same regions from caries of the vertebral bones.

Clinical History. Occipital neuralgia is often bilateral. Usually the pain extends from the back of the head to

the vertex. Sometimes it radiates to the lower jaw and even to the arm. The worst pain, when both sides are affected, is felt between the occipital prominence and the mastoid process. The least movement of the head aggravates the pain. Paroxysms of great severity are almost unbearable if they are of unusual duration.

Vaso-motor disturbances are not uncommon during the greatest violence of the attack. One of the pupils may be contracted, and the ear abnormally red and warm.

The neuralgia is sometimes attended by stiffness of the neck and swelling of cervical glands.

The most constant painful points are found in the nape of the neck where the occipitalis major emerges, and one in the course of the same nerve at the back of the head.

Treatment. Local treatment often suffices in a mild case. The nape of the neck is to be frequently rubbed with an anodyne liniment, and surrounded by cotton, wool or oakum. Patients find much relief from supporting the head. In more severe cases, blistering the nape of the neck with the cantharidal collodion is preferable. A few doses of the salicylate of soda sometimes succeeds in checking the repetition of attacks. In protracted cases, very good results are sometimes obtained from a strong, constant current passed for ten minutes through the mastoid process. Violent paroxysms of pain require morphia.

Brachial Neuralgia.

Etiology. The cutaneous distribution of the ulnar, the median and radial nerves which collectively come from the brachial plexus, may either be affected with isolated or combined forms of neuralgia. The superficial situation

of these nerves exposes them to direct injury and rheumatic influences.

Clinical History. Generally the pain in brachial neuralgia is widely distributed. Sometimes it is continuous or comes on in attacks of great violence. It is usually worse during the night. Movement of the limb aggravates the pain. Not only the arm but also the shoulder and back are often affected with severe shooting pains. Weir Mitchell describes a form of this neuralgia (causalgia) resulting from gunshot wounds. The pain is of an intense burning character.

Among the most constant painful points are 1. The radial point at the lower outer aspect of the arm. 2. A median cephalic point at the bend of the elbow. 3. A shoulder point corresponding to the emergence of the cutaneous branches of the circumflex.

Adventitious symptoms of a vaso-motor or trophic character are sometimes observed. There is a peculiar shining atrophic condition of the fingers (Glossy fingers.) An instructive example came under my notice at the Baltimore City Hospital. A young girl presented herself complaining of violent pains of the left arm in the region of the distribution of the posterior, superior and inferior subcutaneous nerves. The arm was covered on this part with an eruption of large blebs. On the outer side of the bend of the elbow a few black spots were seen that proved to be the ends of rusty sewing needles. The girl confessed afterwards that she had designedly thrust the needles in for the purpose of being admitted into the hospital.

Treatment. The arm should be kept in a sling. Mild cases often yield to sedative or stimulant liniments, but blistering is sometimes necessary. The use of the con-

stant current gives good results in obstinate cases. A full dose of salicylate of soda may be found of service in the rheumatic variety of the neuralgia. The paroxysmal violence of the pain can only be relieved by morphia.

Dorso—Intercostal Neuralgia.

The superficial branches of the seventh, eighth and ninth pairs of dorsal nerves that run in the direction of the intercostal spaces, are generally affected in this variety of neuralgia.

Etiology. Intercostal neuralgia is more often met with in women than in men. It is especially common in factory girls, seamstresses, milliners and nursing women. Females with uterine or ovarian trouble are very prone to suffer from it. The pain usually affects the left side of the chest and is often associated with spinal tenderness. It should be remembered that severe and obstinate side pain attends caries of the vertebræ, aneurism of the thoracic aorta and cancer of the mediastinum.

Clinical History. The pain is felt along the distribution of the intercostal nerves, but quite as frequently it is fixed at a point below the mamma or in the region of the axilla. Coughing, sneezing or even drawing a deep breath aggravates the pain. Herpes Zoster is often associated with this form of neuralgia. The pain frequently persists after the disappearance of the eruption. Severe cases of intercostal neuralgia bear some resemblance to angina pectoris, but the pain is less severe though more continuous, and all the other symptoms characteristic of the latter affection are absent. There is no sensation of constriction of the chest, no embarrassment of respiration nor tumultuous action of the heart. Painful points are

found in the regions where the nerves emerge from the intervertebral foramen, at the bend of the ribs and near the junction of the costal cartilages and the sternum. The course of intercostal neuralgia is frequently protracted in nervous and anæmic females.

Treatment. In recent and mild cases it will often suffice to apply a mustard poultice, and if the pain does not soon yield, blistering may become necessary. The use of the constant current is a good resource in obstinate cases. Speedy relief of the pain is obtained from morphia injections.

Mastodynia (Irritable Breast).

This special form of intercostal neuralgia of the female breast occurs at the period of puberty, and generally in nervous and anæmic women, but it often develops at an advanced age. It is sometimes caused by erosion of the nipples during lactation, and is also liable to appear during pregnancy. Small nodules are often found in the breast.

The breast is so extremely sensitive to the touch that even the pressure of the clothes becomes unbearable. The pain is either continuous or occurs in paroxysms.

Treatment is unsatisfactory. Many cases of mastodynia last for years and appear to baffle every remedy. Patients obtain some relief from a suitable bandage that supports the breast. Friction with chloroform liniment or belladonna ointment, temporarily mitigates the pain. Electricity is occasionally of service. The anode of a stabile constant current is applied to the mamma, and the kathode to the spine. The question of amputating the organ arises when the suffering is so great that it undermines the general health.

Lumbar Neuralgia.

The branches of the lumbar plexus of nerves are relatively seldom affected with neuralgia. The implication of the anterior cutaneous branches of the crural nerve below Poupart's ligament, constitutes *crural neuralgia.* The pain is felt in the inner aspect of the thigh and extends to the calf and foot. Neuralgia of the *obturator* nerve deserves particular attention as it is usually symptomatic of strangulated hernia in the obturator foramen.

Diagnosis. Lumbar neuralgia must be distinguished from affections of the bones and joints in the painful regions. Lumbago is a rheumatic muscular pain and is bilateral.

Treatment is conducted on the general principles that have been described.

Sciatica.

This is one of the most frequent forms of neuralgial The great length and extensive area of distribution of the sciatic nerve subjects it to injurious influences, and besides, the sacral plexus is often involved in intrapelvic disease.

Etiology. Age and sex appear to exert some influence on the causation of sciatica. It is seen most frequently in the middle period of life and is more common in the male than the female. It is also noteworthy that the right leg is oftener affected than the left. In most instances the neuralgia results from strain or the combined effects of cold and dampness. Certain vocations requiring a constant uncomfortable sitting posture may also give rise to the neuralgia. Among the special excit-

ing causes are to be mentioned the gravid uterus, forceps delivery, impacted feces, pelvic tumor or inflammation and psoas abscess.

Clinical History. The pain usually begins in the region of sciatic notch and gradually extends to the buttock, the posterior surface of the thigh, the anterior aspect of the leg and finally to the calf and outer border of the foot. As a general rule the whole area of distribution of the great sciatic is affected. Paroxysms of violent pain which patients describe as of a burning and lightening character are preceded by a feeling of furriness and coldness of the limb. Very often the pain becomes continuous and is worse at night. Flexion of the limb is apt to bring on the pain, and hence the patient adopts a peculiar stiff gait. The concommitant symptoms include tremor and reflex spasms of the calf muscles and a feeling of numbness and tingling of the skin.

One of the tender points corresponds to the part of the trunk of the nerve between the great trochanter and the tuber ischii. There is a fibular point in the superficial course of the peronial nerve and another at the malleolus.

Course. Recent mild cases of sciatica readily yield to judicious treatment, and even chronic cases if there be no irremovable cause, are frequently cured, but relapses are common. Some degree of weakness and stiffness of the limb often persists for weeks and months after recovery.

Diagnosis. The distinction between sciatica and lumbago is not always an easy matter. The pain in lumbago is more diffuse and generally confined to the buttock and is increased by movement and pressure. A careful examination will guard against the error of confounding sciatica with hip-joint disease. The real cause of sciatica may

remain concealed for a long time if there exists an abdominal aneurism or intrapelvic disease.

Treatment. Old standing cases of sciatica which resist ordinary treatment sometimes yield with astonishing rapidity when the true cause is discovered and treated accordingly. The most favorable of chronic cases are those in which the exciting cause is removable, such as habitual constipation, hæmorrhoids, varicose veins and tumor.

In sciatica of recent date it is of the first importance that the patient keep in bed. Warm applications are very grateful. A hot vapor-bath is especially of service in "rheumatic" cases. Such a bath can readily be improvised by heating bricks, wrapping them in wet woolen cloths and placing them between the lower limbs and outside of them. The escape of the steam is prevented by a thick covering of blankets.

Protracted cases often yield to blistering. A good plan is to cross the affected part of the nerve with small blisters about two inches apart.

Sciatica is more frequently cured by the electric treatment than any other form of neuralgia. I use a gradually increasing constant current. The anode is allowed to rest quietly for at least two minutes at a time on different parts over the path of the nerve, and the kathode is applied to the back. Painful spots are preferred for the anodic application.

Internal remedies rarely give satisfaction. Quinia and salicylate of soda may occasionally be found of service. Strychnia has also been recommended. Turpentine in large doses is an old remedy in sciatica. If iodide of potassium is successful in an inveterate case of the neuralgia, it was probably of syphilitic origin. After

every other remedy has failed, nerve-stretching is the last resort which although it may not give permanent relief, is nevertheless a justifiable procedure.

Neuralgia of the Genitals.

The external genital organs are but rarely subject to neuralgia. Spermatic neuralgia or "the irritable testicle" as it was formerly called, demands particular attention. It is marked by violent paroxysms of pain, beginning in the testicle and extending to the spermatic cord. During an attack, the testicle retracts and is very sensitive. This is a most obstinate and harassing species of neuralgia, little amenable even to palliative treatment, and resisting narcotics and electricity. Castration appears to offer the only means of relief.

Weir Mitchell has described "a neuralgia anno-peronialis" occurring among masturbators and smokers.

Neuralgia of the region of the rectum is sometimes seen in persons much reduced in health, especially from malaria cachexia. Dr. Neftel of New York saw good results from the use of the constant current in such cases.

Coccygodynia.

This affection is characterized by severe pains in the coccygial region. It is aggravated when the patient sits or walks, and during defecation. This trouble generally occurs in women, and is probably caused by strain or injury in labor. It is a most intractable affection, and the removal of the coccyx has been resorted to with good success. Before deciding on such a measure, which is only advisable in a desperate case, a fair trial should be given to faradization. One of the electrodes is introduced into the rectum, and the other is applied to the sacrum.

Neuralgia of the Joints.

It is well known that Sir Benjamin Brodie first directed attention to the occurrence of a painful affection of joints in hysterical women. Recently the subject has been studied by Esmarch, whose observations convinced him that cases of this kind in which generally the hip joint is affected, illustrate the existence of an "articular neurosis." The painful joint is exempt from gross anatomical change. Weir Mitchell, in his interesting chapter on the "Mimicry of Disease," refers to the morbid influence of concentrated attention on a particular part of the body on account of some trivial hurt. Hysterical patients are especially prone to the development of a neuritic affection from such a cause.

A case in point came under my observation. The patient, a young married woman fell against her side, which greatly alarmed her, and rendered her very anxious concerning the consequences. A few weeks later she took to her bed, in which she remained for eight months. I found her lying in the position which, I was informed, she had assumed from the beginning. The left leg was extended and slightly rotated inwards. This is the limb that gave her so much trouble after the fall. She described to me, with tears in her eyes, the unbearable suffering she had since undergone. She begged me not to touch the sore limb, as the pain it would cause was unendurable. The slightest movement, she said, the merest touch of the hand, would give her the most excruciating pain. Those about her had so much given away to her entreaties not to be disturbed that they neglected to attend to a very bad bed sore that had developed in the sacral region. Her mother had given up housekeeping so as to be constantly with her daughter. I observed no swelling about the knee or hip joint where the patient located the pain. She appeared to be somewhat emaciated, though her appetite was fairly good and she got sufficient sleep. An attempt to move the limb was out of question, as the patient vehemently protested against it, and would not listen to any reasoning or persuasion. I suspected the character of the affection, and stated my opinion to the attending physician. Of course we could not come to any definite

decision until the limb was more thoroughly examined. The patient absolutely refused to take an anæsthetic. We carried out a conspiracy, with the help of the mother and nurse, and removed her to a lounge amidst her loud wailing and protestations. After the bed sore was dressed, I succeeded in rotating the limb with considerable ease, and to flex the knee in spite of the cries and opposition of the patient. There was now no room for doubt that the leg was healthy, barring the neurosis of the joints. Galvinization constituted subsequently the chief treatment. Gradually the patient was induced by coaxing and occasional firm language to allow her limb to be handled more freely, and to make a few steps with the assistance of her mother and nurse. By and by she learned to walk on crutches. When I paid her my last visit, I found her quite well and walking without any support.

NEURITIS.

Etiology. Inflammation of peripheral nerves is observed in injury from gunshot wounds, laceration and contusion. Nerves in the neighborhood of diseased vertebræ and joints may become involved. Neuritis also occurs as a rheumatic or a syphilitic affection, but often no cause can be assigned.

Anatomical Changes. Generally the neurillema and nerve fibre are conjointly affected. The nerve appears swollen, its capillaries are enlarged and hemorrhagic spots are seen. Disintegration of the medullary sheath and nerve fibre develops in severe cases. Numerous cellular elements accumulate in the neurillema, which is gradually thickened by the formation of connective tissue, and may finally lead to destruction of the affected nerve. An ascending *interstitial neuritis* sometimes occurs which starts from the seat of the lesion. In mild cases regeneration of the nerve takes place. Simultaneously with the degeneration of a motor nerve, nutritive changes of the corresponding muscles ensue that causes atrophy

This atrophy is permanent if the nerve fibre has completely degenerated.

Fig. 24.

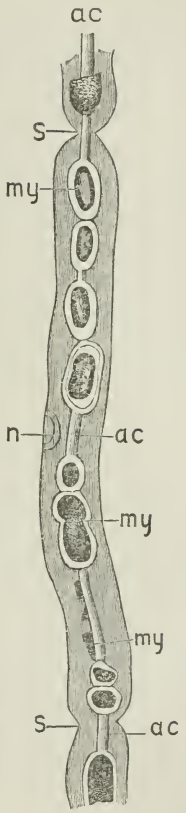

Fig. 25.

Fig. 24. Normal Medullary Nerve Fibre. (Ranvier.)

Fig. 25. Alteration of Nerve Fibre After Section.

ac Axis cylinder.
e Ranvier's nodes.
sch Sheath of Schwan.
my Masses of myeline.
S Strangulation of the medullary sheath.
my Masses of myalines.
n Nucleus detached from the sheath of Schwan.

Clinical History. Considerable febrile excitement marks the onset of acute neuritis. Intense pain is felt in the course of the nerve and its distribution. The parts are exceedingly painful to the touch, and the swollen nerve can be felt through the skin. There is often a subjective sensation of numbness of the affected part, but the motor disturbance that finally makes its appearance is of more importance. A form of atrophic paralysis is established after the subsidence of the acute symptoms. The muscles show the reaction of degeneration. There are mild cases which do not advance to this condition. Others take a tedious chronic course, with eventual loss of function of the part. There is a group of degenerative neuritis, of which the following are the most important.

Multiple Neuritis.

This form of neuritis is chiefly observed to affect motor nerves of the limbs. At a late stage of the inflammation this process extends to the finest nerve filaments that are distributed to the muscles. The nerve fibres are thickened and nodulated in different places (neuritis nodosa). The sarcolemma is increased and infiltrated with fat. This morbid alteration does not extend to the nerve roots. Further investigation will probably disclose the fact that multiple neuritis is the pathological condition underlying the atrophic paralysis of lead and arsenic poisoning.

Symptoms of sensory disturbance occur at the beginning, consisting of tearing pain in the extremities, especially in the hands and feet and attended by paræsthetic sensations about the joints. Motor disturbances develop next, characterized by a flaccid condition of the

muscles, diminution and final loss of the electric reaction, as in all forms of peripheral paralysis. The neuritis in the first stage is marked by fever and violent pains in the limbs. Restoration may take place, but in severe cases an atrophic paralysis develops.

Alcoholic Neuritis.

Chronic alcoholism may give rise to forms of paralysis which are now considered to be dependent on the development of multiple neuritis. The main clinical feature of the disease is atrophic paralysis of the inferior extremities. In some cases additional symptoms appear that bear a close resemblance to sclerosis of the posterior columns of the spinal cord. The disease begins with violent tearing pains in the legs and sometimes in the arms. Gradually, an uncertainty of the gait is established. In well-marked cases there is also a paretic condition of the limbs. The affected muscles show wasting and abnormal electric reaction. There is early abolition of the patellar reflex. The cutaneous sensibility is impaired. It is evident from the character of these symptoms that their resemblance to those of loco-motor ataxia is complete. If, however, atrophic paralysis coëxists, then spinal disease is excluded. In the alcoholic affection there is generally no immobility of the pupils; the girdle sensation is absent and there is no irritation of the bladder.

Treatment. Causal treatment is of the first importance in traumatic neuritis. The antiseptic treatment is indicated in the case of infectious wounds. Primary acute neuritis call for measures to subdue the inflammation. If the arm is affected it should be supported in a sling.

A poultice of crushed ice is very grateful to the patient. Salicylate of soda may be given from the beginning, but to allay the severity of the pain, morphia is often required. The patient should not be allowed to leave the bed too early. The paralysis of chronic neuritis is best treated with electricity. The following method may be adopted: The kathode of a strong constant current is placed on the vertebral column in a position corresponding to the nearest part of the affected nerve. The anode is applied for five minutes daily to paralyzed muscles. The interrupted current is also useful. This treatment may be advantageously assisted by massage and warm baths.

Neuromata.

New growths, differing in histological structure, develop in nerves as they do in other organs. True neuromata consist of newly-formed nerve tissue; false neuromata are fibromatous. The so-called "painful tubercle" is usually of the latter species. It occurs in different parts of the body, and can often be felt as a small nodule under the skin. True neuromata sometimes develop in the stump of an amputated limb. The cause of these growths is unknown. In some individuals a numerous crop of little nodules scattered over the whole body make their appearance. These multiple neuromata do not give rise to symptoms.

Many cases of true neuromata are attended by a violent intractable pain. The only permanent cure is their extirpation. Temporary relief is obtained from narcotics.

CHAPTER V.

PERIPHERAL PARALYSIS.

Under the head of peripheral paralysis, in contradistinction to paralysis of cerebral or spinal origin, are included those individual forms of motor and sensory paralysis, which result from injury or disease of peripheral nerves. The general character of peripheral paralysis may be summarized as follows:

1. Diminution or loss of muscular power in consequence of interruption of the conducting motor path, limited to the affected nerve.

2. Impairment or abolition of sensation in injury or disease of mixed nerves. The disturbance of the cutaneous sensibility is usually insignificant and is often absent.

3. Vaso-motor and trophic disturbances, especially atrophy of the paralyzed muscles, arrested growth of bones, affections of the joints and changes in the skin. The latter are indicated by dilatation of the superficial vessels, local elevation of the temperature, cyanosis and coldness of the surface.

4. Abnormal condition of the electric excitability.

Etiology. In reference to the exciting causes, we distinguish the traumatic, the rheumatic, the toxic and post-febrile forms of peripheral paralysis. The traumatic variety also includes the paralysis from prolonged com-

pression of a nerve, as may occur in cases where the patient, in a state of intoxication, had been sleeping on a hard substance, with his arm bent behind his back. Tight bandaging and ill-constructed crutches may produce a similar effect. Paralysis of one or both of the upper extremities is sometimes caused in new born infants during the operation of version.

Rheumatic peripheral paralysis most frequently affects the face, and less frequently one of the limbs. These parts are especially exposed to rheumatic influences.

The toxic forms of paralysis chiefly include lead palsy and arsenical paralysis.

Among the post-febrile forms of peripheral paralysis, the one of greatest practical importance is diphtheric paralysis.

Anatomical Changes. In the traumatic form of paralysis and probably also in severe cases of rheumatic origin, the anatomical changes are of the nature of an interstitial neuritis with a tendency to undergo the process of degeneration. There are other forms of paralysis usually considered to be of a peripheral character, whose pathology has not yet been satisfactorily determined.

VARIETIES OF PERIPHERAL PARALYSIS ACCORDING TO THEIR DISTRIBUTION.

Paralysis of the Motor Branch of the Trigeminus.

This species of paralysis is very rarely of local origin. In partial paralysis of the masticatory muscles, the act of deglutition is difficult. In complete bilateral paralysis, mastication is impossible. The jaw hangs down and the muscles gradually atrophy.

Treatment. Galvanization deserves a trial, but if the paralysis is of central origin, it requires causal treatment.

Facial Paralysis.

Etiology. This is the commonest of all forms of peripheral paralysis. The causes of the paralysis in their order of frequency are: 1. Exposure to drafts of cold air, as when a person sits near an open window in a railroad car on a cold, windy day. This is the rheumatic form of facial paralysis. 2. Disease of the ear, when the facial nerve is involved in its passage through the Fallopian canal. 3. Disease at the base of the brain where the nerve is implicated after its emergence from the brain. Facial paralysis is therefore frequently observed in intercranial syphilis, and is often associated with paralysis of some of the ocular muscles. 4. Swelling of the parotid gland or its removal in operations. 5. Forceps delivery. The infant is often unable to take the breast. In very mild cases, the paralysis may only be discovered when the child cries, which produces distortion of the face.

Clinical History. Facial paralysis is easily recognized in consequence of the immobility of the muscles of expression of one side of the face. On the paralyzed side no wrinkles are visible on the forehead when the patient attempts to frown. The eye on the affected side cannot be voluntarily closed. This permits the entrance of dust which may cause conjunctivitis. The labio-nasal fold is effaced; the ala nasi is flattened; the cheek "hangs" and flaps during inspiration, and bulges out during expiration. The mouth is drawn over to the healthy side; this becomes very apparent when the patient speaks, laughs or cries. He finds it difficult to whistle, blow or spit. The saliva

escapes from the half-opened mouth. Morsels of food lodge between the gum and cheek, and mastication is rendered difficult on account of the flaccidity of the cheek. The tears run down the face, as the paralysis of Horner's muscle prevents their escape into the lachrymal canal. Speech is indistinct from imperfect movement of the lips. Immobility of the soft palate on the affected side is observed in some cases, but the position and movements of the uvula vary too much in the healthy condition for this symptom to be of any significance.

Impairment of the sense of taste exists in cases in which the chorda tympani nerve is implicated. This nerve accompanies the facial for a short distance, and sends gustatory fibres to the lingualis. Dryness of the mouth, due to diminished secretion of saliva, is dependent on the same cause. Disturbance of hearing occurs where the paralysis originates from aural disease.

The abnormal electric reaction in facial paralysis is of much importance in regard to prognosis. Erb distinguishes three different conditions of the electric excitability:

1. The mild form, which is the one most commonly met with. The electric reaction is perfectly normal. Recovery may be expected within two or three weeks.

2. The middle form. The electric reaction is diminished, but not lost. At the expiration of about three weeks there is an increase of the galvanic excitability of the muscles. The anodal closure contraction (A C C) is greater than the kathodal closure contraction (K C C). Recovery usually ensues at the end of six weeks.

3. In the severe form, there is loss of the faradic and galvanic excitability of the nerve and loss of faradic excitability of the muscles. The reaction of degeneration

is complete, and the prognosis is unfavorable. Recovery, in cases which are at all cured, is very tedious. At a late stage there is often spasmodic contraction of the muscles inserted at the mouth when the patient smiles or laughs, or is engaged in animated conversation.

Course and Prognosis. In the rheumatic variety of facial paralysis much depends, as already mentioned, on the result of the electrical examination in regard to prognosis. If the electric excitability remains normal at the end of three weeks, recovery will speedily be established. Manifest reaction of degeneration offers little hope of curability. The paralysis from ear trouble disappears with the latter. In basal tumor, if not due to syphilis, it is absolutely incurable. The facial paralysis in infants after forceps delivery passes off quickly.

Diagnosis. Slight cases of facial paralysis are only recognizable during the acts of laughing and crying. The distortion of the mouth simulates spasm of the healthy side. In general, the symptoms of facial paralysis are so manifest that mistakes can hardly happen. Of greater importance is the discrimination between peripheral and central facial paralysis. The differential diagnosis must consider the etiology and concomitant symptoms of the individual case. External injury, compression, exposure or aural disturbance indicate the existence of peripheral paralysis. Imperfect closure of one eye points the same way. In the paralysis of cerebral origin there is seldom immobility of the muscles in the region of the forehead and the eye. Complication with paralysis of other cranial nerves, or especially the co-existence of hemiplegia, signifies a central lesion. The electric excitability is not usually abnormal in the latter form of paralysis.

Treatment. The indications of causal treatment in relation to affections of the parotid gland, otitis or syphilis require the first attention. In all other cases there is no remedy equal in efficacy to electricity, though uniform or brilliant results must not be too confidently expected. In paralysis of recent date, it is recommended to apply a weak, stabile galvanic current to the auriculo-mastoid fossa for two or three minutes every other day. But the chief method at a later period is to place the anode in the same fossa, and gently stroke the paralyzed nerves and muscles with the kathode. Faradization of the muscles may also be of service in tedious cases. Injections of strychnia are disappointing.

Paralysis of the Sterno-Cleido Mastoid.

The head is inclined to the healthy side, and the chin is raised and directed towards the opposite side. Motion in the opposite direction is difficult. The deformity is temporarily rectified by passive movement of the head. This cannot be accomplished in spasmodic contraction of the muscle.

Paralysis of the Trapezius.

The shoulder sinks downwards and forwards, and the act of "shrugging the shoulder" is impaired. Raising the arm above the horizontal position is difficult. The normal condition of the scapula in relation to the vertebral column is altered.

Simultaneous paralysis of the sterno-cleido mastoid and the trapezius is often seen, as both muscles are supplied by external branches of the spinal accessory nerve.

Paralysis of the Pectoralis—Major and Minor.

Isolated paralysis of these muscles is extremely rare. The anterior thoracic nerve is distributed to these muscles. Abduction of the upper arm is impossible, and the hand cannot be placed on the shoulder of the opposite side.

Paralysis of the Rhomboidii and Levator Angulæ Scapulæ.

The shoulder-blade cannot be approximated to the vertebral column, nor elevated. This form of paralysis can only be recognized when the trapezius is also effected.

Paralysis of the Latissimus Dorsi (*sub-scapular nerve*) usually results from lifting heavy weights. The hand cannot be placed on the sacrum. There is no deformity observed when the body is at rest.

Paralysis of the Outward and Inward Rotators of the Humerus.

Movements with the teres-major and sub-scapular muscles on the opposite side of the body are impaired. Rotation of the arm outward is abolished when the teres-minor and scapularis are paralyzed. This form of paralysis causes marked interference with the acts of writing and sewing.

Paralysis of the Serratus Magnus.
(*Thoracic Longus Nerve.*)

It is observed, when the body is at rest, that the lower angle of the shoulder-blade is somewhat approximated to the vertebral column and slightly inclined upward and outward. The patient cannot raise the arm above the

horizontal line, and the indentations of the muscle on the side of the chest do not become conspicuous. If the scapula is pushed forward, the patient is enabled to raise the arm, and if the arm is extended, the very characteristic symptom of paralysis of the serratus becomes apparent. We now observe the wing-like projection of the inner border of the scapula, so that a deep fossa is formed, in which the hand can readily touch the inner surface of the scapula.

Paralysis of the Deltoid.

(*Circumflex Nerve.*)

The arm cannot be raised to the head, and the shoulder appears sunken. This form of paralysis can be easily distinguished from anchylosis of the shoulder by passive movement, which is impossible in the latter affection.

Paralysis of the Biceps and Brachialis Anticus.

(*Musculo-cutaneus Nerve*)

is rarely isolated. It interferes with flexion of the forearm when in supination. In pronation, the flexion is assisted by the action of the supinator longus.

Paralysis of the Muscles Innervated by the Musculo-spiral Nerve.

Peripheral paralysis in the distribution of this nerve is of frequent occurrence from injury and exposure. This nerve is especially prone to be affected in chronic lead poisoning; in pressure from badly-fitting crutches and tight bandaging of the arm, and also from compression of the nerve, where it curves around the humerus. The latter causes produce a mild form of paralysis.

Paralysis of the Triceps is unusual. It is recognized by inability to extend the arm; but in the experiment the arm must be raised so as to exclude the weight of the forearm.

Paralysis of the Extensors of the Forearm causes the "wrist drop." The hand hangs down in the flexed position and has lost its "grasp." Dorsal flexion of the hand is impossible. Its lateral movements are difficult. The fingers cannot be extended nor spread apart, but the extension of the terminal phalanges is found to be normal if the first phalanges are supported. Extension and abduction of the thumb is abolished. The flexed forearm can be supinated by the biceps, but if the forearm be extended and pronated it cannot be supinated. The characteristic prominence of the supinator longus is wanting if the forearm is forcibly extended, whilst the patient tries to resist the attempt by fixing the semi-flexed forearm in the pronated position.

The electric excitability of the paralyzed muscle is normal in the early stage and continues so in mild cases. The reaction of degeneration becomes manifest on the occurrence of atrophy. Impairment of the cutaneous sensibility is insignificant, but a feeling of numbness or creeping often ushers in the paralysis.

Ulnar Paralysis.

Isolated ulnar paralysis is relatively of unfrequent occurrence. It is chiefly caused by injury of the elbow, the wrist or the ball of the little finger. Implication of the flexor carpi-ulnaris interferes with complete flexion and lateral movement of the hand towards the ulnar side. Flexion of the last three fingers is imperfect.

The movement of the little finger is totally abolished. Paralysis of the lumbricalis prevents the spreading of the fingers. Flexion of the primary phalanges as well as extension of the terminal phalanges, is impossible, in consequence of paralysis of the interossii. A very characteristic deformity of the hand develops, which is rendered more unsightly by the grooves of the atrophied interossii on the back of the hand. This peculiar position of the hand gives the appearance of the "claw-like hand" (main en griffe).

Combined Paralysis of Muscles of the Arm.

Injuries affecting the distribution of the different nerves proceeding from the brachial plexus may give rise to various combinations of paralysis of the upper extremity Total paralysis of the plexus is caused by severe contusion in the regions of the neck and shoulder. A form of combined paralysis is described by Erb involving the deltoid, the biceps, the brachialis anticus and the supinator longus. Duchenne reports similar cases of paralysis in new born infants, caused by shoulder delivery. The arm in such cases hangs down by the side; the forearm cannot be flexed, but the hand and fingers are not affected.

Treatment of Paralysis of the Upper Extremity. It is only in rare instances that a causal treatment is possible. The chances of cure in traumatic cases are not favorable. Those of a rheumatic origin are more amenable to treatment. The mildness or severity of the paralysis is best judged by the condition of the electric excitability. The greatest reliance in improving the muscular power must be placed in electric treatment, and the more recent the case the better is the prospect of cure. A good method is to gal-

vanize Erb's motor point. Each muscle should also be stimulated by stroking or dabbing it with the kathode. If the test for the reaction of degeneration gives early anodal closure contractions, it is preferable to excite the muscle with the anode, while the kathode is placed on a distant part. Faradization proves useful in exciting reflex action. In all cases patience and perseverance in continuing the electric treatment is necessary if any benefit is to be achieved. The more obstinate the case the more persistent should be the treatment. Massage and the use of liniments should not be neglected.

Peripheral Paralysis of Muscles of the Inferior Extremity.

This form of paralysis occurs in injury of the thigh, disease of the vertebræ, compression from pelvic tumor and in psoas abscess. It is also to be observed that defective innervation of muscles inserted in the vertebral column interferes with walking, in consequence of the insecure equilibrium of the body.

Paralysis of the crural nerve. The symptoms in this form of paralysis are due to the implication of the iliopsoas and the quadriceps extensor muscles. The thigh cannot be flexed on the pelvis, and the trunk cannot be raised from the recumbent position. Anæsthesia is sometimes observed in the lower half of the anterior aspect of the thigh and along the course of the saphenus nerve.

The characteristic symptoms of paralysis of the *obturator nerve* is defective abduction of the thigh. The patient is therefore unable to cross one leg over the other.

Paralysis of the gluteal nerves causes impaired abduction of the thigh and difficulty of rotating it inwards. The

trunk sways from side to side in mounting and descending steps.

Paralysis in the region of the thigh results from injury and disease of the vertebræ, pelvic tumor, hard labor and rheumatic influences. Isolated paralysis of the different branches of the sciatic nerve may occur from any of these causes. Paralysis of the foot is the most common.

In paralysis of the peroneal nerve there is inability of voluntary dorsal flexion of the foot. The tips of the toes and the outer edge of the foot first touch the ground in walking, as the abduction of these parts is abolished. A similar condition is characteristic of infantile spinal paralysis.

Paralysis of the tibial nerve completely abolishes plantar flexion. It is impossible for the patient to rise on his toes. Secondary contraction of the calf muscles gradually develops *talipes calcaneus.* A claw-like appearance of the foot is caused by atrophy of the interossii.

Paralysis of the trunk of the sciatic nerve causes, in addition, paralysis and atrophy of the foot, and inability to flex the leg backward on the thigh. This is due to the implication of the biceps, semi-membranous and semi-tendinous muscles. Walking is still possible if only one extremity is affected, as the quadriceps, which usually escapes, stiffens the limb.

Vaso-motor and trophic symptoms are quite frequent, as shown by the cyanosis, coldness of the skin and atrophy of the muscles.

Impairment of the electric excitability is manifest, as in all cases of peripheral paralysis.

Treatment accords with the same principles observed

in the management of peripheral paralysis of the upper extremity.

Lead Paralysis.

Chronic lead poisoning occurs among type-setters, type-founders, house painters, potters, workers in white-lead factories, and among all artisans who expose themselves to the introduction of small quantities of the metal or its salts. The paralysis usually makes it appearance subsequent to attacks of colic.

As lead palsy belongs to the class of atrophic paralysis, it may be inferred that the toxic action of the metal either produces degenerative changes in the gray substance of the cord or analogous changes in peripheral motor fibres. Certainty in regard to this pathological question has not yet been obtained, but recent investigations tend to show that the anatomical change of nerve fibres is the primary condition.

Clinical History. Lead paralysis is a bilateral affection. In the large majority of cases the paralysis is limited to the extensor muscles of the forearm, but the triceps and the supinator longus remain remarkably exempt. The conspicuous symptom is the "wrist drop." Occasionally the muscles of the upper arm are also implicated. Lead palsy of the lower extremities is rarely seen. Atrophy of the affected muscles develops in all severe cases, and the reaction of degeneration is observed. Tremor of the paralyzed part is sometimes seen in aggravated cases, and attended by anæsthesia.

Treatment. Recent cases of lead paralysis are often promptly cured by the frequent use of warm sulphur baths and the employment of electricity as indicated in

all forms of peripheral paralysis. Iodide of potassium is believed to promote the elimination of the lead poison from the system. Very aggravated cases, that manifest the reaction of degeneration and show much muscular atrophy, do not permit of a favorable prognosis.

Arsenical Paralysis.

This form of paralysis is less frequently met with than lead palsy. It succeeds the symptoms of acute arsenical poisoning. The paralysis affects, in the larger number of cases, the inferior extremities. Severe pain is felt in the limbs and sacrum, attended by anæsthesia. These symptoms are characteristic of arsenical paralysis. Atrophy of the paralyzed muscles finally ensues. It is undetermined whether the anatomical change underlying the paralysis is a neuritis or myelitis. Recovery is sometimes rapid and is hastened by electricity.

Diphtheric Paralysis.

The paralysis of diphtheria usually develops in the course of the third or fourth week after the termination of the primary disease. Paralysis of the soft palate is the first symptom that attracts attention. This is indicated by the nasal twang of the voice and difficulty of deglutition. The ocular muscles are sometimes implicated, causing ptosis, strabismus or mydriasis. A paretic condition of the lower extremities is occasionally observed. Sensory disturbances always accompany the paralysis, especially obtuseness of the mucous membrane of the lips, tongue and cheeks. But the most serious occurrences in diphtheric paralysis relate to irregularity of the heart's action and embarrassment of the respiration. Sudden death may happen from fatal syncope and asphyxia.

Disappearance of the paralysis usually takes place in the course of a few weeks if it is limited to the palate; even severe and protracted cases tend to recovery. The general treatment tends to improve the patient's strength, for which quinia, iron and codliver oil constitute the approved remedies. Electric treatment is of much benefit in acting favorably on the paralyzed muscles.

Reflex Paralysis.

By this term is meant a paralytic affection dependent on an irritation existing in a remote organ. The most common example of such a form of paralysis is the occurrence of paraplegia in urinary disease. Intestinal and uterine troubles may also give rise to this type of paralysis. Leyden attributes the paralysis in such cases to an ascending or migrating neuritis which starts from the diseased part and secondarily affects the spinal cord. Treatment must be directed to the cure of the primary cause, but the paralysis may persist and require special treatment.

CHAPTER VI.

VARIETIES OF LOCAL SPASM.

Spasm of the Muscles of the Eyeball.

Tonic spasm of the internal rectus, which is the most frequent of the local spasms of the ocular muscles, is recognized by the presence of strabismus. It is often caused by long standing paralysis of the external rectus.

Nystagmus is a bilateral affection. It consists of clonic spasm of the muscles of the eyeballs, and is especially noticeable when the patient looks at remote objects. The eyes are affected with oscillatory movements. Nystagmus is associated with various local affections of the eyes. It is also often seen in acute meningitis, and is a frequent symptom in multiple sclerosis of the brain.

Spasm of the Masticatory Muscles.

Trismus is a *tonic* spasm of the muscles of mastication. It is rarely seen as an isolated affection, but constitutes a prominent symptom of tetanus, and occurs in hysteria. The closure of the jaws is caused by the firm contraction of the masseter muscles (lockjaw).

In the *clonic* spasm of the masticatory muscles the lower jaw is constantly moved in the vertical direction, and gives rise to chattering of the teeth.

Both varieties of local spasm probably depend on reflex action, which may be excited either by an irritation in the jaw itself, such as decayed teeth, or by an affection in a remote organ.

Treatment aims at the removal of the discoverable cause. Difficulty of feeding must be overcome by introducing food through the nose by means of a small œsophageal tube. Galvanization is sometimes very serviceable. Hypodermic injections of morphia or atropia and the internal use of the bromides, arsenic and zinc may be tried in succession in obstinate cases.

Facial Spasm (Convulsive Tic).

In the absence of any recognizable cause of this practically most important form of local spasm, we can only conjecture the existence of a hereditary or neuropathic tendency that predisposes to its development. Convulsive tic is sometimes caused by direct reflex action, as in neuralgia of the fifth nerve, or indirectly under the influence of a source of irritation in a remote organ, as in uterine disease. Facial spasm has also been known to occur immediately after violent emotional disturbance. Perhaps in many cases the source of irritation is central. The acquired habit of grimaces, observed in children through imitation, is a tonic spasm of facial muscles.

Clonic spasm of the face is usually a bilateral disease. It occurs periodically, and may be excited by the acts of mastication, talking or a mental impression. The movements produce contortion of the features. The contraction in severe cases may also involve the tongue and the muscles of the neck. There are patients who constantly make the strangest grimaces, while in others they recur

at irregular periods. The affected muscles carry out normal movements during the intervals and are free from pain or other sensory disorders. The so-called "tricks" are of the nature of clonic spasm.

Partial spasm of the eyelids either causes constant winking (nictitating spasm); or the entire orbicularis palbebrarum may be affected with tonic and clonic spasm. The contractions cause firm closure of the eyes, that may last for minutes or hours (blepharospasm). The spasmodic paroxysms are as a rule bilateral. Very often the spasm is excited by affections of the eyes, or tic doloreux, but often no cause is discoverable. The involuntary grin (risus sardonicus) is due to convulsive movements of the muscles supplied by the malar and labial twigs of the facial nerve.

Facial spasm is not easily remedied by treatment, unless the exciting cause of the reflex movements be removed. Resection of the supraorbital nerve is recommended if obstinate neuralgia of this nerve is at the bottom of the spasm. Favorable results from this operation are reported. Improvement of the spasm has been observed from the application of Paquelin's thermo-cautery to the cervical vertebræ. Electricity deserves a fair trial before resorting to severe measures. The anode of a stabile constant current is to be applied to the affected muscles and brought in contact with the tender points of the nerve, which should be searched for. In using the interrupted current it is recommended to begin with a weak strength and gradually to increase it. Some benefit is also claimed from the use of the bromide of potassium in large doses.

Lingual Spasm.

This is rarely an independent affection. The tongue is commonly affected in epileptic and hysterical spasms. When occurring as an isolated condition it interferes with speech and respiration. The tongue during the spasm is thrown backwards and up against the hard palate. Relief is afforded by pulling the tongue forward with a forceps. To allay the spasm it may become necessary to use chloroform.

Spasm of the Œsophagus.

Spasmodic stricture of the œsophagus is usually met with in hysterical women, and is often so persistent as to simulate organic disease. The so-called "globus hystericus" is supposed to be a spasmodic affection of this kind. The œsophageal tube can be passed with little difficulty in the hysterical disorder. An anæsthetic may be required to overcome the obstinacy of the spasm.

Spasm of the Respiratory Muscles.

Spasmodic attacks of the glottis occurring in children is usually described under the name of *laryngismus stridulus*, or "false croup." This affection often coexists with infantile convulsions. The exciting cause in many cases is difficult dentition or derangement of the digestive organs. Ricketty and weakly children are especially prone to attacks.

The paroxysm comes on suddenly without any warning. It consists of complete closure of the glottis. Respiration is arrested and suffocation appears imminent. At the same time the fingers are pressed against the palm of the hand and the lower extremities are extended.

Towards the termination of the spasm, which lasts but a few seconds, respiration is gradually reëstablished, and relief of the glottis is announced by a sonorous, crowing inspiration, provided there is no occurrence of general convulsions. Death from asphyxia during one of the attacks often happens.

It is seldom that the physician arrives in time to witness the spasm. He usually finds the little patient in a warm mustard bath, which is probably as good as anything that can be done for the moment.

Complicated Spasms of the Respiratory Muscles are seen only in hysteria. They consist of fits of laughing and crying, a brassy cough, gurgling and squeaking sounds, snuffling, fluttering at the heart and extreme rapidity of the respiration.

Spasm of the Diaphragm (*Singultus*).

Ordinary hiccough is a sudden contraction of the diaphragm. Persistent hiccough is often a hysterical symptom. It is sometimes exceedingly troublesome and even exhausting. The worst case of hiccough I have ever seen occurred in a young healthy man, who had been two days under treatment without receiving relief. It happened to him after having taken a hasty lunch at a railroad station. I found him sitting in an arm-chair in a reclining position, and with both hands firmly grasping a stick to support himself. The hiccough was incessant and violent. The spasm was promptly checked by several ten-grain doses of musk. Hiccough is an ominous symptom in severe affections of the bowels, and in peritonitis. Persistent hiccough isusually arrested by morphia or a

few whiffs of chloroform. Galvanism is also useful. One pole is to be applied to the side of the neck and the other to the epigastrium.

Spasm of the Muscles of the Neck.

The mild form of "wry neck" is usually of rheumatic origin. The spasm is a tonic unilateral contraction of the sterno-cleido mastoid. The more severe variety of "torticollis spastica" is seen in disease of the cervical vertebra. Frequently several of the muscles of the neck are simultaneously affected.

In unilateral spasm of the sterno-cleido mastoid, the head is turned to the opposite side and the chin is raised. If the trapezius is similarly affected the head is turned backward towards the shoulder of the same side.

In *spasm of the splenius*, whether isolated or combined with spasm of other muscles of the neck, a firm ridge is seen beneath the outer portion of the trapezius.

Bilateral Clonic Spasm,

in which the deep muscles of the neck are affected, usually occurs in children. It causes nodding or rotatory movements of the head, which may attain to great severity.

General Treatment. All cases of chronic spasm of the muscles of the neck offer great difficulty to treatment. The greatest amount of relief is obtained from mechanical apparatus that supports the head, as the spasm usually ceases when the head is at rest. Electric treatment is occasionally of benefit. Either current may prove useful by methods which experience in the special case may indicate. A resort to the red-hot iron to the back is

recommended by some observers. Internal remedies appear to be of little use. Morphia injections may become indispensable to afford the patient some respite from the painful or constant spasmodic contractions.

Spasm of the Muscles of the Upper Extremity.

Convulsive movements of the muscles of the upper extremity are usually seen in central disease and in hysteria. The tonic spasm of the arm in brachial neuralgia is a reflex action.

Tonic spasm of the *rhomboidii* causes a peculiar position of the scapula. Its inner border runs obliquely upward and outward, and its lower border approximates the vertebral column. The arm cannot be raised in the upright direction, which also is seen in paralysis of the serratus; but in the latter affection the scapula stands off from the spine.

Tonic spasm of the *pectoralis major*, the *deltoid* and the *latissimus dorsi* and analogous isolated contractions of muscles of the back and shoulder are of rare occurrence. Their existence is recognized by the hindrance they offer to normal movement of the parts and the rigid condition of the affected muscles.

The Flexors of the Hand and Fingers

are frequently subject to tonic spasm. The hand is flexed towards the radial side and is rendered concave. In *tonic spasm* of the muscles of the fingers supplied by the ulnar nerve, the thumb is adducted and the little finger is strongly flexed.

Treatment. All these isolated forms of spasm are best treated with the constant current. The rule is to apply

the anode of a stabile current to the spasmodic muscles and the kathode to an indifferent point.

Saltatory Spasm.

This is a peculiar form of reflex spasm affecting the lower extremities, but only when the patient attempts to stand or walk. The moment the soles touch the floor very energetic contractions begin in the muscles of the leg, which forces the patient to jump or to hop. This affection is occasionally seen in nervous and hysterical individuals.

WRITERS' CRAMP AND ALLIED PROFESSIONAL NEUROSES.

Among the disorders of co-ordination in persons following occupations that require the constant use of the hand and fingers, none is as frequently seen as "writers' cramp." There is no defect of the gross muscular power of the hand, but as soon as the patient begins to write, he loses the control of the associated muscular movements needed in using the pen. The right hand being the one usually employed is oftenest affected. No other cause is known than the constant use of the pen.

Symptoms. The trouble generally develops gradually. At the beginning, writing is rendered difficult, and finally it becomes impossible. There are various ways in which the movements in using the pen are disturbed. A frequent variety of the affection consists of a spasmodic condition of the fingers as soon as the pen is seized. The pen is so firmly pressed by the fingers that it cannot follow the impulse of the writer. In another form the arm is quickly tired out and at last becomes powerless to con-

duct the pen, or the limb is affected with a tremor so that the letters are distorted and the writing illegible. Occasionally improvement takes place after the patient has desisted for some time from using the pen, but in general he is finally compelled to relinquish the effort. Persons affected with writers' cramp are often subject to other functional disturbances of the nervous system.

Treatment. Cessation of writing for weeks or months is absolutely necessary whatever plan of treatment be adopted. It is of no avail for the patient to learn to write with the other hand, for that will very soon be similarly affected with the cramp. Many contrivances have been devised for facilitating the act of writing. The one which appears to give much satisfaction is a sort of bracelet to which a penholder is attached, that supports the outstretched fingers. In mild cases the patient may succeed in writing if he rests the whole arm upon a low desk.

The judicious and persevering use of galvanism has given some good results. One of the methods is to apply the anode of a weak current to the muscles of the arm and fingers, and the kathode to the region of the cervical vertebræ. Interruptions are to be avoided. The faradic current appears to do more harm than good.

Brilliant cures have lately been reported from systematic massage treatment. No special rules can be laid down for the varied and complicated manipulations which are necessary to achieve success. To acquire the proper skill it is indespensable to be familiar with the nice and harmonious play of the muscles in the act of writing.

Professional neuroses are also met with among pianoforte players, telegraph operators, type-writers, cigarette-rollers, tailors, shoemakers and engravers.

Milkmaids' Cramp.

In most cases both hands are affected with spasmodic contractions on attempting any kind of movement, but particularly that of prehension. The median and superficial radial nerves are involved. Treatment is the same as that of writers' cramp.

CHAPTER VII.

MINOR NEUROSES

Headache.

The clinical importance of headache is to be rated in accordance with its persistence and association with other symptoms. In all febrile affections and grave diseases of the brain, headache is a very frequent concomitant. Severe and constant pain in the head attends cerebral meningitis, brain tumor and cerebral syphilis. Migraine and neuralgia of the trigeminus and disease of the upper cervical nerves must be distinguished from ordinary headache. Headache is also a common reflex symptom in a variety of morbid conditions. Gastric derangement and sluggish bowels are often accompanied by frontal headache. A distressing form of headache occurs in cerebral anæmia and neurasthenia. Rheumatic headache affects the scalp, usually the occipital region. Toxic influences such as alcoholism, chronic lead and nicotine poisoning, frequently give rise to headache.

Physical overexertion and mental excitement constitute daily causes of headache. On excluding all these manifold sources of headache, there still remains a peculiar form of the malady which is usually called "nervous headache" or "habitual headache." Its paroxysmal occurrence has been quaintly termed a "nerve storm."

There are people who are never without headache. Probably in numerous cases this functional affection is due to a constitutional predisposition. The pathology of habitual headache can only be conjectured. It may be said that the brain substance is in an irritable condition. We know, however, that sensory filaments from the trigeminus are sent to the dura mater. Circulatory disturbances of the brain of the nature of congestion, arising under well-known circumstances, may become manifest as cephalalgia.

Habitual headache greatly varies in intensity and frequency of occurrence in different individuals. Some patients can foretell by vague sensations that an attack is coming on. An attack may continue for half an hour or last a day or two. The headache may only amount to a dull, heavy feeling, diffused over the whole head, or the pain is limited to a particular spot. Patients often give graphic descriptions of the severity of the pain. They say, it feels as if the head were pressed in a vice; or if it were crushed or split. During, and even some time after an attack, many of the patients manifest much irritability of temper; they declare themselves unfit for physical or mental exertions.

Habitual headache is hard to cure. There may be a particular exciting cause which the patient can perhaps avoid. Overworked and debilitated persons should seek rest or be sent to the country or the seaside. Tonics, especially iron, may often be prescribed with advantage. The coexistence of indigestion, which is usually accompanied by sluggish bowels, may require attention. Patients often ascribe their headache to a particular article of diet, or think they suffer from dyspepsia, and then take too little nourishment. In general they are great

consumers of cathartic medicines. This is all wrong. If a mild aperient is indicated it is best to order small doses of Carlsbad salts or the fluid extract of cascara sagrada.

The majority of patients have long ago come to the conclusion that nothing will help their headache, and are resigned to their fate. But they still continue to resort to certain remedies, which they think do them some good, or which really give them some relief. Remedies which stand in much repute are cold applications to the head; mustard plasters behind the ears or on the temples; a hot footbath, strong green tea, etc.

There is no lack of remedies from which to choose. Quinia takes the first rank, and next comes arsenic, in the form of Fowler's solution. Salicylate of soda in one large dose sometimes averts an attack. Guarano in powder form, 10 to 20 grains every two hours, is occasionally of service. The Eff. bromo-caffein is a very popular remedy. The monobromate of camphor in pills, from 4 to 6 grains, is also much prescribed. Nervous persons may receive benefit from frequently repeated doses of aromatic spirit of ammonia or sweet spirits of nitre. Prompt relief is often obtained from phenacetin and antipyrin. Excellent results are now and then observed from a very weak constant current. For a minute or two the electrodes may be applied to the temples, or one over the forehead and the other to the nape of the neck.

HEMICRANIA.

(Migraine. Sick Headache.)

Etiology. By hemicrania is understood a peculiar form of headache, affecting only one side. The pain is generally attended by vaso-motor disturbances. Women are

chiefly liable to this affection. Uusually it dates from the period of puberty, but genuine migraine also occurs in young children. The cause of this malady is obscure, nor is it known with certainty what part of the nervous system is primarily involved. Although the concomitant vaso-motor symptoms manifest disturbance of the cervical sympathetic, they may nevertheless be of a reflex character, due to a central irritation. Heredity is probably always a predisposing cause. Hemicrania is especially common in nervous and hysterical women. Mental excitement is very liable to bring on an attack.

Clinical history. Paroxysms of migraine recur at irregular periods, though in some women they coincide with the menstrual flow. A majority of the patients enjoy their usual health during the intervals, while others still continue to be troubled with various nervous complaints. The onset of an attack is often announced by shuddering, sighing and yawning; a feeling of heaviness and of pressure of the head; flickering before the eyes; noises in the ear, and a feeling of general weakness. The attack begins with a boring or thumping pain, first in the frontal, then in the temporal, region, and finally invades one-half of the head, in the majority of cases the left side. The pain has not the intermittent character of neuralgia, but gradually increases in intensity. There are no painful points in the distribution of the trigeminus, but the scalp becomes very sensitive to the touch.

At the height of the attack occur the disturbances of the special senses that had already appeared in a minor degree during the prodromal period. The ocular symptoms are very prominent, consisting of shimmering before

the eyes, flashes of light and temporary hemiopia (hemicrania ophthalmica).

The vaso-motor symptoms deserve especial notice, as they support the theory of migraine being an affection of the sympathetic. During the acme of the attack, in a number of cases, one-half of the face shows decided pallor, the skin is cool, the temporal arteries are contracted, the pupils are dilated, and the flow of saliva is increased. Towards the close the spastic condition of the arteries relaxes, the affected side of the face reddens, and the skin becomes warm (hemicrania sympatico-tonica). In another form of migraine there is unilateral flushing of the face, the skin appears puffed, its temperature is raised, the pupils are contracted, the temporal arteries dilated, and sometimes there is one-sided sweating of the face (hemicrania sympatico-paralytica). Although the sympathetic nerve is certainly involved in many cases, it cannot be said that the difficulty of explaining the character of the affection is thereby removed. The occurrence of pain has still to be accounted for.

Attacks of migraine vary greatly in duration and severity in different persons. They may last an hour or a whole day. Nausea or vomiting frequently supervenes towards the termination, and is succeeded by nervous depression and a strong inclination to sleep.

In view of the chronic course of migraine it is advisable to be cautious in regard to promises of cure. Patients learn to submit to the periodical visitations of their sick headache. When an attack comes on they usually retire to their rooms, darken the windows, and refuse to be disturbed.

Treatment. Relief is often obtained from cold applications to the head. Morphia does not allay the pain as promptly as in neuralgia and is often ill borne, as are all the other narcotics. Phenacetine and sulfonal are sometimes effective. A large dose of quinia at the commencement of the attack may succeed in arresting it. Powders of guarano in half-drachm doses are at times of benefit. Salicylate of soda is another remedy worthy of trial. This remedy is best taken in *café noir*. Two scruples may be given at once. I have seen some good effects from teaspoonful doses of the effervescent citrate of caffein every thirty minutes. Inhalation of nitrite of amyle has been suggested in the spastic form of migraine.

NEURASTHENIA.

Etiology. The late Dr. Beard of New York described, under the name of neurasthenia, a functional disorder of the nervous system, which he alleged to be extremely common among the adult male population of the United States. He says: "One reason why neurasthenia has been so long neglected is that the symptoms are in some instances so subtle and difficult of analysis and classification. One, who has never seen and carefully examined a large number of cases of this disease, would not believe it possible that it should manifest itself in so many different ways."

Nearly all the morbid phenomena which are said to characterize neurasthenia have usually been grouped among different affections, chiefly hysteria, spinal irritation and hypochondriasis. It will hardly be disputed that many vague and ill-defined symptoms come frequently under notice which cannot be satisfactorily

referred to those disorders of uncertain pathology and inconstancy of clinical features. Experience fully sustains what physiological teachings lead us to expect, that numerous and diversified disorders of the nervous system are directly traceable to the depressing influences of physical overexertion and mental strain. Whatever other causes may be assumed to favor their occurrence, it is but fair to consider nervous exhaustion a fruitful source of manifold functional disturbances. The practical importance of recognizing such a condition under the many disguises it may assume is undeniable. It is a curious instance of the change of meaning that words undergo, that the word "nervous" was originally employed to imply the idea of vigorous, racy, forcible: usage has now assigned to it the very opposite meaning. We still speak of the "nervous style" of a writer, but a feeble, excitable person is characterized as being "nervous." Dr. Beard's happy choice of the word "neurasthenia," which denotes nervous exhaustion or nervous debility, was at once taken up and has gained currency among medical men. The adverse criticism which Dr. Beard encountered in setting up a new disease was certainly undeserved, for neurasthenia has the same claims as an independent nervous affection as hysteria or spinal irritation. But his assertion that neurasthenia is *par excellence* an American disease cannot be sustained, as it is found to be a widespread nervous affection since medical observers have become better acquainted with its characteristics.

The greater number of neurasthenic patients are found in our large cities, the centers of culture and traffic, but also the places that incite to excesses. Men in the prime of life are its usual victims, and this fact is significant.

The expenditure of nerve-force is enormous in the competition for wealth and position. The present state of society, especially in our country, holds out numerous and tempting promises not only to the resolute and those of a vigorous constitution, but also to those of a weaker fibre and stamina. There is, in consequence, a restless, dissatisfied spirit abroad that strives and strains for the attainment of the prizes of life. The unavoidable concomitants of vexation and worry of those not favored by a robust nervous system gradually undermine the very roots of healthy existence. Irritability, feverish excitement and discontent unsettle the poise of the coördinate powers of life; then comes the shock of disappointment, the depressing consciousness of failure and the rebound from blasted expectations. These deleterious influences, varied as they are in different individuals, do the work silently until mental and physical disturbances make their appearance that are often difficult to trace to their original causes. Neurasthenia is said to be "the penalty we pay for our high-pressure civilization, and for the wear and tear of body and mind in the hot race for wealth and distinction." The latter clause comes certainly nearer to the truth.

A predisposition to neurasthenia or an inherited tendency, as in allied affections, must be admitted to exist, for otherwise we could not conceive why among individuals, who are exposed to the same order of influences, some should be affected while others go free. It has, for instance, not been observed that shipwrecked sailors, or soldiers undergoing the hardships of long marches, or professional or business men of a robust constitution who are constantly engaged in the pursuit of their labor-

ious and trying avocations, are picked out as victims of the disorder. If a certain proportion of these classes of persons become neurasthenic a predisposition must be presumed to have operated that tended to the development of this morbid condition. But neurasthenia, as has been shown, may be acquired by the influence of the exciting causes that heavily tax the integrity of the nervous system, diminish the capacity of properly performing its manifold and delicate functions and bring about an "irritable weakness" that lays the foundation of the functional disorder. It is hardly necessary to enumerate all the more special exciting causes that eminently tend to its development.

Clinical History. The distinction of cerebral and spinal neurasthenia is based on the prominence of certain classes of symptoms in individual cases. Cerebral neurasthenia is generally observed among men who do much "head-work," or are engaged in positions of great responsibility, whose extensive and complicated business affairs make great demands on their constant attention. The literary man, for example, begins to find himself unequal to his task; he soon feels exhausted; the work before him becomes irksome and he is finally unable to bring himself to carry on a sustained intellectual effort. He complains of headache, vertigo, insomnia, an indifferent appetite, muscular weakness and a feeling of general debility. A merchant is worn out by unremitting attention to business, anxiety concerning heavy ventures, perhaps financial embarrassment, and the like. His health breaks down; he becomes dyspeptic; he feels the necessity of rest, which he denies himself; he is subject to frequent attacks of fainting, his sight grows weak, and he has

"swimming" of the head. One of these symptoms may be more obtrusive than the rest, which fixes his attention upon a particular organ as the seat of his trouble. A hypochondriac disposition is thus apt to show itself.

A very singular form of psychical disturbance sometimes develops in this class of patients. The disorder relates to a morbid fear, which the patient is unable to suppress. It may assume different forms, the most common being agraphobia (fear of open places); claustraphobia (fear of inclosed places); androphobia (fear of crowds of people) and mysophobia (fear of contamination). I knew of a gentleman, who had been unfortunate in stock speculations, and whose morbid fear consisted in the dread of fatal accidents that might be due to his negligence. He tortured himself with the accusation of having caused the death of an intimate friend because on a cold winter day he did not close the door of the sick-room on his last visit. He picks up bits of fruit parings he finds on the pavement and throws them into the gutter lest some one might slip on them and break a limb. He stops in his walk to fasten a loose brick on the pavement, and hammers in nails which he finds sticking out in houses and fences which he passes. The man's intelligence is perfectly clear.

The pathophobia in neurasthenia is not to be confounded with hypochondriasis. The former implies an unsubstantial dread; the fancied disease of the latter is founded on a real morbid sensation.

Disturbances of the special senses are also quite frequent in neurasthenia. Asthenopia often exists, but hallucination of hearing is the most troublesome and distressing of this group of symptoms. A nursing

woman, who complained to me of a large number of neurasthenic disorders that had only lately begun to affect her, consulted me particularly concerning a distinct voice that terrified her almost continually with the command to kill her child. She was perfectly aware of the unreality of the voice, which was proved by her desire to avail herself of medical assistance.

A peculiar species of headache, which may properly be called the "neurasthenic headache," is also a very harassing and obstinate symptom. It is not precisely a pain, but a sensation as if the head were pressed upon by a heavy weight, or distended to bursting. Patients declare they will "go mad" if not relieved.

Spinal neurasthenia gives rise to numerous symptoms. The most prominent and constant one is pain in the back and the lower extremities. The patient complains of an unaccountable feeling of fatigue even if he is inactive. Micturition and defecation may cause much discomfort. The genital function is often impaired. There is an unpleasant feeling of coldness, numbness or formication of the limbs or trunk. Many neurasthenic patients suffer only from the minor disorders that have been described and get little sympathy. The more severe forms of the affection unfit patients for all physical and mental effort.

Diagnosis. Dr. Beard was undoubtedly correct when he laid stress on the difficulty of recognizing the existence of neurasthenia. Diagnosis may err in many ways. In the presence of a complexity of symptoms, that do not agree with the picture of a well-known individual disease, a particular symptom of prominence may be falsely singled out by the observer to which the rest are consid-

ered subordinate. It may happen, for instance, that dyspeptic derangement is obtrusive, which suggests serious disease of some of the chylopœdic viscera, or the muscular weakness and numbness of the lower extremities may suggest paraparesis. The diagnosis of neurasthenia certainly requires caution, and often must be arrived at by exclusion.

Prognosis. Neurasthenia is not a dangerous affection, but it takes a chronic course and is subject to exacerbations. The greater number of patients make a complete recovery, while others of a neuropathic tendency are never entirely cured.

Treatment. It is of the first importance, in the treatment of neurasthenia, that the patient change the mode of life which led to his brain exhaustion or spinal irritability. He may require absolute rest and quiet, or be benefited by outdoor exercise that does not fatigue. The recuperative influence of mountain air, or a visit to the seaside, should be strongly recommended when the patient is much run down. All kinds of mental excitement should be avoided. He may require sufficient sleep, a substantial diet and a judicious course of tonic remedies. The nervous dyspepsia can be better overcome by assuring the patient of his ability to digest the ordinary dishes to which he has been used than to ply him constantly with stomachic remedies. In his case the indigestion is due to the want of a proper innervation of the muscular structure of the stomach, and probably the same fault affects the secreting apparatus. A minute direction in regard to the articles of diet to be selected only confirms him in his pusillanimity.

Next in importance is the moral treatment, differing, however, from that followed in hysteria. The dread and apprehension of the neurasthenic patient is to be allayed by constant assurance of the curability of his disease, and confident expressions in regard to his progressive improvement at every visit. Severe nervous debility in women must be systematically treated, for which Weir Mitchell's method is excellently adapted.

The refreshing and invigorating influence of electricity is much appreciated by neurasthenic patients. It is a constitutional treatment that should be persistently employed. The methods of application may be varied. General faradization and central galvanization are the most effective. In timid persons it is preferable to use the electric bath or the "electric hand." The restoration of the muscular vigor is also assisted by massage. Surf-baths and swimming are of analogous benefit.

Symptomatic treatment requires a few remarks. Habitual constipation, which always attends nervous dyspepsia, is best overcome by a change of diet. Kneading of the abdominal walls is sometimes quite effective in exciting the peristaltic action of the bowels. Sexual weakness is benefited by douches to the back. Continence should be advised. Wakefulness, which is a standing complaint in neurasthenia, tempts to the abuse of morphia and chloral. Before even prescribing the less powerful hypnotics some other means may be found to secure sufficient sleep. A warm bath before bedtime or a wet cloth wrapped around the head is often effectual. Very weakly persons unused to alcohol frequently sleep well on a hot rum-punch, or a glass of ale. The bromide of potassium in large doses occasionally promotes sleep.

A resort to the following hypnotics offers much choice. They include sulfonal, urethan, paraldehyde, canabis indica and phenacetin. But in the end all these drugs disappoint. In fact, the amelioration of insomnia, and all the other symptoms of neurasthenia, will come with the general improvement of the patient.

SPINAL IRRITATION.

Etiology. Formerly, when this disorder was thought to be of frequent occurrence, extravagant notions were entertained concerning its importance, as the common pathological factor of a legion of diseases. Spinal tenderness is now known to attend many affections of which it constitutes an insignificant symptom. Moreover, the same symptom is so often observed in hysterical females that neurologists of the present day are inclined to discard "spinal irritation" as an independent disease. But, on the other hand, it is hardly permissible to label every obscure nervous disorder in females with the convenient title, hysteria. The physician in actual practice cannot ignore the fact that he has frequently to face a group of symptoms, which seem to stand in no other relation to each other than their co-existence with painful vertebræ. The circumstance that eccentric pains and an exalted sensibility constitute symptoms almost invariably associated with spinal tenderness tells strongly in favor of the assumption that spinal irritation is at least a malady of clinical importance.

Excessive physical exertion trying to the spine is probably, in many instances, the exciting cause. Anstie says: "I believe the starting-point of the disorder will very often be found in some strain or blow to the back."

Clinical History. Patients are often unaware of the existence of tender vertebræ until they are firmly pressed upon. The painful points are usually limited to the cervical or upper dorsal region. In severe cases the whole vertebral column appears to be tender to the touch. Areas of hyperæsthesia about the neck and between the shoulders are also recognized.

Neuralgiform pains of a shifting character are felt in different parts of the chest and abdominal walls. Their locality frequently corresponds to the distribution of the sensory fibres that proceed from the tender portions of the spinal column. A crampy condition of the muscles of the neck often gives rise to a distressing sensation of choking. There is sometimes a dry, harassing cough, attended by disturbed respiration and cardiac anxiety, which greatly alarms the patient. Insomnia is much complained of, and in aggravated cases the feeling of languor and depression causes the patient to seek the bed or lounge in the daytime. As the disorder is almost exclusively confined to the female, it is no wonder that the menstrual function is often irregular. It would be unprofitable to enumerate all the "functional symptoms" that, by a choice of phrase, might also be named hysterical or neurasthenic.

Although spinal irritation is a chronic disorder, exceedingly variable in the severity and number of its symptoms, hard to deal with and prone to relapse, it nevertheless tends to recovery.

Treatment. As spinal irritation often occurs in females who have undergone much hardship in life, both physical and mental, or subjected the nervous system to debilitating influences, such remedial measures are to be rec-

ommended that invigorate the general constitution. Symptomatic treatment is often called for to meet special disturbances. All observers are agreed that the alcoholic stimulant is of decided benefit in giving relief to the manifold ailments of this disorder, though its dangerous fascination is a great drawback to its employment as a remedy. Blistering of the tender vertebræ should be continued until the pain disappears. The hyperæsthesia often yields to stabile galvanization of the affected parts.

CHAPTER VIII.

DISEASES OF THE MEMBRANES OF THE SPINAL CORD.

Preliminary Remarks. Ideopathic inflammation of the membranes of the spinal cord is usually attributed to the influence of cold. The disease is more frequently due to direct injury of the vertebral column, or to extension of the inflammatory process in the bones, to the meninges. Of greater practical importance is the associated inflammation of the cerebral and spinal pia mater, which constitutes the distinctive anatomical change of the infectious disease known as "epidemic cerebro-spinal meningitis." Implication of the spinal meninges is of subordinate importance in inflammatory disease of the substance of the cord (meningo-myelitis). Spinal meningitis may complicate tubercular cerebral meningitis. It sometimes develops in pyæmia, typhoid fever and the acute exanthemata.

PACCHYMENINGITIS SPINALIS.

Suppurative inflammation of the external surface of the dura mater often arises in caries of the vertebral bones (pacchymeningitis externa). The inflammation often involves the inner surface of this membrane and gradually spreads to the pia mater (pacchymeningitis interna). As

there are no peculiar symptoms that distinguish the different forms of spinal meningitis, it will suffice to describe the primary acute inflammation of the spinal pia mater.

SPINAL MENINGITIS.

(Leptomeningitis Spinalis Acuta.)

Anatomical Changes. The pia mater in the early stage of the disease is injected and thickened. As the disease advances, an effusion of coagulable lymph is thrown out, which is most abundant upon the posterior surface of the cord. Later, a sero-purulent or purulent exudation collects in different parts of the surface of the cord, and sometimes is found to cover the whole length of this organ.

Clinical History. In many cases of spinal meningitis there is a combination of spinal and head symptoms, due to the simultaneous implication of the cerebral and spinal pia mater. The symptoms of spinal meningitis may also complicate acuter infectious diseases. The clinical character of a primary spinal meningitis is likewise varied or modified by symptoms dependent on inflammation or the disturbed circulation of the cord. By a careful analysis of the symptoms in complicated cases it will not be found difficult to distinguish those that refer to the spinal inflammation. They chiefly consist of severe pain in the back and the inferior extremities, accompanied by stiffness of the trunk and the affected limbs. The least movement of the body starts and increases the pain and the muscular rigidity. This is the reason why the patient is found lying perfectly still on his back. He

shuns every movement and even tries to repress coughing and sneezing. The voluntary immobility of the patient presents the appearance of loss of muscular power, but paralysis is not a symptom directly due to spinal meningitis. Hyperæsthesia is often well marked and attended by increase of the reflex excitability. The patient dreads to be moved or to change his position in bed in fear of bringing on spasmodic movements. In severe cases he is annoyed by the opening and shutting of the door, and even by a footfall on the floor. The bladder is irritable and the bowels are confined. There is always more or less fever. The development of anæsthesia or paralysis indicates implication of the cord.

Diagnosis. An uncomplicated case of acute spinal meningitis is chiefly recognized by the rather sudden onset of a febrile affection attended by rhachialgia, rigidity of the trunk, and pain and stiffness of the inferior extremities. When these symptoms develop in connection with those of cerebral disease, they are overshadowed by the far more serious import of the latter; and the same is true when the appearance of paraplegia indicates implication of the substance of the spinal cord. It is of importance to recognize the grave character of spinal symptoms arising in pyæmia or typhoid fever when the cervical cord becomes involved. This occurrence is manifested by disturbance of the respiration, disordered action of the heart and pupillary changes.

Prognosis. Recovery may be expected in the primary form of acute spinal meningitis, due to exposure to cold, but the prognosis of pacchymeningitis and of that form of the disease accompanied by cerebral symptoms is exceedingly unfavorable.

Treatment. An active treatment is indicated in the primary form of the disease. The therapeutical measures that tend to subdue the inflammation include wet cups in the painful regions of the vertebral column, followed by warm or cold poultices. A saline cathartic is often of much benefit, not only for moving the bowels, but also as a derivative. This may be kept up with advantage by small doses of calomel. The bladder demands attention. For the relief of the rhachialgia nothing can take the place of the judicious administration of morphia. Iodide of potassium is of benefit in the later stage or when the disease is protracted.

HEMORRHAGE OF THE SPINAL MENINGES.

(Spinal Apoplexy.)

Injury of the vertebræ is the usual cause of extravasation of blood from the venous plexus between the bones and the dura mater. Aneurisms have been known to burst into the spinal canal.

The symptoms of spinal apoplexy develop suddenly and with great violence. The pain in the back is intense; the patient falls over as if shot; the lower extremities become powerless and the nervous prostration is extreme. After a variable time, if the patient recovers from the effects of the shock, the group of symptoms characteristic of spinal meningitis make their appearance.

CHRONIC SPINAL LEPTOMENINGITIS.

Since the recent advances in the more precise pathology of the spinal cord the existence of a primary form of chronic spinal meningitis has become exceedingly doubtful. The possibility of the acute disease becoming

chronic is not disputed, but according to observation it must be of very rare occurrence. Inflammatory adhesions of the pia and dura mater are often found in various affections of the spinal cord, but are of slight clinical importance.

PACCHYMENINGITIS CERVICALIS HYPERTROPHICA.

Etiology. This is a chronic disease of the cervical dura mater. It is met with in persons addicted to excessive use of alcohol, and is also attributed to exposure to cold and damp.

The **Anatomical Change** consists of marked induration of the dura mater, the pia being only slightly affected. The thickening of the membrane irritates and subsequently compresses the nerve roots. Finally the motor tract of the spinal cord is involved.

Clinical History. The disease begins with severe neuralgiform pains in the occiput, neck and arms. There is also a feeling of numbness in the upper extremities, on which occasionally an herpetic eruption makes it appearance. In the second stage an atrophic paralysis develops in the muscles of the forearm, which are supplied by the median and ulnar nerves. In some cases the muscles to which the musculo-spiral is distributed are also implicated. This gives rise to a peculiar deformity of the hand. The hand is strongly flexed, the fingers are on a line with the metacarpal bones and are flexed, and the thumb is bent upon the palm of the hand. If the change in the cord takes a downward direction the inferior extremities become also paralyzed and anæsthetic, but there is no wasting of muscles. The additional symp-

toms include painful swelling of the small joints of the hand, dilatation of the pupils and troublesome hiccough. Ross reports two typical cases of the disease, one of which recovered. Joffroy recommends the thermo-cautery to the back of the neck.

THROMBOSIS OF CEREBRAL SINUSES.

Besides the inflammatory thrombosis of the cavernous and petrosal sinus occurring from extension of otitis to the dura mater another form of thrombosis is met with affecting the longitudinal sinus, which is seen in ill-nourished children and marasmic adults. The clinical history of these cases is not sufficiently pronounced to permit of a certain diagnosis. Either the symptoms characteristic of meningitis are prominent or there is a low typhoid condition.

CHAPTER IX.

DISEASES OF THE SPINAL CORD.

Diseases of the spinal cord may be divided into two large groups. The pathological change in the one is more or less diffused in the transverse direction of the cord, involving both the white and gray substance. This group includes the different forms of transverse myelitis. The anatomical change in the other group is confined to definite tracts of the cord. This remarkable limitation of the lesion gives rise to special symptoms, that distinguish the "systemic diseases" of the spinal cord. They chiefly include locomotor ataxia, poliomyelitis anterior acuta, amyotrophic lateral sclerosis and progressive muscular atrophy. The clinical peculiarities of this class of diseases are essentially due to the situation and not the nature of the lesion. We are thus enabled in cases which do not correspond to typical spinal affections to form a diagnosis concerning the definite part of the cord which is involved. The simultaneous or successive implication of different nerve tracts of the cord give rise to combined forms of spinal disease. This is usually dependent on the extension of the original lesion.

The following figure reproduced from Young is an instructive diagram illustrating a number of functional disturbances of the spinal cord.

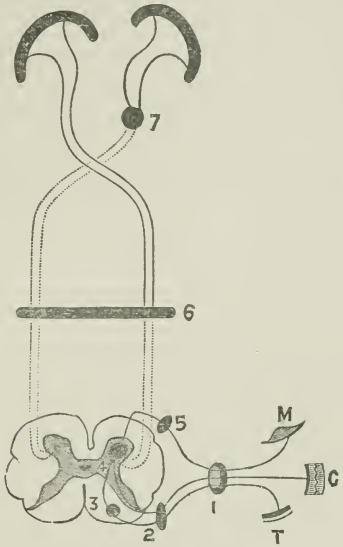

FIG. 26.

C, Skin.
T, Tendon.
M, Muscle.
1, Lesion of the peripheral nerve.
2, Lesion of the posterior root.
3, Lesion of the posterior root zone.
4, Lesion of the anterior gray horn.
5, Lesion of the anterior root zone.
6, Transverse lesion of the spinal cord.
7, Local lesion of the cerebral hemisphere.

The different nerve fibres of C and T terminate in the ganglion cell of the anterior gray cornua from which issues an efferent nerve, which connects the cell with the muscle M. The lesion 7 of the pyramidal tract in the hemisphere causes hemiplegia of the opposite side. The dotted lines indicate descending degeneration of the pyramidal tract.

Lesion of the peripheral nerve at 1 causes atrophic paralysis, loss of sensation and abolition of both kinds of reflexes, but no paralysis. Lesion of the posterior root nerve at 2 causes loss of sensation and abolition of both kinds of reflexes, but no paralysis (pacchymeningitis). Lesion of the posterior root zone at 3 causes loss of tendon reflex only (locomotor ataxia). Lesion of the anterior gray nerve at 4 causes atrophic paralysis, but no sensory disorder (poliomyelitis). Lesion of the anterior root at 5 causes the same symptoms. Transverse lesion of the spinal cord at 6 causes paralysis of the lower extremities with excess of both kinds of reflexes.

ACUTE AND CHRONIC MYELITIS.

Etiology. The usual causes of ideopathic myelitis are attributed to physical hardship, especially outdoor work during rough weather. It is much less certain that sexual excesses or violent emotion cause the disease. Individuals are affected in whom the existence of a predisposing or exciting cause cannot be ascertained. The most frequent causes are injuries to the vertebral column, such as fracture, dislocation, penetrating wounds, falls, and blows on the back. Another important factor in the causation of myelitis is compression of the cord in caries of vertebral bones. Secondary myelitis may result from suppurative spinal meningitis. There is also strong evidence that myelitis may be of syphilitic origin.

Anatomical Changes. On inspecting the spinal cord in recent cases of myelitis no marked changes are seen. An expert may, however, recognize in some places an abnormal hardness or softening of the cord. The outlines of the gray matter on cross sections appear less distinct than normal and the white substance is of a reddish-gray color.

Microscopic examination gives more satisfactory evidence of the effects of the inflammatory process. Staining

brings out a prominent difference between the healthy and diseased parts of the cord. The nerve fibres in the latter are reduced in number, some are atrophied and others have entirely disappeared; the axis cylinders are round or contracted and have lost their processes. A striking change is recognized in the neuroglia, consisting of increase of connective tissue, which replaces the destroyed nerve elements. The "spider cells" represent the many branched nucleii of the connective tissue cells. Compound granular corpuscles are seen in abundance between the meshes of the neuroglia, but are less numerous in old sclerosed parts. Marked changes are also found in the blood-vessels; they are dilated and congested, this condition gives rise to hæmorrhagic spots. The walls of the vessels have undergone "hyaline degeneration." These anatomical alterations usually occupy a transverse section of the cord and extend for a very short distance in the longitudinal direction of the organ. The only difference in regard to the character of the anatomical changes of acute and chronic myelitis is the unimportant circumstance that the diseased part of the cord in the former is of less consistence. Clinically we speak of acute myelitis, when there is a rapid development of the symptoms.

Clinical History. A description of the motor and sensory disorders, including abnormal reflex action and implication of the bladder, the rectum and sexual functions, embraces the essential clinical history of all forms of transverse myelitis. The special symptoms observed in individual cases depend on the particular region of the cord which is the seat of the disease.

Motor Disturbances. The initial motor symptoms indicate the development of paralysis. The patient complains of weakness and a sensation of fatigue in the lower extremities, and he has an unsteady gait. Gradually the weakness of the limbs increases until marked paraplegia is established. These symptoms take a different course when the upper region of the cord is affected. Signs of motor irritation are also observed in the early stage, consisting of the spontaneous occurrence of twitching and cramp in the paralyzed muscles. Paraplegia is the constant and conspicuous symptom of transverse myelitis, as the motor tract of each lateral column of the cord is involved.

Sensory Disorders are not well marked in the early period of mild forms of myelitis. Some slight numbness and furriness of the lower extremities is often present. At a later stage the cutaneous sensibility in these parts is impaired and complete anæsthesia is finally established. This occurrence indicates implication of the gray posterior cornua and the posterior columns of the cord. Total anæsthesia of the paralyzed parts speedily ensues in traumatic myelitis. In lumbar myelitis the anæsthesia is on a line with the umbilicus; in myelitis of the lower dorsal region the anæsthesia is on a level with the lower part of the sternum; in myelitis of the upper dorsal region the anæsthesia reaches the axilla, and in cervical myelitis the sensibility of the arms is impaired.

The Reflexes. Both the cutaneous and tendon reflexes are morbidly affected, either in consequence of interruption of the centripetal paths of the nerves or interference with the inhibitory fibres. In severe inflammation of the dorsal or cervical cord there is marked increase

of the reflexes. When the disease involves the lumbar enlargement the patellar and cremaster reflexes are abolished.

Disorder of the Bladder and the Rectum. Impairment of the functions of the bladder and the rectum is a very common symptom of myelitis. At the outset there is considerable vesical irritability. The evacuation of the bladder is attended by a straining effort, and there is often retention of the urine. At an advanced stage incontinence ensues, which frequently leads to cystitis. This is always a serious complication, as it threatens the development of pyelo-nephritis. Obstinate constipation of the bowels exists from the beginning, and as the paralysis increases the discharges pass away involuntarily.

The sexual function is weakened early in severe cases, and finally may be wholly lost. In the case of a young married man whom I attended the myelitis, which resulted from the kick of a vicious mule, had so far improved that the patient was again able to walk with the assistance of a cane, while his sexual powers still continued to be entirely lost.

Trophic Disturbances. The paralyzed limbs in disease of the dorsal and cervical cord do not waste. The muscles are, however, flabby from disuse. The electric reaction is normal. When myelitis affects the lumbar cord the muscles of the lower extremities atrophy and manifest the reaction of degeneration.

Vaso-Motor Disturbances. The paralyzed limbs present a mottled appearance or are cyanotic. The surface is cool and œdema appears.

Bed sores constitute serious complications in myelitis. They usually develop in the sacral region and inner side of the knees. The decubitus is chiefly due to pressure and the effect of irritating discharges from the bladder and rectum. Malignant decubitus is supposed to be due to trophic changes.

Psychical Symptoms are absent during the whole course of myelitis. In rare cases the brain is involved from upward extension of the disease, which gives rise to bulbar symptoms.

Changes of the Pupils are observed in cervical myelitis. Sometimes optic neuritis is discovered by the ophthalmoscope.

Diagnosis. Transverse myelitis in which the diameter of the cord is implicated can hardly give rise to diagnostic difficulties. The essential symptoms are paraplegia, anæsthesia, implication of the sphincters of the bladder and rectum, impairment of the sexual function, absence of atrophy in lesion of the dorsal and cervical regions, but wasting of the paralyzed muscles and abnormal electric reaction in advanced cases of lumbar myelitis. It remains yet to consider the special symptoms presented when the disease attacks different segments of the cord.

Cervical Myelitis. In myelitis of this region of the cord some or all of the muscles of both arms and legs are paralyzed. Atrophy of single muscles is occasionally observed, and in a late stage also anæsthesia. The tendon reflexes in the lower limbs are exaggerated and there is disturbance of the sphincters of the bladder and rectum. Pupillary changes are sometimes noticed.

Dorsal Myelitis. The paraplegia is confined to the

inferior extremities, and eventually anæsthesia develops. There is no degenerative atrophy of the paralyzed muscles, but increase of the tendon reflexes. The bladder and rectum are involved.

Lumbar Myelitis. There is motor and sensory paralysis of the legs, the upper extremities are free. The cutaneous and tendon reflexes are diminished. Sometimes there is atrophy of the paralyzed muscles and reaction of degeneration. The functions of the bladder, the rectum and the sexual organ are impaired.

Course and Prognosis. Chronic myelitis, which is not the continuation of the acute form, begins slowly and in an insidious manner. The vague paræsthetic sensations and rheumatoid pains in the limbs, the slight muscular weakness, the occasional vesical trouble and slight impairment of the sexual function may at the beginning cause little anxiety to the patient. Gradually the paresis increases, although the cutaneous sensibility be still intact until the paralysis is completely established. Chronic myelitis may remain stationary for years and even for a lifetime. Some improvement may occasionally be observed, but recoveries are extremely rare. A fatal termination is threatened on the occurrence of cystitis or the appearance of malignant bed sore.

Treatment. The faintest suspicion of a history of syphilis indicates the prompt employment of the specific remedies. This causal treatment is of course to be abandoned if not followed by improvement after a reasonable time. Ergotine is recommended by Brown-Sequard. This remedy may be of service in the early stage. Russel Reynolds speaks favorably of the tinct. ferrichloride. Among the other internal remedies generally employed in myelitis are

included the iodide of potassium, strychnia and nitrate of silver.

Very little must be expected from other curative measures in confirmed myelitis. Electricity may be of considerable benefit, though it can as little restore degenerated structure, as any other remedy. Galvanization promises most. The method recommended is to place two large electrodes on the vertebral column near the supposed seat of the disease in the cord, and to pass through it a stabile or slow labile current of medium strength. The position of the electrodes may be varied in different sittings. Stimulation of the paralyzed muscles with the constant and interrupted current is also to be practiced. The electric brush is adapted for the anæsthesia. The bladder and rectum may be separately galvanized with suitable electrodes. A warm bath, which should never be over 98° F., is an excellent means of relieving many unpleasant sensations attending the paralysis. It often relieves tremor and twitching of the paralyzed muscles. The warm mud baths of the Arkansas hot springs stand in much reputation for the treatment of paraplegia.

Attention to the functions of the bladder and the rectum is required throughout the whole course of the disease. To prevent retention of urine nothing can replace the use of the catheter. Severe cystitis is treated with chlorate of potassium freely diluted, benzoic acid and liquor potassæ.

Bed sores are often prevented by cleanliness and the proper adjustment of the bedclothes so as to avoid the formation of folds and wrinkles. A water or air cushion may become necessary. The abrasions of the skin are

sometimes satisfactorily treated with a combination of castor oil and balsam of copaiba spread on a piece of thick linen cloth. Suppurating sores are best treated with iodoform liniment.

Constipation should be overcome by an appropriate diet and enemata. The restlessness and insomnia of old paralytics may sometimes be best relieved by a hot rum punch at night.

ACUTE ASCENDING PARALYSIS.

Landry's Disease.

This is a peculiar form of paralysis which was first described by Landry and has since been often seen by other observers. The etiology of the disease is obscure. It attacks apparently healthy persons and men more frequently than women.

Clinical History. The development of the paralysis is always announced by premonitory symptoms. For the first few days the patient complains of headache, loss of appetite, a dragging sensation in the limbs, pain in the back and some fever. Quite suddenly one leg becomes paralyzed, then the other, next the trunk and lastly the hands and arms. Voluntary movement of the lower extremities is completely abolished, and the paralyzed limbs are flaccid. In some cases the reflexes and the electric excitability remain intact, but usually the tendon reactions are diminished or entirely abolished and the electric excitability is lost.

The sensory disorders are insignificant. Anæsthesia is very seldom observed. There is occasionally some ting-

ling or numbness of the fingers. None of the cranial nerves are affected. The legs are sometimes œdematous and often covered with perspiration. The bladder and rectum are not implicated.

A favorable turn of the symptoms after the patient has lingered from two to four weeks may lead to complete recovery. A fatal termination is imminent if the disease in its upward progress involves the medulla oblongata. Respiration becomes then difficult, the movements of the diaphragm are diminished, and before death ensues the paresis frequently affects the lips and soft palate. But patients have been known to rally even after the appearance of these formidable symptoms and to make a tedious recovery.

In the absence of anatomical changes affecting the nervous system, it has been conjectured that the disease may be of an infectious origin. The character of the premonitory symptoms and the attending fever lend support to this view.

The **Diagnosis** rests on the development of a paralysis commencing in the feet and rapidly extending upwards. Primary multiple neuritis presents similar clinical peculiarities, but this affection is distinguished by marked sensory irritation, consisting of violent pain in the affected parts. These symptoms are absent in acute ascending paralysis.

Treatment. The disease suggests the treatment of acute myelitis. In addition to the usual remedies recommended in that disease it may be of service in the early stage to apply wet cups along the vertebral column.

SPINAL PARALYSIS FROM GROWTHS AND CAVITIES IN THE CORD.

Paraplegia is in rare instances caused by tumor of the spinal cord and more seldom still by an aneurism making its way into the spinal canal. These forms of "pressure paralysis" are accompanied by violent shooting pains and stiffness of the limbs and the usual symptoms characteristic of transverse myelitis. Unilateral paralysis develops when the tumor is confined to one side of the cord.

Cavities found in the spinal cord arise from dilatation of the central canal (hydromyelia), or develop near it (syringo-myelia).

It is impossible to diagnose the existence of a tumor or cavity of the cord during the life of the patient. These lesions give rise to varieties of myelitis according to their situation. Small cavities develop no symptoms.

Unilateral Lesion of the Cord.

Traumatism is the usual cause of this form of spinal paralysis. The peculiarity of the paralysis consists in this, that motor paralysis is confined to the side of the lesion, and anæsthesia to the opposite part of the body. Physiology furnishes an explanation of this phenomenon.

CONCUSSION OF THE SPINAL CORD.

Railway Spine.

Although this is usually considered a surgical disease, yet symptoms of an essential nervous character may develop in the course of weeks or months after the immediate effects of the injury have passed off. Since the introduction of railroad travel, accidents affecting the spinal

cord in consequence of concussion have become frequent and have given rise to suits for damages. Erichsen in his little book on this subject proposed the name "railway spine" to characterize spinal concussion in which there is absence of gross injury to the vertebræ, such as fracture, dislocation or hemorrhage. A knowledge of the finer changes taking place in the spinal cord from concussion, independent of a palpable lesion, is not claimed. The inference that this organ is in a morbid condition is based on the appearance of a group of symptoms that are evidently spinal. Such an inference is strengthened by the analagous fact that no coarse lesion of the nervous system has been found in many cases of death occurring a few hours after concussion of the brain.

Clinical History. The symptoms that develop or become troublesome by their persistence after the accident are chiefly subjective, and for this reason admit of different interpretations. They mainly consist of a general muscular weakness and painful sensations in diffent parts of the body. There is no actual paralysis, but many patients are quickly tired out from standing and walking. The gait is stiff, slow and dragging, and often requires the aid of a stick or crutch. The pain is mostly felt in the back and limbs, and there is a sensation of constriction about the trunk. Numbness of the tips of the fingers is frequently complained of and on closer examination well-marked anæsthsia will be found in different areas of the skin. In some cases there is contraction of the field of vision. The tendon reflexes are diminished or may be entirely abolished. If cerebral symptoms are present they generally consist of headache, dizziness, attacks of

faintness, ringing in the ears, specks before the eyes and often nervous irritability and mental depression.

Recovery or much improvement may be expected from treatment in favorable cases. The trouble is, however, usually protracted and tends to develop a serious spinal affection. The hope of having made a fortunate escape from the consequences of the concussion is disappointed. Paresis and anæsthesia of the legs and disturbance of the functions of the bladder and rectum and sexual organ render it apparent that the previous mild symptoms have culminated in the establishment of a grave spinal disease. The occurrence of difficulty of speech, insomnia, defective memory and general nervous prostration indicate cerebral complication. Though casual improvement may still take place it soon becomes clear that the patient is continually losing ground. Emaciation and marked weakness are manifest and the fatal termination is hastened by the occurrence of any incidental complication. It may be fairly assumed that the concussion has finally given rise to grave lesion of the spinal cord and brain.

The **Diagnosis** of spinal concussion should not be lightly made, for symptoms showing irritation of the spinal cord and functional motor disturbances may be due to a different cause, or may be simulated. The circumstance that the initial symptoms are all subjective and partake of the hysterical or neurasthenic character render it incumbent to make a thorough examination. The judgment in regard to the early symptoms should take into consideration the influence of fright, the exaggeration of impressible individuals and a possible interested motive.

Due weight must, however, be given to the condition of the reflexes and other physical signs of disease.

The Treatment of the condition that persists after the shock has passed off consists of rest in bed, cold sponging, followed by friction of the back and galvanization of the vertebral column and of the limbs. General faradization is also of benefit. The judicious administration of iodide of potassium, ergotine and strychnia may be found of service.

CHAPTER X.

SYSTEMIC DISEASES OF THE SPINAL CORD.

Loco-motor Ataxia.

(*Tabes Dorsalis.*)

Etiology. Loco-motor ataxia is a striking example of the class of systemic diseases of the spinal cord. It is a chronic disease, anatomically distinguished by degeneration of the posterior columns of the spinal cord and clinically marked by peculiar motor symptoms. The predisposing and exciting causes are usually referred to a hereditary tendency, physical and mental overexertion and sexual excesses; but the uncertainty in regard to these alleged etiological factors must be admitted. The disease appears with greater frequency in men than in women, and usually in the middle period of life. The fact has been ascertained as the result of recent investigations that a very large percentage of ataxic patients have had a history of syphilis. Fournier rates the proportion at 75 per cent; Erb as high as 90 per cent. Other observers give lower figures, but the probability of a connection between ataxia and syphilis is very strong. Secondary or tertiary symptoms are only occasionally observed in ataxic patients. The objection raised against the supposed relation existing between

ataxia and syphilis is the circumstance that the anatomical changes in ataxia differ histologically from those characteristic of syphilitic degeneration. It is nevertheless highly probable that loco-motor ataxia is somehow connected with a syphilitic influence. In female tabetic patients a history of syphilitic infection can be made out in nearly every case.

Anatomical Changes. Inspection of the spinal cord in patients who have died in the advanced stage of loco-motor ataxia discovers considerable alteration of this organ. A streak of gray discoloration running along the whole length of the shrunken cord is seen through the pia mater. This membrane at the under surface is thickened. On a cross-section it is noticed that the smallness of the cord is due to atrophy of the posterior columns. These parts appear thin and flattened and are distinguished from other portions of the cord by their darker color. The posterior cornua and posterior nerve roots present sometimes a similar appearance.

The microscope shows unequal distribution of the degeneration. In the lumbar cord the morbid change is well marked in the middle and posterior portion of the posterior columns. Nearly the whole of the posterior columns of the dorsal cord is degenerated. Goll's column in the cervical cord is chiefly implicated, and also fibres of the posterior root zones that enter Burdach's column in the lumbar cord. Areas of degeneration are also seen in the gray matter of the posterior cornua, and many of the medullated fibres of Clarke's column have disappeared. The degeneration in advanced cases can be traced to peripheral nerves of the posterior nerve roots, especially in the trunk of the sciatic. It is note-

worthy that every case of loco-motor ataxia presents the same anatomical change in the symmetrical nerve tracts of the spinal cord that subserve the same physiological functions.

The pathological process in the diseased structure is a primary degenerative atrophy of the nerve elements and an increase of the connective tissue. The grayish color of the posterior columns is due to the loss of the medullary sheaths. A few fat granules and numerous corpora amylaceæ are also found in old cases.

Clinical History. It is convenient in considering the clinical history of loco-motor ataxia to divide it into three stages, although there is great variation in the intensity of the symtoms, their order of occurrence and their combination.

1. *The stage of sensory irritation.* Usually the disease develops in an insidious manner and this condition may continue for a considerable length of time before the more significant symptoms make their appearance. Patients begin to complain of wandering pains in the lower extremities, which they are apt to ascribe to rheumatism. The arms and back are often similarly affected. But the most characteristic symptom at the early stage is the occurrence of darting, lancinating or lightning-like pains in the legs. These pains are of great intensity and recur at first at irregular periods, but as the disease advances they are almost continuous. Sometimes "stabbing" pains are felt in the joints. A headache like migraine is also a common tabetic symptom. The "girdle sensation" is often well pronounced. The patient has the feeling as if a rope were tightly fastened around his trunk or abdomen, or there is a feeling as if the

calves of the legs or the insteps of the feet were tightly bandaged.

The other sensory disturbances are less constant. Certain areas of the skin are found hyperæsthetic. The patient is especially very sensative to changes of temperature. Some degree of numbness in the tips of the fingers is often present. On examining a patient it may be found that at first he does not feel the prick of a pin, but a few minutes afterwards he feels pain at the point of contact. The tactile sensation is frequently abnormal. On being touched in one place he may declare that he felt two simultaneous impressions. It is only in the later stage of the disease that evident anæsthesia and anomalies of the muscular sense can be satisfactorily recognized. Disorders of the bladder and rectum, which are usually well marked in advanced cases, are often conspicuous at an early period, and the same may be said of the sexual function.

Ocular symptoms sometimes appear at an early stage. Immobility of the pupils is often noticed, especially the "Argyle Robertson symptom," or the pupils are very much contracted. Paralysis of ocular muscles is not as frequently observed at the beginning and may only be temporary. A diplopia may disappear and not return. Dimness of sight, contraction of the field of vision terminating in total blindness, also belong to the occasional initial symptoms. The latter complication of the disease is due to optic atrophy. In a small percentage of cases auditory symptoms develop consisting of tinnitus, deafness and vertigo as in Menier's disease.

2. *The ataxic stage.* The appearance of the ataxic symptoms may be considered the second stage of the disease.

There is no paralysis, for the patient is able to offer great resistance to an attempt to bend the limb, but there is inco-ordination of movements. The mechanism which adjusts and controls the harmonious action of muscles in carrying out intended movements is impaired. The act of standing is insecure and uncertain, the gait is oscillating and tottering. In the act of rising from his seat the patient has much difficulty to gain a secure footing, which he tries to overcome by bracing himself with his hands. These irregularities of motion are of much importance and may be observed before the disease has made much progress. The patient himself may have noticed that his body sways when, for instance, he bathes himself in the morning. When mounting steps he is apt to stumble. He has a straddling gait, he places his feet wide apart to gain greater support; in lifting them he raises them too high and they come down on the ground with a stamp. On making a quick turn, as in the military "face about," he loses his equilibrium. In standing he seeks for support by leaning against something, and in walking he needs a stick. There are many ways of testing the existence of inco-ordination. When the patient lies on his back and is told to touch the knee of one leg with the heel of the other foot he makes several attempts before he succeeds. When he is asked to throw one leg over the other he makes too wide a sweep with the raised leg.

The impaired cutaneous sensibility and disorder of the muscular sense constitute the causes of "Romberg's symptom." If the patient closes his eyes whilst standing his body begins to reel; he is unable to sustain his center of gravity, and if not supported he would fall over. It is interesting to observe the influence of sight

in supplementing the loss of control over the movements of locomotion. The patient fixes his eyes on every step he takes. The upper extremities are much less affected. There is sometimes a want of precision in manipulations, especially in those that require nicety of execution. Patients experience much difficulty in buttoning a sleeve or threading a needle. Anæsthesia is frequently well marked; at least some of the different qualities of common sensation are either blunted or entirely abolished. There is impairment of tactile and muscular sensibility. This explains the fact that patients cannot tell the position of their limbs when in bed or in the dark. A more constant sensory symptom is an abnormal feeling of the nature of numbness. Patients compare it to a feeling of furriness, especially experienced in the soles of the feet, as if they were standing on a soft cushion or on a bag filled with air. In the hands the sensation produces the impression of being covered with gloves. There is also a morbid sensitiveness to changes of temperature.

The cutaneous reflexes, as a general rule, are normal, but the *abolition of the patellar tendon reaction* is such a constant symptom of loco-motor ataxia that it constitutes one of the most important diagnostic signs of this disease. Exceptional cases may be met with in which many of the tabetic symptoms are present, while the patellar reflex is not deficient. This circumstance does not diminish the significance of the absence of the knee jerk. On the other hand there are healthy individuals in whom the patellar tendon reflex can be but faintly elicited, which of itself would not be considered an abnormal condition.

Trophic disorders are not conspicuous in ataxia. An

eruption of pemphigus or herpes sometimes appears on the inferior extremities along the course of painful nerves. Occasionally perforating ulcer of the foot develops. Of greater interest is the occurrence of painful swelling of one of the large joints. The "*arthropathic tabetique*" either affects the hip or the knee joint.

Intercurrent attacks of gastralgia, nausea and vomiting —the *crisis gastrique*—are observed in many cases of ataxia. *Laryngeal crisis* consists of the occurrence of severe dyspnœa and spasmodic cough. Violent lumbar pain, resembling an attack of nephritic colic, is less frequently observed. Periodical acceleration of the pulse is sometimes noticed during the course of the disease.

Incoördination. The peculiar motor disturbance which is such a prominent symptom of loco-motor ataxia, brings up the consideration of the nature of coördination and the mechanism that subserves this physiological function. It is now generally understood that co-ordination is the machine-like execution of movements by which different muscles are brought into harmonious action. The voluntary impulse incites the special movements, but consciousness is not concerned with the action of the individual muscles. The exercise of the will is all that is necessary to set the muscles into action to carry out the purposed movement. The mode in which co-ordination is established we may learn from the efforts of a child when it begins to walk, or when we atttempt new and complicated movements. We call into play our tactile sense and our sense of sight, and, above all, our past experiences of graduating our muscular energy in accomplishing a desired act. In other words, we make use of the "muscular sense" in co-ordinating our move-

ments. Gradually an organic connection is established between certain nervous elements, so that consciousness is no longer engaged in the movements after they have once been incited by the will. Although the existence of centers and conducting paths of co-ordination cannot be anatomically demonstrated, yet there is strong reason to infer that such exist in the spinal cord and are implicated by the lesion that gives rise to loco-motor ataxia. We have seen that an ataxic patient resorts again to the aid of the sense of sight to relieve, in a measure, the loss of co-ordination.

3. *The Paralytic Stage.* Numerous ataxic patients succumb to some intercurrent disease, so that they are spared the wretched condition of helplessness which marks the paralytic stage. As in all severe affections of the spinal cord that tend to a fatal termination, paraplegia sets in attended by bedsores and nephritic complications.

Diagnosis. Loco-motor ataxia, when fully established, presents such clearly cut clinical features that diagnosis meets with no difficulty. At an early stage, when the neuralgiform pains in the inferior extremities constitute the most conspicuous symptom, diagnosis may be doubtful. It is well to remember that persistent or paroxysmal pain in both legs, especially if attended with eye symptoms and vesical trouble, should always raise suspicion of serious spinal disease; and if the patellar reflex is much diminished, or absent, the diagnosis of loco-motor ataxia is certain.

Prognosis. Experience confirms the bad prognosis, which may be *a priori* entertained of a disease marked by a progressive destroying lesion. Recoveries if they

ever occur, are extremely rare. Still the forecast need not be absolutely gloomy, for loco-motor ataxia sometimes assumes a mild type, and, under favorable conditions, life may be prolonged for many years. The course of the disease is very chronic, though occasionally serious symptoms develop rapidly. Much can be done for the relief of the patient in palliating the severity of the pain in the limbs and other symptoms as they arise, which sustains his hope and courage.

Treatment. The early recognition of the disease affords the opportunity of adopting an appropriate treatment, with the expectation at least of effecting some improvement, if not arresting its advance. The patient should consider himself an invalid, and abandon any avocation that calls for much physical and mental exertion.

An energetic anti-syphilitic treatment in the early stages is indicated if there should be the least suspicion of the venereal taint. Enough would be gained in checking the advance of the disease, although the mercury and the iodide of potassium be "found wanting" for the removal of the mischief that has already occurred.

Different methods of electric treatment are recommended. An ascending constant current may be passed through the cord in the manner that has been mentioned for the treatment of chronic myelitis. Another method consists in brushing the skin of the back with a strong faradic current for five or ten minutes. Peripheral galvanization is often of benefit in allaying the pain in the limbs and the irritation of the bladder. The methods of electric treatment may be varied, but whichever is selected should be kept up for months.

The Suspension Treatment. An estimate of the merits of

the suspension treatment in ataxia, recently introduced into practice by Charcot, can hardly yet be decided upon, although in many of the reported cases the improvement was incontestible. Further experiments will determine whether the benefit derived from this treatment is a permanent one or not. Suspension is made by Sayre's well-known apparatus. It is advisable to commence with half-minute suspensions, and then according to the susceptibility of the patient they may gradually be prolonged to the maximum of four minutes every other day.

Nerve-stretching of the sciatic for the relief of the pains often produces a good effect, though usually it is transient.

Internal remedies are indicated on the same general principles that call for their employment in the treatment of myelitis. The nitrate of silver is highly recommended. One-fourth of a grain in pill is the usual dose to begin with. It is given three times a day before meals.

Symptomatic treatment is often indicated, more particularly for the mitigation of the pains in the limbs, for which hardly any other remedy but morphia will suffice. Vesical trouble also requires attention.

HEREDITARY ATAXIA.

Friedrich's Disease.

This very rare form of ataxia occurs far more frequently in young females than in males. There is no initial stage of pain. It begins with ataxia of the inferior extremities and soon extends to the arms. The abolition of the tendon reflexes is observed in most cases. The cutaneous sensibility remains intact. There

is no vesical disturbance nor affection of sight. A peculiar defect of speech develops during the course of the disease, which appears to be due to inco-ordinate action of the lips and tongue. The disease runs a very chronic course and finally atrophic paralysis of the extremities is established. The anatomical change consists of disease of the posterior and lateral columns of the spinal cord. Treatment is unsuccessful.

POLIOMYELITIS ANTERIOR ACUTA.

(Infantile Spinal Paralysis.)

Etiology. This disease was described by older writers under the name of "the essential paralysis of children." It occurs in early childhood between the ages of six months and four years. Healthy children are as likely to be attacked as those of a sickly constitution. Some observers conjecture that the disease is of an infectious nature. It is not identical with the forms of paralysis seen after attacks of diphtheria, scarlatina and measles.

Anatomical Changes. The character of the anatomical change in recent cases appears to be an acute inflammation of the anterior cornua of the spinal cord. In old cases the cornua are found in the condition of atrophic degeneration, implicating to some extent the white substance in their vicinity. The degenerated cornua are transformed into a dense tissue from which many of the ganglion cells have disappeared. A secondary degeneration involves the anterior nerve roots, that correspond either to the cervical or lumbar cord, according as the upper or lower extremities had been affected with atrophic paralysis.

Clinical History. The disease in the majority of cases begins with a sudden onset of acute symptoms. A high grade of fever develops, accompanied with headache, delirium and stupor, which continues with brief intermissions for a few days or much longer. Sometimes the initial symptoms are of a mild character and brief duration. After their subsidence or, in some cases while apparently the child is in good health, the little patient is seen to have lost the use of one or both of the lower extremities or the paralysis is more extensive. The arms or some muscles of the trunk may at the beginning be involved but very soon the affection becomes limited to one leg or much less frequently to one arm. The paralysis in the leg is often confined to the muscles supplied by the peroneal nerve. The general health of the child does not seem to be affected and the paralysis usually improves up to a certain point and then becomes stationary and frequently permanent. There is marked flaccidity of the paralyzed muscles from the beginning. Gradually they atrophy and often to an extreme degree. The atrophy may be concealed by an abundant deposit of fat.

Sensory disorders are absent during the whole course of the disease. The functions of the bladder and the rectum are rarely affected.

Reflex action of the paralyzed muscles is much diminished or entirely abolished.

The electric excitability shows marked changes in the course of the few weeks next succeeding the onset of the disease. The faradic reaction of nerves and muscles is rapidly lost and the reaction of degeneration is clearly manifest. For the next two or three months an increase

of the galvanic contractility of the muscles is sometimes observed, which then diminishes and is finally lost.

The surface of the paralyzed limbs is cyanotic or mottled and the skin is flabby and cold. Shortening of the affected limb frequently results from arrested growth of the bones. The relaxed condition of the ligaments of the joints and the lack of muscular support allows of unusual passive motion of the paralyzed limb. The deformities which at a late period develop are partly produced by the predominant action of the antagonistic muscles and partly by the weight and position of the limb. In this manner the different forms of "club foot" develop. In bad cases the stunted, withered limb is reduced to a useless appendage of the body.

Diagnosis. Retarded development in small children may be confounded with some of the symptoms of spina. paralysis. These children are slow in learning to walk and exhibit much awkwardness in their movements durthe period of their growth; but there is no history of acute symptoms, no real paralysis, nor muscular atrophy.

The distinction from other forms of paralysis in children can easily be made if we remember that the essential features of the spinal type are an acute onset, a flaccid paralysis joined with atrophy, loss of the reflex action, and the reaction of degeneration, with retained sensibility.

The temporary paralysis of children described by Kennedy can be usually traced to exposure and is a mild affection that disappears in the course of a few weeks.

Prognosis. After the acute stage has passed off the prognosis is entirely favorable as regards danger to the life of the patient, but the complete restoration of the

motor function is very doubtful. If the paralysis does not continue to improve after the first few months, little further improvement must be expected. By a judicious and persevering course of treatment during the first years it may still be possible, as experience has shown, to induce a noticeable change for the better in the paralyzed parts.

Treatment. When called upon to treat the acute stage of the disease those therapeutical measures will be indicated that are usually employed to subdue febrile excitement and to quiet the nervous system. The recognition of the true state of the case at that period is difficult.

Galvanization holds out the best prospect in the treatment of the paralysis. The most approved method is to place a large electrode on the vertebral column in the region of the cervical cord if an arm is paralyzed, and to the upper dorsal region if a leg is affected. The other electrode is applied to the paralyzed nerves and muscles or dabbed over them. At first a constant current of medium strength is used, which is gradually increased in intensity. During the electrization the current may occasionally be reversed. Sometimes the kathode and then the anode should be slowly passed over the affected muscle. A sitting should last from two to five minutes and be repeated every other day or more frequently. Local faradization of the muscles is often of decided benefit. Electric treatment to be of any avail must be carried out with persistence.

Some benefit is derived from passive exercise of the paralyzed limb, and this is best accomplished by massage. It is possible that this procedure may prevent or at least improve deformities of the limb. The good effects of

various liniments and embrocations, if they have any, are probably due to the vigorous friction that accompanies their use.

Good results are occasionally obtained from the use of brine baths during the Summer months. A brine bath may be improvised by dissolving a few pounds of sea salt in a tub of tepid water.

An orthopedic apparatus will be often found necessary to facilitate walking.

Internal remedies are of little use. It is recommended to give hypodermic injections of strychnia from $\frac{1}{60}$ to $\frac{1}{20}$ grain until its physiological effects are produced.

ACUTE AND CHRONIC POLIOMYELITIS IN ADULTS.

A form of spinal atrophic paralysis identical in anatomical and clinical characters with the disease occurring in children, as described in the foregoing section, has been repeatedly observed in adults. The acute symptoms are far less marked. At first the patient complains of muscular weakness in the lower extremities, and a few days later he is unable to walk. After a short time the arms are similarly affected and become also paralyzed. Extensive atrophy of the paralyzed muscles ensues, followed by loss of the electric excitability. In severe cases the degenerative reaction is manifest and the cutaneous reflexes are lost. In exceptional cases the muscles of the neck, the tongue, the lips and the pharynx are attacked. There are no sensory disturbances, and the functions of the bladder and rectum remain normal. After the complete development of the paralysis there is a halt in the disease for months.

Improvement gradually takes place, and the paralysis may finally disappear; oftener, however, recovery is imperfect and the patient is more or less disabled for life.

Further investigations are required to determine whether the symptoms which have been described are really dependent on a poliomyelitis. They certainly bear a close resemblance to those of multiple neuritis.

SPASTIC SPINAL PARALYSIS.

(Tabes Dorsal Spasmodique. Primary Lateral Sclerosis.)

Although paresis of the lower extremities, increased muscular tension and exaggeration of the reflexes constitute a group of symptoms differing from other well-known forms of spinal disease, it cannot be said that the anatomical basis of this combination of symptoms has as yet been satisfactorily determined; but they strongly point to implication of the lateral columns of the cord. Autopsies of cases that had presented the clinical characters of spasmodic paralysis do not always show evidences of disease of these columns. In amyotrophic lateral sclerosis there is a class of symptoms that include spasmodic paralysis of the inferior extremities, but they appear in association with muscular atrophy, and bulbar phenomena, dependent on extensive destruction of the pyramidal tract. Spasmodic forms of paralysis are met with in chronic hydrocephalus, transverse myelitis of the upper dorsal and cervical cord, tumor of the same regions of the cord, multiple sclerosis and hydromyelia.

Etiology. The exciting cause of ordinary spastic spinal paralysis is unknown. It occurs more frequently in men

than in women, and usually develops about the middle period of life.

Clinical History. The essential symptoms of spinal spastic paralysis consist of a more or less paretic condition of the lower extremities and exaggeration of the tendon reflexes, especially the patellar and the ankle clonus. In many cases there only exists a slight muscular weakness of the limbs, while in others the paralysis is pronounced and extensive. But the chief characteristic of the disease relates to disturbance of motion that depends on stiffness and contracture of the muscles. When the reflex actions are excessive any passive movement of the inferior extremities, even their own weight, as when they hang free over the edge of the bed, brings on contractions. The rigidity of the flexors opposes the bending of the knees. Plantar flexion is sometimes so strong that the soles of the feet cannot be raised from the floor. At times the contractions are attended by such violent tremor that the limbs and the trunk are shaken.

The exaggeration of the reflexes is especially conspicuous when the patient attempts to walk. The muscular tension is then so great that flexion of the limbs becomes very difficult, which causes the characteristic "spastic gait." In consequence of the stiffness of the legs the patient has much trouble to raise his feet; they seem to cling to the ground, and in bringing them forward the toes make a scraping noise. As the stiffened muscles oppose the elevation of the leg, the patient helps himself by rotating the pelvis, first on one side, then on the other. This produces a waddling gait. Sometimes the calf muscles contract so strongly that the patient stands on tiptoe and can only advance with a sort of

hopping movement. He appears to be walking on stilts. The tip of the foot catches on little inequalities of the ground; he constantly stumbles and is always in danger of falling. At last the gait is reduced to a mere dragging of the legs, and the body requires support to keep it erect. The patient, on becoming bedridden, finds it even impossible to sit up on account of stiffness of the legs. Sometimes late in the course of the disease the trunk and upper extremities become also involved. The spastic condition of the hand and fingers is then quite apparent.

The cutaneous reflexes and electric excitability rarely show any alteration. Sensory disorders are absent. The functions of the bladder, the rectum and sexual organs remain normal.

The **Prognosis** in all cases of confirmed spastic paralysis is unfavorable. The disease is always chronic, but unaccompanied by pain.

The **Treatment** is the same as that of chronic myelitis.

AMYOTROPHIC LATERAL SCLEROSIS.

For the first accurate description of this disease we are indebted to Charcot and Ioffroy. A typical case presents a group of well-defined symptoms. The distinctive anatomical changes include degeneration of the pyramidal tract of the lateral columns of the spinal cord, and degenerative atrophy of the ganglion cells of the anterior cornua and certain nerve nuclei of the medulla.

Etiology. The cause of the disease has been attributed to violent physical exertions, but nothing of a positive character is known concerning its etiology. The male sex is more frequently affected than the female. The

disease usually makes its appearance between the ages of thirty-five and forty-five years.

Anatomical Changes. A cross-section of the spinal cord, in advanced cases, shows a simple degeneration of the whole pyramidal tract of the lateral columns of the cord, including both its crossed and uncrossed portions. The sclerosis also involves the ganglion cells of the anterior horns, the pons, the crura, internal capsule, and often some of the nerve nuclei in the floor of the fourth ventricle. Cases are also reported in which the degeneration had implicated ganglion cells of the central convolutions. The nerve nuclei in the medulla which are most prominently affected are the hypoglossus and the spinal accessory. The pathological change in some cases appears to involve the whole motor tract from the center to the periphery. The nerve fibres and cells of the diseased structures are atrophied and many of the nerve elements have disappeared. There is an increase of the connective tissue and slight alteration of the blood-vessels, but these are secondary changes. Destruction of the nerve tissues in the cord and the medulla are due to an identical pathological process. The same system of nerves is involved in every case.

Clinical History. The encroachment of the disease is first noticed in one arm. The patient feels an unusual degree of fatigue in the limb. Soon after the other arm is similarly affected and this paretic condition of the limbs gradually increases. Wasting of the muscles of the hand and fingers is the next symptom, commencing in the balls of the thumb and the little finger. This is succeeded by atrophy of the interossii and extensors of the

forearm. The flexors also become affected, but in a slighter degree. Among the muscles of the upper arm the triceps and the deltoid show more wasting than the biceps and the muscles of the shoulder. In the course of a few months the atrophy also invades the inferior extremities, but to a less extent. The spastic symptoms are very prominent and as well marked as those described in the foregoing section. Although the disturbance of motion is influenced by muscular weakness, the chief difficulty of locomotion is due to spasmodic stiffness of the legs, brought on by excessive tendon reaction. The patellar tendon reflex is easily elicited and is very energetic. Ankle clonus is almost continually kept up by exaggerated reflex excitability of the calf muscles. On testing the reflex reactions in the upper extremities an equal increase of reflex excitability will be noticed. A slight tap on the end of the radius causes vigorous reflex contractions of the biceps and triceps. Spasmodic contractions of the hands and arms sometimes develop spontaneously in the late stage of the disease.

The sensibility continues unchanged and the cutaneous reflexes show no marked alteration. Micturition and defecation remain normal.

A new set of symptoms develop at a later period. Speech and deglutition become difficult. This constitutes the third and last stage of the disease. The tongue and lips are now attacked with atrophy. The articulation of words is indistinct and swallowing is much impeded. It is noticed that the tongue trembles and twitches and shows irregularities upon its surface. The puckering of the mouth for the acts of sucking, blowing and whistling is

much impaired. Difficulty of deglutition interferes with the introduction of a sufficient quantity of food and the embarrassment of the respiration finally leads to a fatal termination.

All the symptoms of amyotrophic lateral sclerosis depend on the disturbance of the physiological functions of the parts of the nervous system which are involved in the degenerative change. The paretic and spasmodic phenomena are due to the destructive lesion of the great motor tract in the lateral columns of the spinal cord, the muscular atrophy corresponds to the lesion of the anterior gray horns, and the bulbar symptoms accord with the implication of the nerve nuclei in the medulla. In explanation of the exaggerated reflex contractions, it may be fairly assumed that they depend either on irritation of reflex loops in the cord that are intact or on failure of inhibition.

Diagnosis. Amyotrophic lateral scleroses presents an assemblage of symptoms which makes its distinction from allied diseases an easy matter. The co-existence of muscular atrophy, increase of the tendon reflexes, the bulbar symptoms, and the absence of sensory and vesical disturbance constitute reliable diagnostic points.

The **Prognosis** must be considered as exceedingly unfavorable. The tendency of the disease to a fatal termination has never yet been influenced by any method of treatment.

PROGRESSIVE MUSCULAR ATROPHY.

Wasting Palsy.

Etiology. Individuals in the prime of life, who are actively engaged in occupations that call for hard physi-

cal exertions, are thought to be especially liable to wasting palsy, but often the exciting cause is unknown. Cases in which a hereditary tendency can be traced belong to another class of muscular atrophy. The disease is sometimes observed to follow syphilis and acute infectious diseases, though probably the muscular atrophy in cases of this kind is not the genuine spinal disease.

Anatomical Changes. We owe to Duchenne and Aran the first excellent description of progressive muscular atrophy, but to Cruveilhier belongs the merit of having recognized the spinal origin of this disease. He located the morbid alteration in the anterior gray cornua of the spinal cord. Opinions were still divided in regard to the correctness of Cruveilhier's statement, for other eminent observers considered wasting palsy a disease of the muscles, until Lockhart, Clarke and Charcot succeeded in demonstrating the spinal origin of the disease. This view is now generally accepted. The gradual atrophy of the muscles which follows a regular type corresponds to degenerative changes of peripheral motor nerves, motor nerve roots and ganglion cells of the anterior gray cornua. There is a high degree of probability that progressive muscular atrophy, amyotrophic lateral sclerosis and bulbar paralysis are of an identical pathological nature. The anatomical difference between these separate diseases rests solely on the circumstance that in each the lesion is localized in a definite portion of the cerebro-spinal axis. A peculiar form of muscular atrophy of a myopathic character will be considered later.

The lesion in the spinal cord is most evident in the anterior gray cornua of the cervical region. Numer-

ous ganglion cells have disappeared and others are much atrophied. Fine connective tissue, studded with spider cells, has replaced the neuroglia. The lateral columns are perfectly normal. Anterior nerve roots and nerve fibres corresponding to the degenerated portions of the anterior horns are also affected. Microscopical examination of the shrunken, pale muscles shows diminution and a waxy or fatty condition of the muscular fibres. The stria are, however, still retained. The interstitial connective tissue of the atrophied muscles is always increased and a deposit of fat is interposed between the remaining muscular fibres. In this condition of the muscles the reaction of degeneration is manifest.

Clinical History. The first sign of the insidious development of wasting palsy is atrophy of the small muscles of the hand, usually the hypothenar eminences. No other symptom at the beginning is experienced by the patient except difficulty in performing certain movements. The atrophy alters the position of the ball of the thumb and approximates it to the second metacarpal bones. Gradually a further deformity of the hand occurs from the wasting of the interossii and the lumbricalis muscles, such as is seen in ulnar paralysis. The next point of attack is the extensors of the forearm, or the atrophy jumps to the muscles of the shoulder. The arm gradually loses its natural contour as the different muscles become affected with atrophy, and finally appears thin and wasted. In the upper arm it is always the deltoid which first atrophies; then comes the biceps, the triceps holding out the longest. If the atrophy is much advanced in the upper extremities the arms hang dangling by the

sides as if suspended by a string, and nothing appears to remain of the shoulders but the projecting acromion and coronoid processes. In their turn the muscles of the back, the chest and abdomen become also more or less affected, those of the lower extremities being but rarely attacked. A curious configuration of the body is produced when there is an irregular wasting of the muscles, only a part of a large muscle being atrophied, while its next neighbor is intact. The bellies of the sound muscles strangely contrast with the grooves left by the wasted muscles. Bones and tendons become prominent in situations where the volume of the muscles is diminished.

Corresponding with the extent of the atrophy there is a functional weakness of the muscles which does not amount to true paralysis, though the patient is finally reduced to a pitiable state of helplessness. For a long time he is still capable of carrying out movements by calling into action supplementary muscles that yet retain their structural integrity. A striking instance of this phenomenon I observed in the case of a so-called "living skeleton." The trapezius was much atrophied so that the head often dropped forward, but he contrived to put it straight again by a violent jerk and retain it in position by means of the deep muscles of the neck. On attempting to rise from his seat he had the trick of giving his body a sudden twist that brought him to his feet. In this case the lower extremities were much atrophied, and to some extent also the pectoralis major, the serratus and other muscles of the back.

In addition to the atrophy and impaired functions of the muscles they are also affected by fibrillary twitching and tremor. These symptoms disappear when the

atrophy becomes complete. The muscular tremor can be easily excited by giving a slight blow to the bellies or tendons of the muscles.

The electric exploration of the diseased muscles varies in results. In general it is found that the electro-muscular contractility diminishes in proportion to the extent of the atrophy. So long as there are some of the muscular fibres left contractions, though feeble, can be obtained. A completely atrophied muscle, or one which is mostly replaced by infiltrated fat, ceases to give responses to either current. Increased galvanic excitability is sometimes observed, and, on the other hand, a decided diminution of electric reaction is occasionally noticed in muscles before atrophy is perceptible.

The tendon reflexes of the superior extremities are much diminished, in striking contrast to their increased reaction in amyotrophic lateral sclerosis. This difference is due to the circumstance that in the latter disease degeneration of the pyramidal tract occurs prior to the atrophy, which is not the case in progressive muscular atrophy. When the lower extremities are involved there is absence of the patellar reflex.

Trophic changes are infrequent and of little significance when they occur. If the atrophy is attended with much fatty infiltration it may be difficult to recognize the wasting, but the atrophied muscles have a soft and pasty feeling and the electric excitability is diminished. The skin is sometimes cyanotic, thick and fissured, and the nails are brittle.

In very few cases a relatively sudden development of the atrophy is preceded by vague rheumatic-like pains, but sensory disorders, as a general rule, are absent

throughout the whole course of the disease. The cutaneous sensibility is preserved and the sphincters of the bladder and rectum are unaffected.

The symptoms of bulbar paralysis make their appearance in protracted cases in consequence of the extension of the disease from the anterior cornua to the nerve nuclei of the medulla oblongata, that innervate the muscles of the tongue, the lips, the pharynx and the respiratory muscles. We have here the identical complication which more frequently and at an earlier date occurs in amyotrophic lateral sclerosis.

Diagnosis. Errors of diagnosis can be easily avoided if the marked peculiarities of genuine progressive muscular atrophy be kept in view. They include the typical course of the disease; the appearance of the wasting, first in the small muscles of the hand; the gradual extension of the atrophy to other muscles; the absence of sensory disorders and the immunity of the sphincters of the bladder and the rectum. Progressive muscular atrophy is excluded in diseases where the wasting is only a subordinate symptom. It is distinguished from amyotrophic lateral sclerosis by the absence of spastic symptoms and the normal condition of the reflexes. The differential diagnosis from the myopathic form of muscular atrophy can easily be inferred from the description of the latter disease in the following section.

Prognosis. Patients may survive for many years if the atrophy advances slowly and makes frequent pauses. Recovery even under the most favorable conditions is exceptional. The fatal termination is hastened when the respiratory and masticatory muscles become affected.

Treatment. Electricity appears to be the only means of exciting some favorable influence on wasting palsy. Duchenne recommends to begin the treatment at once with strong faradic currents to the affected muscles. Galvanization of the spinal cord in the usual method should be combined with it. A systematic course of massage may assist in arresting the advance of the atrophy.

PSEUDO-HYPERTROPHY OF MUSCLES.

(Lypomatous-hypertrophy of Muscles.)

The myopathic class of muscular atrophy is distinguished from the spinal form of the disease by the difference that the anatomical changes in the former develop in the muscles, the nervous system being not involved. Myopathic atrophy occurs in the greater number of cases in the young members of the same family. I once saw three brothers who presented the typical form of the disease. Boys are more disposed to the affection than girls. Nothing is noticed until the patient has begun to walk. The parents cannot understand why the child becomes unsteady on its feet and constantly stumbles in walking, although its limbs are straight and finely developed. The little patient makes a poor effort at mounting steps, falls over when slightly pushed and when down has much trouble to get up. The arms and hands appear perfectly normal, but the attitude in standing is odd and the walk is a mere waddling. In fact the nature of the child's disease can be recognized at a glance by observing the position of the body and the character of the gait. The shoulders and the upper part of the vertebral column incline backwards, the dorsal curvature is deeply arched,

the abdomen protrudes, the feet are kept wide apart and the patient cannot approximate them without the risk of falling. At a late period he cannot bring the heels to the ground whilst in the erect position, but balances his body on the toes so that he easily loses his equilibrium when slightly pushed. The characteristic gait resembles the movements of a duck, the body oscillates from one side to the other. Dr. Ross remarks: "When the feet are kept widely apart the center of gravity must be carried at each step over the side of the active leg in order that the line of gravity may pass through the center of the arch of the foot planted on the ground. It is therefore necessary that at each step the body should be inclined well over the side of the active leg, and the patient aids himself in maintaining the center of gravity vertically above the ball of the foot on the ground by moving his arms about like a rope dancer."

The series of movements which the patient adopts in raising himself from the floor when there is nothing near him to lay hold of is characteristic. He gets on all fours, plants his feet on the floor and props himself with his extended arms, then gradually straightens his legs so that his trunk raises and assumes an inclined position from the buttocks downwards. Having gained this position he next grasps one knee with the one hand, and while the other hand is firmly fixed on the floor, he stiffens the arm to support himself, and then quickly freeing the hand he grasps with it the other knee, and in this manner the trunk is brought into a vertico-horizontal position from the shoulder downwards. The last grand effort to gain the erect position he accomplishes by

thrusting the body forwards and climbing the thighs with his hands.

On observing for the first time the precarious attitude and waddling gait of the patient, attention is attracted by the remarkable development of some of the muscles. The gastrocnimii especially have gained in volume. The gluteal muscles are also massive, and in older patients the thigh and the deltoid are sometimes increased in thickness. "This pseudo-hypertrophy" is caused by an increase of interstitial fat. The real cause of the feebleness is the atrophy of numerous muscles which strangely contrast with the excessive volume of others. In many cases the thighs are thin and wasted, while the gastrocnimii are of an enormous size. The muscles of the upper extremities, with the exception of the deltoids, are more frequently atrophied than enlarged. Those of the shoulder and upper part of the body are occasionally attacked. Usually the distribution of the hypertrophy is such that the arms and upper part of the trunk appear emaciated in comparison with the development of the buttocks and the calves.

Erb's juvenile form of hereditary muscular atrophy is also a disease of early youth. It usually attacks the female members of the same family. The atrophy begins in the shoulders and arms and gradually extends to the trunk and the inferior extremities. In all cases there is a remarkable regularity in the order in which the different muscles are attacked. The motor disturbance corresponds to the extent and severity of the atrophy. There is no apparent increase in the volume of the affected muscles. The disease is very chronic, but sometimes a

sudden fatal termination takes place from asphyxia in consequence of atrophy of the diaphragm.

Duchenne reports cases of atrophy in which the disease began in the facial muscles and then extended to other muscles of the body.

In all the forms of myopathic atrophy no change occurs in the nervous system. There is no fibrillary twitching of the muscle and the reaction of degeneration is not manifest.

Treatment has hitherto proved unavailing.

BULBAR PARALYSIS.

(Glosso-labio-laryngeal Paralysis.)

Etiology. The special disease of the medulla oblongata which Duchenne originally described under the name of glosso-labio-laryngeal paralysis, is caused by a degenerative atrophy of the nuclei of nerves that arise on the floor of the fourth ventricle. To the group of symptoms resulting from this lesion that chiefly affects speech, deglutition and respiration, the term bulbar paralysis is now generally applied. No particular exciting cause of the disease is known, but a history of syphilis exists in many cases. Men between the ages of 40 and 70 years are chiefly affected.

Clinical History. Bulbar paralysis always develops very insidiously. Slight premonitory symptoms, such as pain in the back and front of the neck, precede the impediment of speech that first attracts the attention of the patient. He experiences difficulty to articulate certain consonants, usually r, s, l, k, g and t. Among the vowels he pronounces i but poorly. This defect is mainly due to

disturbed innervation of the tongue, which interferes with the mobility of this organ. As the disease advances an analogous difficulty occurs in chewing and swallowing food. On examination the tongue is found atrophied, it is thin and flabby and grooves form upon its surface. At a later period the tongue can no longer be protruded. Morsels of food remain in the mouth, as they cannot be carried back by the tongue into the pharynx.

A similar debility is observed to affect the lips. Reading aloud becomes quickly tiresome. The acts of whistling, blowing and sucking are but imperfectly performed, and finally puckering of the mouth is impossible. When the paralysis of the orbicularis oris increases the patient is unable to pronounce the vowels o and u, and he finds it difficult to articulate the consonants p, f and b.

The next trouble is due to the extension of the paralysis to the pharynx and larynx. Swallowing is interrupted and the voice becomes monotonous. Liquid food regurgitates through the nose in consequence of the paralytic condition of the soft palate.

Inability to modulate the voice may be the only sign of implication of the laryngeal muscles for a long time, but more serious symptoms develop in the course of the disease. The voice becomes extremly feeble and hoarse, and the incomplete closure of the larynx permits the entrance of liquids and even solid food into the air passages.

A very characteristic change in the expression of the face is observed if in addition to the paralytic condition of the lips the lower facial muscles are also implicated. The mouth stands wide open; the lower lip hangs down; the naso-labial fold is deepened, while the muscles of the

upper part of the face and of the eyeballs are normal. There is a constant trickling of saliva over the chin so that the patient is seen constantly holding a pocket handkerchief to his mouth. Exceptionally the muscles of mastication are involved, which in addition to the paralysis of the tongue and lips, renders chewing extremely difficult. Morsels of food would drop out if the patient did not prevent it by pressing the palm of the hand against the mouth.

At an advanced period it is not unusual to witness attacks of dyspnœa and fainting fits. These symptoms indicate implication of the pneumogastric nerve.

When the disease has reached its height another symptom is occasionally added to the catalogue of troubles, dependent on the implication of the spinal accessory, and causing atrophy of the muscles of the neck. The patient in consequence experiences much difficulty to keep the head in the erect position.

Reflex action of the paralyzed muscles is diminished, or entirely abolished. Tickling of the root of the tongue does not cause the patient to gag.

It is a noteworthy circumstance that all the symptoms enumerated are exclusively motor. The cutaneous sensibility and the senses of taste and smell continue intact.

Course and Termination. During the early stage of the disease there is nothing in the outer appearance of the patient indicating the beginning of a fatal affection. The paralysis invades in the order that has been stated, first the tongue, then the lips, then the soft palate and lastly the larynx. The general health of the patient does not seem to suffer until serious difficulty of deglutition develops. At a late period the patient presents a woeful picture of

wretchedness. Speech is gone, the desperate attempts to swallow food are futile, the lips are thinned to transparency, the mouth constantly gapes, the saliva dribbles away, and amidst all this the intelligence is clear and only the motions of the eyes convey the expression of misery. Death may be postponed for three or five years.

Complications. Bulbar paralysis stands in close relation to the allied affections of progressive muscular atrophy and amyotrophic lateral sclerosis. The degenerative atrophy of the nerve nuclei in the medulla and of the corresponding muscles is analagous to the lesion and its consequences that characterize the latter diseases. The best evidence of the degenerative process in the medulla is found in the nucleus of the hypoglossus nerve, more or less also in the vagus and the accessory and only sometimes in the nucleus of the facial and of the glossopharyngeus. A similar anatomical change is observed in the nerves that start from these nuclei.

The similarity between the nature of the pathological changes in bulbar paralysis and progressive muscular atrophy is complete. In both of these diseases the degenerative atrophy involves the motor and trophic nerve tracts and the corresponding muscles. The lesion in bulbar paralysis affects the nerve nuclei in the medulla, and in progressive muscular atrophy the lesion involves the ganglion cells of the anterior gray cornua. The difference implies only a difference in the localization of an identical lesion. It is now easy to understand why during the course of bulbar paralysis we often meet with atrophy of the extremities and conversely that toward the late period of progressive muscular atrophy

symptoms of bulbar paralysis not infrequently make their appearance.

The complication of amyotrophic lateral sclerosis with bulbar paralysis has also been met with. The occurrence of spastic symptoms in cases of this kind shows the extension of the primary disease in the medulla to the lateral columns of the spinal cord.

Diagnosis. Symptoms of bulbar paralysis have been observed in cases which turned out to be multiple sclerosis, obliteration of basal arteries or tumor of the medulla. In all such cases, however, there is a clinical history which markedly differs from the symptomatology and course of a typical case of primary bulbar paralysis. It would be a serious mistake to confound with this disease the aphonia, the choking sensation and excessive salivation suddenly occurring in a hysterical woman.

Prognosis. Trousseau makes the remark in reference to the prognosis of bulbar paralysis: "I do not believe that a single case is on record in which the progress of this disease has been arrested for a single minute."

Treatment. An effort should at least be made to check the advance of the disease. Duchenne and Kussmaul observed temporary improvement of the speech and deglutition from faradisation of the palate and tongue. Galvanization may possibly be of some service: the electrodes should be applied to the mastoid processes. Difficulty of swallowing may sometimes be relieved by applying the kathode to the side of the larynx and the anode to the nape of the neck. Excessive salivation is restrained by atropia.

CHAPTER XI.

DISEASES OF THE MEMBRANES OF THE BRAIN.

Diseases of the cerebral meninges are nearly always of a secondary nature, and more or less involve the substance of the brain. Each of the membranes may be separately affected, but frequently the inflammatory process commencing in the dura mater extends to the pia.

INTERNAL HEMORRHAGIC PACCHYMENINGITIS.

(Hæmatoma of the Dura Mater.)

Etiology. Hæmatoma of the dura mater is far more frequently met with on the dissecting-table than recognized during the life of the patient. It is sometimes found in connection with acute febrile diseases, as typhoid fever, smallpox pneumonia, and is often one of the post-mortem appearances in the general paralysis of the insane, senile dementia and chronic alcoholism. Usually the disease occurs among old people and more frequently in men than in women.

Anatomical Changes. Hæmatoma is generally considered to originate in inflammation of the internal surface of the dura mater, which leads to the formation of a new membrane and extravasation of blood within its meshes. In old cases the connective tissue of which the mem-

brane consists is much thickened and filled with blood. The new growth exerts much pressure on the underlying part of the brain. Its rupture gives rise to "meningeal apoplexy." The usual seat of the hæmatoma is the parietal region.

Clinical History. Symptoms referable to hemorrhagic pachymeningitis are not characteristic though they are sometimes very serious. The difficulty of diagnosis is due to the variable location, size and recurrence of the hemorrhage. The disease gives rise to headache, vertigo, a slow pulse, vomiting and stupor: sometimes hemiparesis or twitching of muscles of one side of the body is a conspicuous symptom, or only one extremity is paralyzed and affected with convulsive movements. The paralysis, if the hemorrhage is copious, may become bilateral. Aphasia occurs in some cases. The disease may begin abruptly like an apoplectic attack. Drowsiness is often a prominent symptom in old people, and when conjoined with contracted or dilated pupils the possible existence of a hæmatoma is indicated.

The course of the disease is very variable. In many cases improvement takes place; the paralysis and the other symptoms may even entirely disappear, but only to return at irregular intervals on the recurrence of the effusion. Death from coma suddenly ensues in aggravated cases.

Diagnosis is impossible when hæmatoma occurs as a complication in affections distinguished by other prominent symptoms. The variable clinical features the disease assumes must at any rate embarrass its recognition. The following diagnostic points are to be considered :

Chronic cerebral disease, alcoholism, sudden onset of severe cerebral symptoms, their improvement and recurrence, unilateral spasms, hemiplegia or monoplegia.

Treatment. It is impracticable to recommend any special line of treatment in a disease of this kind. The apoplectiform attacks would call for a cooling treatment and derivatives, and paralysis for the electric treatment, etc.

DISEASES OF THE PIA MATER.

The varieties of cerebral meningitis are distinguished partly by their etiology and partly by the particular seat of the inflammation. The pia mater is chiefly affected. We distinguish :

1. Tubercular meningitis.
2. Meningitis of the convexity.
3. Epidemic cerebro-spinal meningitis. This being an acute infectious disease it cannot be properly classed among nervous diseases.

TUBERCULAR MENINGITIS.

(Acute Hydrocephalus.)

Etiology. Tubercular meningitis is generally believed to be a secondary affection, but the focus of infection in remote parts of the body cannot always be found at the autopsy. In the larger number of cases the meningeal disease in adults takes place in the course of pulmonary tuberculosis. The influence of age is marked. Tubercular meningitis most frequently affects children.

Anatomical Changes. Miliary tubercles develop in greatest abundance in the pia mater at the base of the brain, hence the disease is often characterized as "basilar meningitis." The greater number of tubercles are found along the course of the large blood-vessels and furrows between the convolutions. They can easily be detected on stripping the hyperæmic membrane from the brain. The inflammatory exudation chiefly consists of a sero-fibrinous effusion, which is sometimes cloudy from the presence of a scant amount of pus. Hemorrhagic spots are frequently found in the inflamed pia. Often the brain substance is also involved and shows deposit of tubercles and capillary hæmorrhage. Flattening of the convolutions is seen when the exudation is large. A copious effusion, more or less of a sero-purulent appearance, is usually found in the ventricles, hence the disease was formerly termed "acute hydrocephalus." The pia mater of the spinal cord is often conjointly affected. This membrane being inflamed and sometimes studded with tubercles explains the combination of spinal and cerebral symptoms.

Clinical History. It is convenient for description to divide the clinical history of the disease into three stages. The first stage includes the prodroma due to cerebral irritation. The second stage marks the complete development of the disease, and the third stage is the stage of collapse corresponding to grave impairment of important nerve centers. But the sudden or slow development of the inflammatory exudation, the variable extent and severity of the cerebral implication, and the degree of compression exerted by the effusion constitute factors that greatly

modify the symptomatology and course of the disease in individual cases.

The onset of tubercular meningitis is occasionally tumultuous. In topers the disease not seldom begins as a delirium tremens. Generally, however, the disease is preceded for a longer or shorter period by a precursory stage. For a week or two patients complain of headache, aversion to food, constipation, sleeplessness and a feeling of general illness. Vomiting is often a conspicuous initial symptom. This condition grows worse, the headache increases, delirium comes on, and soon the marked signs of a formidable brain trouble make their appearance.

In children, who are more frequently the victims of tubercular meningitis than adults, the invasion presents some peculiarities. It often appears as if a previous attack of measles, hooping cough, or other affections to which children are prone, had hastened into activity the dormant process of tuberculosis. But apparently healthy children are often unexpectedly taken with the disease. There are families who lose their little ones, after they have arrived at a certain age, from "brain fever." An infant refuses the breast, sleep is disturbed, the tongue is coated, the bowels are constipated, and towards evening fever appears. The little patient has short spells of crying, is exceedingly restless or is drowsy and often vomits without a palpable cause. Usually these symptoms are attributed by the mother or nurse to difficult dentition, indigestion, worms, or disordered bowels. An older child has headache, refuses food, is restless, abandons its playthings, and with remarkable frequency complains of pain in the chest and abdomen. In very young children it is

not unusual for the disease to set in suddenly with elevation of the temperature and general convulsions.

The second stage in adults may begin with a chill, but the most prominent and constant symptom, when the disease is completely established, is violent pain in the head. It persists, with short intermissions, as long as the patient is at all conscious. He breaks out in loud complaint of its severity, and even when overcome by the advancing drowsiness the contortions of the face express its continuance. (I once saw a little girl who in this stage constantly beat her forehead with the fist.) Very young children manifest the headache by the repetition of an abrupt shrill cry and by boring the head into the pillow or tossing it from side to side.

Delirium is an early symptom in adult patients. They sing, shout or whistle, throw off the bed covering and make attempts to escape from the room. Sometimes the delirium is low and less noisy, the muttering being unintelligible and the patient picks the bedclothes.

The most conspicuous motor symptoms are stiffness of the neck and retraction of the head due to implication of the upper region of the spinal cord. Various symptoms showing irritation of cranial nerves at the base of the brain make their appearance. There is twitching of the facial muscles, strabismus, rolling of the eyeballs and later ptosis and partial facial paralysis. The pupils show much irregularity, they may be unequal, contracted or dilated. Usually they are sluggish and finally do not respond to light. Spasmodic movements of the limbs are occasionally observed, but more frequently a rigid condition of the inferior extremities exists. In children a boat-shaped appearance of the abdominal walls is

observed toward the close of the second stage. If the patient does not fall early into a state of unconsciousness there is generally well marked hyperæsthesia and intolerance of light and sound.

The temperature varies. It rarely rises above 103°, but at times there is an elevation reaching 105° to 106°, or the temperature may suddenly become subnormal.

Remarkable changes of the pulse are noticed. In the early stage it often gets as low as 60 or 50 beats per minute, and later it becomes exceedingly rapid and feeble. The "tache cerebral" is well marked in small children.

The respiration is often sighing and nasal. At the early stage before stupor comes on the breathing is accelerated, but toward the end it is hardly perceptible.

Constipation of the bowels persists throughout the disease. It has been noticed that the cough, dyspnœa and the profuse perspiration in phthisical patients cease on the development of meningitis, but the marasmus rapidly increases. The second stage of the disease averages about eight days.

The third or final stage shows the signs of speedy dissolution. Swallowing of food is now impossible. The breathing is irregular and occasionally assumes the character of the "Cheyne Stokes respiration." The pulse can hardly be counted, the extremities are cold, and the coma is profound. In children the fontanels are sunken and often death is ushered in by general convulsions and paralysis. Cases of recovery from tubercular meningitis are reported, but it has been asked—how about the diagnosis ?

Diagnosis. There are few diseases of infancy that offer greater difficulties to diagnosis than the prodromic period

of tubercular meningitis. A decisive judgment in regard to the nature of the symptoms will often be held in suspense until positive evidences of cerebral trouble appear. It is advisable when diagnosis is doubtful not to disregard the symptoms of incipient pulmonary tuberculosis and to examine the chest. Scrofula and diseased joints are of similar import. Suspicion should always be awakened if a child begins to vomit without an assignable cause and shows great irregularity of the pulse. If the invasive stage of tubercular meningitis in adults is very protracted and accompanied by high fever it may suggest the development of typhoid fever. Here again, a cautious opinion must be formed until the disease declares itself. The ophthalmoscope may come to the assistance of diagnosis if tubercles are found in the choroid. The differential diagnosis between the tubercular and simple variety of meningitis chiefly rests on the question of etiology.

It is sometimes surprising to find at the autopsy very insignificant changes, that seem insufficient to account for the gravity of the symptoms.

Treatment. An infant should be provided with a healthy wet nurse when tuberculosis is hereditary on the mother's side. Superabundance of clothing and a hot sleeping-room are rather injurious to children disposed to brain trouble. Tepid bathing and friction of the skin should be recommended, and, in fact, all the well-known prophylactic measures.

Energetic treatment in tubercular meningitis is of doubtful benefit, but cold applications to the head are very serviceable when steadily kept up. Purgatives are indicated at the early stage. Calomel is best adapted for children. Iodide of potassium deserves a trial. These

little patients can easily tolerate from two to three grain doses. Warm baths give temporary relief to the cerebral excitement, but small doses of Dover's powder or a few drops of a morphiæ solution should not be withheld. Children under two years of age should never be blistered. In the stage of collapse stimulants are indicated, but they are of little avail.

MENINGITIS OF THE CONVEXITY.

Etiology. Simple meningitis of the convexity is very rarely an idiopathic disease. It is probable that sporadic cases may be examples of that variety of the disease which is known as the epidemic cerebro-spinal meningitis. In fact simple meningitis is so frequently found to be a secondary affection that many observers doubt its occurrence as a primary inflammation. In place of making the divisions of "traumatic meningitis," "metastatic meningitis," etc., an enumeration of the remote causes that give rise to purulent inflammation of the cerebral membranes will answer every practical purpose.

Disease of the middle ear is a very frequent cause of secondary purulent meningitis. In caries of the petrous portion of the temporal bone, which is usually due to otitis media, the extension of the disease into the cranial cavity is easily accounted for. Irruption into the interior of the skull may take place, or the inflammation proceeds from the mastoid cells, or it creeps along the sheath of the nerves. Suppurative phlebetis of a venous sinus may ensue when the dura mater is implicated. Persons with running ears are in constant danger of such a complication. The meningitis occurring in cases of this kind

begins suddenly and with violent symptoms. Disease of the upper portion of the nasal cavity may possibly also develop meningitis.

Injury to the skull is one of the most frequent causes of purulent meningitis, and calls for surgical treatment. The bursting of an intracranial abscess invariably develops meningitis. All other cases of cerebral meningitis which are not traceable to a direct or palpable cause are probably due to the transmission of an infectious agent from a remote organ. "Metastatic meningitis" sometimes develops in the course of pneumonia, typhoid fever, erysipelas, articular rheumatism, empyæmia, and very rarely in pyæmia.

The anatomical changes in secondary meningitis are as a general rule limited to the convexity of the brain. The pia mater is injected and infiltrated with pus. Thick greenish-yellow pus is often found in the subarachnoid spaces and along the course of the meningeal blood-vessels. The arachnoid presents in places an opaque appearance. Spots of softening are often observed in the substance of the brain where the pia is adherent to the cortex.

Clinical History. It so often happens that the symptoms of secondary meningitis are mixed up with those of the primary affection that the latter attracts the first attention. In traumatic meningitis the symptoms of compression predominate. The base of the brain is involved, as Hutchinson has pointed out in cases where hemiplegia exists. In a case of meningitis of the convexity, which appears to be primary the symptoms resemble in nearly every feature those of the tubercular variety.

A precursory stage in meningitis of the convexity is either not well marked or entirely absent. There may be an initial chill, but usually the invasion of the disease is announced by violent headache and febrile excitement. The pain in the head is intense and dominates the attention of the patient. It may be limited to a fixed spot or be diffused over the whole head. There is often considerable elevation of the temperature, but it is generally varying. The patient is restless and irritable, he shuns the light and is annoyed by loud sounds; the eyes glisten; the pupils in the beginning are usually contracted; a circumscribed flush is seen in the face; the respiration is somewhat hurried or irregular; speech is slow and at times incoherent; an active or low delirium soon sets in; stupor develops, and finally a deep coma is established. Stiffness of the neck and retraction of the head may supervene.

Remarkable variations of the pulse are observed. It may be excedingly rapid at times, but quite as often it is very slow, irregular and intermittent.

Symptoms corresponding with implication of cranial nerves as in tubercular meningitis are noticed in individual cases, especially disturbances of the motor oculi. Twitching of muscles and paralysis of the hemiplegic type may likewise occur.

The course of the disease is often rapid, death taking place in two or three days, either preceded by coma or convulsions. Sometimes the fatal termination is postponed beyond a week. Recovery is exceptional.

Cerebral meningitis occurring in persons of an advanced age presents certain peculiarities. The symptoms develop insidiously; there is but slight headache, the

fever is moderate, delirium comes early and is often quickly succeeded by coma. In other cases there is merely mental confusion, a vacant expression of the face, tremor and rapid sinking of the vital powers.

The very dangerous cephalic symptoms sometimes met with in acute articular rheumatism resemble those of meningitis, though the post mortem appearances are not decisive.

Diagnosis. It is a difficult point of differential diagnosis to discriminate between meningitis and encephalitis. Practically it is of no moment, for the brain is more or less involved in all cases of cerebral meningitis. Diagnosis may be embarrassed when cephalic symptoms in severe cases of typhoid fever, pneumonia or general tuberculosis are exceptionally prominent. Careful analysis of the symptoms will usually overcome the difficulty. Those indicating the existence of meningitis include violent and persistent headache, the early onset of cerebral excitement, delirium, stupor, rigidity of the neck, ocular paralysis and the gravity which the disease quickly assumes.

Treatment. There is a great temptation in encountering the grave symptoms that characterize all forms of cerebral meningitis to adopt an energetic plan of treatment. Formerly it was the rule to practice venesection, now we are content to apply leeches to the head. More reliance is to be placed on the application of cold to the head. The ice helmet or irrigator answers this purpose far better than bladders filled with ice. In addition a woolen cloth wrung out in warm water may be wrapped around the lower limbs. In desperate cases it is recommended

to shave the scalp and to raise a blister. I have never seen a good effect from it. Iodide of potassium in frequent doses is indicated. The bowels are best kept open by small doses of calomel. Morphia injections may become necessary to allay extreme cerebral excitement.

CHAPTER XII.

LOCALIZATION OF CEREBRAL DISEASES.

The diseases which chiefly produce focal lesions of the brain are cerebral hemorrhage, softening from occlusion of cerebral arteries and cerebral tumor. The symptoms to which these diseases give rise do not depend on the nature of the pathological change, but on its locality. It makes no difference what the character of the lesion may be, which for example destroys any part of the pyramidal tract in the brain, it is always followed by hemiplegia. In an analogous way, whatever may be the nature of irritation or interruption that implicates an excitable area of the cortex, it will either cause a monospasm or monoplegia. Aphasia develops when a part of the speech mechanism in the brain is involved by any kind of lesion. The following brief summary of the pathological diagnosis of cerebral diseases includes the results of clinical observations and experiment.

Lesion of the central convolutions, produces hemiplegia of the opposite side of the body. Implication of separate parts of this region gives rise to corresponding forms of partial hemiplegia. We are thus enabled to localize disease in this region. It will be recollected, that the center of movement for the leg is in the upper part of the central convolution or in the paracentral lobule; for the movements of the arm in the middle third of the anterior

ascending convolutions ; for the movements of the facial muscles in the lower third of these convolutions and that for the tongue somewhat lower. Disease affecting any of these parts causes either isolated monoplegia or a combined form of monoplegia in conformity with the position and extent of the lesion. The commonest form of such a combined paralysis is that of the arm and face. The simultaneous paralysis of the leg and face has never been seen, for the reason that the intermediate arm center would not escape in a lesion affecting the other two centers.

If a cortical lesion of these centers causes irritation, then we have either "monospasm" (muscular twitching, tremor), or simultaneous spasm of the muscles of the face, arm and leg. It has been demonstrated that these motor centers are involved in cases of unilateral epileptic convulsions associated with hemiplegia of the same parts.

Disease of the second and third frontal convolutions cause no marked disturbance of function, but mental symptoms develop if these regions in both hemispheres are affected. Lesion of the third or inferior frontal convolution of the left hemisphere gives rise to the interesting phenomena of aphasia.

Parietal convolutions. It is uncertain whether cortical lesion of the parietal lobe, exclusive of the ascending parietal convolution, gives rise to any symptom. Impairment of the cutaneous and muscular sense has been observed in a few cases.

Temporal lobe. There exists satisfactory evidence, that extensive disease of the superior convolution of the temperal lobe gives rise to the aphasic symptom of "word deafness."

Occipital lobe. Experiments and pathological investigations leave no room for doubt that the occipital lobe contains a center of vision. Destruction of the occipital lobe does not cause paralysis.

Centrum ovale. Lesion of the centrum ovale may exist without causing any symptoms. This immunity is due to the circumstance that a sufficient number of nerve fibres remain intact to conduct innervation. If paralysis occurs, it cannot be distinguished from ordinary hemiplegia.

Island of Reil. The island is not infrequently involved in extensive disorganization of neighboring parts. Aphasia has been observed in exclusive lesion of the island.

Thalamus Opticus. Although the thalamus is very frequently involved in cerebral hemorrhage and occlusion of cerebral arteries there is considerable discrepancy among observers in regard to the symptoms peculiar to lesion of this basal ganglia. Cases are reported in which old standing lesions of this ganglia had remained entirely latent. In the great majority of instances the ordinary type of hemiplegia existed and was sometimes accompanied by hemianæsthesia. Hemiplegia occurring in lesion of the thalamus is probably due to a co-existent lesion of the internal capsule or of the corpus striatum. The occasional occurrence of hemianæsthesia in connection with a lesion of the thalamus, may be due to the implication of the posterior inferior portion of the internal capsule which contains sensory paths. Nothnagel, in an exhaustive analysis of published cases of lesion of the thalamus, comes to the conclusion, that a focal lesion of this basal ganglia never gives rise to motor paralysis.

The symptoms most frequently observed are post-hemiplegic chorea, athetosis and tremor of the paralyzed limbs. Visual disturbances, especially hemianospia, have also been observed. There exists a connection between the pulvinar (posterior extremity of the thalamus opticus) and the optic tract. It should also be mentioned that temporal and nasal hemianopsia are often diagnostic of a lesion in the frontal fossa, implicating the optic tract.

Corpus Striatum. Destruction of the Corpus Striatum from hemorrhage or softening gives rise to the most common form of hemiplegia. The exceeding frequency of lesion of this central ganglia is chiefly due to its vascular and friable texture. The caudate and lenticular nucleus of the striated body has been divided by Duret into different vascular districts, which, according to Charcot, may be separately involved and thus give rise to modified forms of hemiplegia. In all of them the internal capsule is more or less implicated. The lesion in the ordinary form of hemiplegia is situated in the central portion of the corpus striatum. The lesion in the severe form of hemiplegia affects the region of the corpus striatum where the internal capsule runs between the caudate and lenticular nuclei, or between the thalamus opticus and the lenticular nuclei. In the latter region the lesion may cause either a permanent hemianæsthesia alone, or the combination of hemiplegia and hemianæsthesia.

The diagnosis of a lesion of the corpus striatum is probably correct in the large majority of cases of hemiplegia, when the paralysis affects the arm, leg and face with temporary paralysis of the hypoglossus, no other

cranial nerve being affected. The paralysis of the face and tongue which usually attends hemiplegia in lesion of the corpus striatum shows the close proximity of the central paths of the facial and hypolgossus nerves to the motor fibres of the internal capsule, which are distributed to the extremities. It is now generally conceded that hemiplegia in lesion of the corpus striatum, is invariably dependent on implication of the internal capsule. Cases are reported in which an old focal lesion existed in the striated body, that had not given rise to hemiplegia. In such cases the internal capsule was evidently not involved.

Disturbance of sensation rarely accompanies hemiplegia in disease of the corpus striatum, but the following exceptional forms of paralysis have been observed in lesion of this ganglia:

1. Hemianæsthesia of the same side as the hemiplegia, but disappearing at an early date. The sensory paralysis in such a case is only an indirect symptom.

2. The hemianæsthesia like the hemiplegia is a permanent symptom.

3. Hemianæsthesia alone permanent or complicated with cross paralysis of special senses.

The anæsthesia affects the same side as the hemiplegia when these forms of paralysis co-exist. The cutaneous sensibility in such cases is impaired or entirely abolished on the affected side from head to foot, and is attended by impairment of sensibility of the corresponding mucous membranes and of the muscular sense. The most prominent symptom in regard to the implication of the special senses is cross amblyopia or amaurosis.

Pathological diagnosis in all cases of destructive disease of the corpus striatum, which are marked by the typical form of hemiplegia and attended by disturbances of sensibility, locates a lesion that involves the region of the posterior division of the internal capsule.

Charcot has drawn attention to the appearance of vaso-motor symptoms in hemiplegia due to disease of the corpus striatum. These are the occurrence of œdema and changes of temperature in the paralyzed limbs, besides some other symptoms relating to the sympathetic nerve. Although the intracerebral path of this nerve is undetermined, it is known that it passes the crura cerebri and must therefore occupy a part of the internal capsule.

Post-hemiplegic chorea is not due to lesion of fibres of the internal capsule, as it was formerly supposed, but to a lesion involving certain fibres that come from the thalamus optici.

Corpora Quadrigemina. These basal ganglia are supplied by arterial branches which are also distributed to adjacent parts of the cerebrum. This accounts for the meagre report of cases of hemorrhage in which this lesion was solely located in the corpora quadrigemina. The information concerning the special symptoms manifested in lesion of these bodies is gathered from the effects of tumors. Apart from the symptoms common to all cerebral tumors, it appears that lesion of a part or of all the corpora causes visual disturbances chiefly affecting branches of the motor oculi. In another class of cases symptoms of incoördination were prominent.

The Cerebellum. Hemorrhage and embolism of the cerebellum are of very rare occurrence. A clinical distinction between cerebellar and cerebral apoplexy is not practicable. The extravasation of blood into the cerebellum is always marked by violent symptoms. The hemorrhage affecting one of its hemispheres usually bursts into the fourth ventricle, involving the medulla and the pons. Histories of total destruction of the lateral half of the cerebellum from softening show complete latency, but symptoms of much significance are often witnessed from the presence of tumor, usually tuberculous, in the vermiform process and the crura cerebelli. The characteristic symptoms of lesion of the worm include a reeling swaying gait, vertigo and vomiting. Accessory symptoms, such as amblyopia, amaurosis, epileptiform convulsions and severe pain in the head are incidental to all cerebral tumors.

Certain remarkable forced movements are observed in lesion of the middle peduncle of the cerebellum (ad pontem). These phenomena consist of lateral deviation of the head and eyes and rotatory movements of the body. As these symptoms have not been observed in focal lesion of other parts of the brain, they may be considered diagnostic of disease of the cerebellum.

Pons Varolii. Hemorrhage of the pons is of rare occurrence and is not easily distinguished from hemorrhage of other parts of the brain. Speedy death is exceedingly common on account of the proximity of the medulla to which the extravasation usually spreads. Marked stertor and irregularity of the heart's action are witnessed from the outset. Deviation of the eyes and rotation of the head, which are always grave

symptoms in cerebral apoplexy, also occur in hemorrhage of the pons. The pupils are sometimes so extremely contracted, that in connection with the coma, opium poisoning is simulated. Symptoms of motor irritation quite often develop in pons lesions. They consist of partial spasm or epileptiform convulsions. In large hemorrhage of the pons there is usually a general relaxation of the whole muscular system. The most reliable evidence of the existence of a pons lesion, whether it be a hemorrhage, embolism or tumor, is a peculiar form of paralysis (alternate paralysis). It differs from typical cerebral hemiplegia in this, that the paralysis affects the upper and lower extremities of one side of the body, opposite to that of the lesion and the face on the same side as the lesion. In this form of paralysis there is sometimes implication of the hypoglossus. The abducens and the trigeminus are less frequently involved. If ordinary hemiplegia exists as is seen in lesion of the basal ganglion, the diagnosis of a pons affection is impossible.

Crura Cerebri. In hemorrhage or embolism of either the thalamus opticus or the inferior extremity of the corpus striatum, one of the crura is sometimes indirectly involved. This complication arises from the circumstance that these parts are supplied by branches of the posterior cerebral artery. But in a focal lesion confined to one of the cerebral peduncles a very characteristic form of paralysis is seen. The hemiplegia affects the extremities on the side opposite to the lesion, and the motor oculi of the same side as the lesion. All the ocular muscles to which branches of this nerve are distributed are paralyzed, and, in consequence, divergent

strabismus and diplopia develop. The facial and hypoglossal are usually involved on the same side as the extremities are. Anæsthesia is also noticed, always on the side opposite to the lesion, but it is a subordinate symptom. If a lesion of the crura does not produce the alternate form of paralysis, as stated, but only the usual form of a cross paralysis, it cannot be diagnosed.

Medulla Oblongata. The causes that give rise to focal lesions in different parts of the brain seldom affect the medulla oblongata. Hemorrhage of this organ, which is extremely rare, usually causes instantaneous death. If the patient survives the onset symptoms arise that cannot be discriminated from those presented by ventricular hemorrhage or a large clot in the pons. Cases of inflammatory softening of the medulla are described under the head of "apoplectic" or "acute bulbar paralysis," which is marked by a group of symptoms greatly resembling those of the chronic degenerative disease of the medulla known as "labio-glosso-laryngeal paralysis." The latter is an entirely different affection. The most prominent symptoms of the former include cross hemiplegia or paraplegia and very grave disturbance of the respiration and circulation.

APHASIA.

Anomaly of speech was the first symptom that led the way in the search for the "localization of the functions of the brain." Although it would be misleading to speak of a "center of speech," for the faculty of language requires the action of a complicated mechanism, yet pathological investigations and experiment leave no room for doubt

that definite parts of the cerebrum stand in intimate relation to the function of speech.

Aphasic disturbances of speech are manifested in many different ways, and various forms of the disorder are often observed in the same individual. In complete loss of the memory of language, the patient may know very well what he wishes to express, and correctly answer questions by gesticulations, but the words have escaped him. He may understand the meaning of the word that is spoken to him, or he may not. He may repeat a word or short sentence he has heard spoken, but he cannot of himself express a thought. Sometimes an aphasic patient uses one or several words, whether they have a meaning or none, as the only vehicle of his thoughts. I once attended a hemiplegic woman who made invariable use of the senseless word "ninny" and none other, whenever she desired to ask for something or intended to answer a question, although she understood spoken and written language. Other aphasics command a limited vocabulary, and are foiled when desiring to express a long or complicated sentence. It is an interesting fact that certain aphasics are able to speak with great facility under the momentary influence of strong emotional excitement. Graves reports the case of an aphasic patient who only knew the initial letters of words and had to consult the dictionary for the rest. Such forms of partial aphasia are very common. An aphasic person may forget his own name or those of his wife and children. In all these instances of aphasia the fault is on the intellectual side. Another important form of aphasia relates to the fault on the motor side. There are

aphasic patients who have a complete knowledge of words that correctly express their ideas and perfectly understand what is spoken to them, but they have lost the capacity for the movements by which language is articulated, although the muscles engaged in speech are sound and not paralyzed. A third chief variety of aphasia consists in the use of wrong words. The patient is not aware of this defect, and will often show his vexation at not being understood.

The question arises, what part of the brain is involved in aphasia? It is now universally believed, on the strength of reliable statistics, that in the vast majority of cases of aphasia the lesion is located in the left hemisphere. Of 260 cases of aphasia, Seguin found the lesion in the left hemisphere in 243; in the right in 17: a proportion of 14.3 to 1. In Lohmayer's table, out of 53 cases of aphasia there are 34 in which the third or inferior frontal convolution of the left hemisphere was diseased. This is the convolution in which Broca first discovered the aphasic lesion. The island of Reil comes next in frequency as the seat of the lesion. Other portions of the left hemisphere which are less often involved are the superior temporal convolution bordering on the fissure of Sylvius and the corpus striatum of the left hemisphere.

Kussmaul classifies aphasic symptoms under the following heads:

1. *Ataxic* (or motor) *aphasia*. There is incapacity of motor innervation of words. Patients have lost the power either totally or partially of coördinating the movements for articulate speech. They fully understand

the language spoken to them. The gross muscular power of the organ engaged in articulation is retained, but the ability to associate the movements required in uttering words is lost.

2. *Amnesic aphasia.* (This is the *sensory aphasia* of Wernike.) There is incapacity for the recollection of words as aggregate acoustic sounds. The idea is present, but the word is wanting, although articulation is at the service of the word.

3. *Word dumbness*, or the inability with good hearing and sufficiently preserved intelligence to understand words. This defect is also called "word deafness."

4. *Paraphasia.* There is an inability to properly connect word images and corresponding conceptions. Instead of words expressing the idea intended to be conveyed, they are misplaced, or confused word images present themselves to the aphasic.

Aphasic disturbances in individual cases may range from slight defects of speech to its entire abolition. In ataxic or motor aphasia, the mental images of words are intact. The stock of auditory representatives of words is retained. There is in this form of aphasia no actual loss of muscular power of the organ of speech, but the revival of the motor images of speech, that formerly readily responded with corresponding articulation is imperfect or abolished. The patient has lost the gift of adequately adjusting the associated movements for the formation of words. Many of us in the healthy condition have often experienced an analagous difficulty in states of mental excitement to find words to express our thoughts, which under ordinary circumstances would flow freely. It

appears as if the resource on which we always confidently rely for supplying us with words had for the moment failed us.

Mental images of words in amnesic aphasia cannot be recalled, or only to a partial extent. The general intelligence is clear, and the corresponding function for the articulation of words may be perfectly intact, but language, either vocal or written, has lost its meaning. The conceptions as they arise in consciousness do not excite memory of the corresponding words, or the words that are heard fail to evoke the mental images they represent. The patient hears the words, he is not deaf, but he does not understand what the words signify.

Word dumbness often occurs as the only indication of aphasia. A patient may fail, for example, to recall the name of his father or his own child, but perfectly understands who is meant when he hears the name spoken.

In paraphasia there is an interruption of the association of an idea and its corresponding word. A patient may ask for a spoon, when he means a knife, and persist that he is correct. Sometimes this use of wrong words renders his conversation unintelligible.

Agraphia. Alexia. Incapacity to convey thoughts in writing, as well as in speech, is more or less observed in amnesic aphasia. It is clear that words, which cannot be recalled, can as little be communicated in writing. The agraphia in some patients is modified in so far, that they can correctly copy the writing of others. Alexia is usually also present. The written characters convey no meaning to the patient. Finally it has been noticed that in aphasia there is sometimes a loss of the language of

gesticulation and pantomime. Patients wrongly indicate by their gestures the intention they wish to express.

TOPOGRAPHY OF THE SKULL IN RELATION TO THE SURFACE OF THE BRAIN.

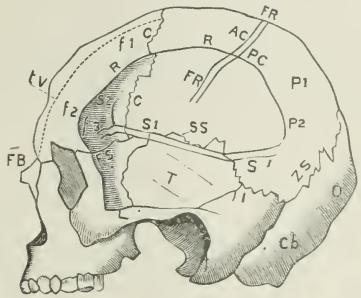

FIG. 27.

FR	Fissure of Rolando.	AC and PC	Anterior and posterior central convolutions.
S^1	Fissure of Sylvius.	O	Occipital lobe.
P1, P2	Upper and lower parietal convolutions.	T	Temporal lobe.
Cb	Cerebellum.	f_1	First frontal.
F	Frontal lobe.	f_2	Second frontal.
R, R	Parietal ridge.	f_3	Third frontal convolutions.
C, C	Coronal suture.	tv	Transverse vertical line.
FS	Fronto-Sphenoidal fissure.	FB	Angle of frontal bone.
LS	Lambdoidal suture.	SS	Squamous suture.

The area corresponding to the first, second and third frontal convolutions is bounded anteriorly by a transverse line ending at the angle of the frontal bone, behind by the coronal suture and below by the fronto-sphenoidal fissure.

The area corresponding to the anterior and posterior central convolutions is bounded in front by the coronal

suture, behind by a parallel line intersecting the parietal ridge and below the squamous suture. The superior portion of the central convolutions is above the parietal ridge, its inferior portion is below the ridge. The supramarginal convolution corresponds to the region of the parietal eminence.

The area of the temporal lobe is bounded above by the squamous suture. The occipital lobe is bounded in front by the lambdoidal suture.

CHAPTER XIII.

DISEASES OF THE BRAIN.

Cerebral Hyperæmia.

(Congestion of the brain.)

Etiology. Circulatory disturbances of the brain used to play an important role in the pathology of various cephalic symptoms for which no other cause could be assigned. It is not easy to understand under what circumstances arterial hyperæmia can occur, except it be from simple hypertrophy of the left ventricle of the heart. Full-blooded or rather "plethoric" people are popularly thought to be peculiarly liable to severe head affections. If by the latter be meant cerebral hemorrhage or cerebral embolism and it can hardly mean anything else, then morbid anatomy is at fault. Symptoms like headache, dizziness, throbbing of the carotids and a flushed face are often enough seen after a debauch or a heavy meal or in states of great mental excitement. It would not be hazarding too much to surmise that such causes tend to lessen arterial tension which induces increased blood pressure. The condition thus induced is probably meant by the phrase "a rush of blood to the head."

Passive congestion of the brain is better understood. It is invariably a secondary affection resulting from impediment to the return of blood from the brain. This con-

dition occurs in cardiac and pulmonary diseases, which obstruct the venous circulation. The face in well-marked cases has a purplish tinge and the lips are of a bluish color. The head feels heavy and full, the breathing is oppressed, there is a feeling of languor and the patient is indisposed to physical and mental exertion. Now, a brain that carries too much venous blood is a badly nourished and actually an anæmic brain, and the symptoms correspond with this condition.

Treatment. Persons who are liable to attacks of active congestion of the brain should be warned of the causes that bring them on. In the majority of cases it suffices to act briskly on the bowels and to apply leeches to the head. A course of aperient medicine, for which the bitter waters are well adapted, is often of much benefit.

The treatment of venous hyperæmia of the brain is chiefly that of the primary disease. The symptoms may be temporarily relieved by gentle aperients, cold to the head and warm footbaths. Formerly it was the fashion to practice abstraction of blood in these cases, and no doubt it often gave prompt relief. One is sometimes tempted to take up this practice again under certain circumstances. Some years ago I attended a young woman who suffered from constant violent headache, flushed face and heavy breathing, that depended on valvular obstruction of the heart. Nothing else gave her any relief but venesection, which, of course, could not be safely repeated as often as she demanded it.

CEREBRAL ANÆMIA.

Etiology. The sudden development of extreme cerebral anæmia is witnessed in profuse hemorrhages, failure of

the heart's action and profound mental impressions. It is manifested by great pallor of the face, loss of consciousness, slow and sighing respiration, a feeble pulse, dilatation of the pupils, blackness before the eyes, sometimes vomiting and general convulsions. The gradual establishment of cerebral anæmia is seen in chronic diseases, chlorosis, general anæmia, prolonged lactation and debilitating discharges.

Clinical History. The symptoms of chronic cerebral anæmia consist of various functional nervous disorders. Headache, vertigo and nausea are its constant attendants. Patients show mental irritability and complain of languor and weakness. They are liable to fainting fits. One of the most unpleasant symptoms is a feeling of drowziness. Hallucinations of sight and hearing sometimes develop in aggravated cases. Many of the ailments attributed to neurasthenia are really the effects of cerebral anæmia.

The Treatment when general anæmia exists is obvious. Attention must be particularly directed to the special cause that underlies the head symptoms. A disregard of the causal indications render all the reputed nervines and sedatives of no avail.

CEREBRAL HEMORRHAGE.

(Apoplexy.)

Etiology. Disease of the coats of the cerebral blood-vessels is the chief cause of cerebral hemorrhage. The degenerative change far more frequently consists of an arterio-sclerosis than atheroma. In consequence of the thinning of the vascular walls, miliary aneurisms develop which on rupture permit the effusion of blood. The

formation of these minute dilatations of cerebral arteries is favored by the absence of the adventitia. Charcot and Bouchard found miliary aneurisms in every one of the seventy-seven cases of cerebral hemorrhage they had examined. Increase of blood pressure will certainly facilitate the rupture of the aneurisms, but arterial tension alone without disease of the walls of the vessels is insufficient to cause the rupture. There are many exciting causes which in consequence of increased blood pressure lead to cerebral hemorrhage. It is a matter of experience that apoplectic attacks are often seen after strong physical efforts. Even relatively slight exertions, such as coughing, sneezing and especially straining at stool may burst a diseased vessel of the brain. The indulgence in alcoholic stimulants and the use of the cold plunge-bath may be followed by the same consequences. Mental excitement has sometimes a similar effect.

Cerebral hemorrhage is also often observed to occur in hypertrophy of the heart, especially in that form of cardiac trouble which develops in Bright's disease. In those cases characterized by the existence of the granular contracted kidney, the accompanying arterio-sclerosis facilitates the rupture of cerebral blood-vessels under the influence of the exaggerated action of the heart. In 55 cases of cerebral hemorrhage cited by Charcot, hypertrophy of the heart was found in 22. The kidneys were affected in 32½ per cent of 49 cases.

Extravasation of blood in the brain has been met with in scurvy, pernicious anæmia and leukæmia. Capillary hemorrhage of the brain occurs in cases of pernicious infectious diseases, smallpox, pyæmia, etc. This acci-

dent is insignificant in comparison with the grave nature of these diseases.

Alcoholism, syphilis and gout must also be considered as occasional etiological factors of apoplexy.

Age has a decided influence on the occurrence of cerebral hemorrhage. Persons under forty years are rarely attacked. Sex has a similar important bearing. The proportion of men who are subject to cerebral apoplexy far exceeds that of women.

A predisposition to cerebral hemorrhage must be admitted in the sense that certain families show a hereditary tendancy to arterial degeneration. The wide-spread belief that thick-set, short-necked men of a florid complexion are prone to be affected with cerebral hemorrhage does not accord with experience.

Anatomical Changes. Certain parts of the brain are more often subject to hemorrhage than others. Those parts in the vascular districts supplied by the middle cerebral artery are especially frequent situations of blood clots. They include in the order of frequency, the caudate and lenticular nuclei, the thalamus optici, the internal capsule and centrum ovale. Hemorrhage of the convolutions, the pons and cerebellum is much less frequent. The crura cerebri and the medulla are very rarely affected. The blood sometimes makes its way into a ventricle or escapes to the surface of the brain. It always tears up the brain tissue and then forms into a clot. The blood clots are of various sizes in different cases; they may not exceed the size of a pea or be as large as a man's fist. Very large hemorrhages flatten the convolutions.

A recent clot presents the appearance of a dark, pitchy mass, which is composed of the effused blood and the debris of the destroyed brain tissue. The contiguous portion of the brain is infiltrated with blood and is softened. The edges of the cavity where the clot lodges are ragged and irregular. The clot itself undergoes certain changes. It is gradually absorbed and the surrounding parts tend to resume their normal condition. New connective tissue develops in the cavity which gradually forms a cyst containing an ochre colored fluid. Such an apoplectic cyst is often found in old cases of cerebral hemorrhage. Sometimes the fluid contents of the cyst are absorbed, so that nothing remains but a cicatrix of a rusty color, from the intermixture of blood pigment.

Clinical History. A person is said to be threatened with apoplexy when certain symptoms or "warnings" make their appearance. Such premonitory symptoms are identical with those ascribed to cerebral congestion. It is probable they are observed in cases where the hemorrhage begins with a slow escape of blood. The onset of the apoplectic attack may be protracted if only a small twig of an artery ruptures. But cerebral hemorrhage that develops slowly is often fatal. The patient begins to feel dizzy and nauseous, his gait is unsteady, or his body inclines to one side; his mind becomes confused, his speech is thick, he feels drowsy and finally the stupor is succeeded by profound coma.

Sometimes the apoplectic condition is absent and hemiplegia, whether complete or partial, is the first sign of the hemorrhage. The compression exerted by the clot in such a case is supposed to be insignificant. At autopsies in cases of this description clots of no larger size

than a hazel-nut were found. In case II of "Andral's Observations" the hemorrhage occurred without an apoplectic attack. The seat of the clot was in the optic thalamus, of the size of a large cherry. Rosenstein reports a case in which neither unconsciousness nor paralysis occurred, but only motor aphasia. A coagulum of blood of the size of a hazel-nut occupied the white substance of the third left frontal convolution.

Ingravescent apoplexy is a form of cerebral hemorrhage characterized by the peculiarity that fatal coma develops for hours or several days subsequent to the appearance of hemiplegia and head symptoms. In cases published by Broadbent, the hemorrhage had begun in different parts of the brain and later either burst into the ventricle or broke through the pia.

Delayed apoplexy differs from the foregoing in the circumstance that the symptoms develop gradually. There is headache, dizziness, nausea, delirium; one arm or one leg or the whole of one side is paralyzed and finally unconsciousness sets in. The hemorrhage in such cases accumulates slowly until the amount of the effused blood brings on coma.

In severe attacks of cerebral hemorrhage there are no premonitory symptoms, the patient is suddenly and unexpectedly thrown into a condition of profound unconsciousness and utter insensibility. In popular language he has a "stroke of apoplexy." The patient may sometimes have just time enough to lie down or sink into a chair before he becomes completely comatose. He presents then the following appearance: The face is flushed, the eyes are watery, the pulse is full, but often slow, the respiration is noisy or stertorious, the mouth is

drawn in with each inspiration and the cheeks bulge out with every expiration. Many patients in this condition rapidly sink. The temperature is seldom altered, but immediately before the fatal termination it may quickly rise or fall. In very bad cases there is sometimes lateral deviation of the eyes and rotation of the head in the same direction. There is no characteristic change of the pupils. They may be normal, contracted or dilated. Whilst the patient is in the comatose condition there is complete relaxation of the muscles.

Examination of the urine after an ordinary apoplectic attack frequently detects traces of albumen and sugar. Quite often there is retention of urine.

Many patients never recover from the initial symptoms of cerebral hemorrhage. In this condition all the vital functions begin to fail. The stertor is replaced by rattling in the throat, the saliva runs down the chin, the respiration becomes exceedingly shallow, the body feels cool, the pulse is very slow and feeble or extremely rapid, the eyes are sunk deeply into their sockets, the cornea is opaque, the face is pallid and the cheeks are fallen in. Death may happen within an hour or the patient may linger for a day or two.

A more favorable termination is however witnessed in a large proportion of cases. Consciousness gradually returns, the patient gives signs of the clearing of the intelligence, he opens his eyes, looks around, changes his position and soon recognizes those that surround him. The amount of damage done to the brain after the subsidence of the shock can now be judged by the severity of the hemiplegia.

The distinction between direct and indirect symptoms

of cerebral hemorrhage is of practical importance. Hemiplegia is eminently the symptom which represents the direct effect of the focal lesion, but it may be an indirect symptom if the pyramidal tract is only secondarily implicated. The coma, which often quickly disappears in favorable cases, is evidently a cortical symptom, and is the result of shock or compression. As it occurs wherever the clot may be situated in the brain, the impaired consciousness must be considered in the light of an indirect symptom. But coma may have the significance of a direct symptom, when the lesion affects the prefrontal lobe. It may be stated in general terms that those symptoms are of a direct character that denote permanent disturbance of special functions of the brain. Those are of an indirect character which are transitory and subordinate. We know from experience the temporary nature of certain symptoms that attend cerebral hemorrhage, but often we are only able to determine their true character by the future course of the individual case. Under the head of indirect symptoms are usually included: disturbance of speech; early rigidity, transitory aphasia; changes of the urinary secretion; deviation of the eyes and myopia. The facial paralysis in hemiplegia is quite often an indirect symptom.

Hemiplegia is the clinical evidence of injury or indirect implication of the pyramidal tract in any part of its course in the brain. As the lesion in cerebral hemorrhage in the large proportion of cases involves the central ganglia and adjacent parts, it is obvious that the internal capsule being almost invariably involved necessarily gives rise to hemiplegia of the side opposite to the lesion.

The paralysis of the extremities is the most important

feature of hemiplegia. In some cases there is total paralysis of the arm and leg. In others there may only exist a slight hemiparesis. A variable degree of improvement of the paralysis takes place in numerous cases. It sometimes appears so early and well marked that probably the paralysis to a great extent was an indirect symptom. But usually the improvement is only partial. It begins as a general rule in the inferior extremity. Many patients are again able to walk with the weak leg, whilst the arm is still useless. Paralysis of the face is usually not a prominent symptom in cerebral hemiplegia. It is confined to the muscles supplied by the lower division of the facial nerve, and may be so slight that it is only recognized when the patient smiles or shows his teeth. In impairment of the hypoglossus, the tip of the tongue inclines towards the paralyzed side. The soft palate is sometimes affected. It hangs lower down, and is also directed towards the paralyzed side.

The tendon reflexes in nearly all cases of hemiplegia are exaggerated on the affected side. Vigorous contractions are excited when the tendons and bones of the arm and the leg are tapped. On the other hand the cutaneous reflexes are always diminished on the hemiplegic side.

Sensation is rarely impaired. Hemianæsthesia in connection with hemiplegia is observed in lesion of the inferior third of the internal capsule.

Contraction of the paralyzed muscles is often seen in the late stage of hemiplegia. This "late rigidity" affects the upper extremity more than the lower. The fingers are fixed in the position they assume when at rest; the upper-arm is adducted by the pectorales major and the forearm is in pronation. Moderate contraction of the

calf muscles of the leg is occasionally observed. It is held that the muscular rigidity in hemiplegia is due to secondary degeneration of the pyramidal tract. "Associated movements" constitute occasionally interesting phenomena of hemiplegia. It is observed that movements of the paralyzed muscles are excited when voluntary movements are carried out by the healthy side. Post-hemiplegic chorea is a rare symptom of cerebral hemorrhage.

Vaso-motor symptoms are observed soon after the apoplectic attack. The paralyzed limbs are warmer and redder than those of the healthy side. Congestion and even effusion of blood into the lung, pleura, endocardium and kidneys has been found in fatal cases.

By the trophic symptoms in hemiplegia are understood the development of malignant bed sores and painful joint disease.

Atrophy of the paralyzed muscles is noticed in old standing cases of hemiplegia, but it is not of the degenerative sort. The faradic reaction of the muscles is normal.

Impairment of the mental capacity in hemiplegic patients is sometimes very evident. It may escape notice, when they engage in ordinary affairs of life, but they are often incapable of sustained intellectual efforts. The mental weakness is usually recognized by forgetfulness of recent events and the undue display of emotional excitement. Old paralytics are frequently seen to weep or whimper without any apparent provocation.

Bastian gives the limit of four weeks, beyond which time little improvement of the paralysis may be expected. The general health is often fairly good, and many hemi-

plegic patients grow even corpulent. Later on, when they become bedridden and marasmus develops, they are apt to succumb to slight intercurrent affections.

Diagnosis. The differential diagnosis between cerebral hemorrhage and cerebral embolism will be discussed in connection with the latter disease. Although cerebral apoplexy presents the striking phenomenon of sudden loss of consciousness, which in the majority of cases renders diagnosis easy, yet the recognition of the true state of the case may remain uncertain in instances of very rapid death, say within half an hour or less. Cerebral hemorrhage does not usually kill instantly. Such a mode of death, preceded by coma, is more likely to be due to meningeal hemorrhage from a traumatic cause, the rupture of an aneurism or sudden failure of the heart's action in valvular disease. The most embarrassing cases however are those in which persons of whose previous history nothing is known are found in a state of coma resembling that of cerebral hemorrhage. Suppose a man deeply comatose is picked up in the street by a policeman, or a stranger at a hotel is found in a complete state of stupor, from which he cannot be roused, it would be hazardous to express a positive opinion concerning the true condition of such a patient. If in an instance of this kind the patient is advanced in years, if the artery at the wrist is rigid, or the signs of cardiac or renal disease are evident, there is great probability that the loss of consciousness is either the effect of cerebral hemorrhage or embolism. Still this does not exclude the possibility of injury to the head, deep intoxication, opium poisoning, uræmic or epileptic stupor or meningitis. It has often happened that the extreme prostration presented by a drunken man

has induced compassionate people to ply him with brandy, and contrariwise a man with a clot in his brain has sometimes been arrested by the police on the charge of drunkenness. If paralysis co-exists the difficulty of diagnosis vanishes at once, for whatever may have been the mode of onset, there is some lesion of the brain in the case. Very confusing complications occasionally arise that tend to lead diagnosis astray, as illustrated in the history of the following case: A man profoundly comatose was sent from the police station, where he had been placed in a cell the previous night, to the hospital. The house surgeon was informed that the man was seen in a deep state of intoxication, led about by a companion, and that he had a heavy fall on the curbstone. As he was still unconscious in the morning and could not be roused he was brought to the hospital in a patrol wagon. There was a strong smell of liquor about the man. The house surgeon on examination discovered no injury, and concluded that from all appearances more time would be required for the effects of intoxication to pass off. The man died three hours afterwards. At the autopsy an enormous meningeal hemorrhage and a linear fracture of the skull were found. Undoubtedly the fracture which caused the hemorrhage resulted from the fall during the state of intoxication.

Prognosis. Whether a patient who is down with cerebral apoplexy will come out of it or not depends on the mildness or gravity of the onset, or rather upon the quantity or localization of the hemorrhage. If the coma be not profound and the insensibility incomplete; if there is little or no stertor and the pulse and temperature keep within normal limits, the patient in all probability will recover

from the coma. The case is unpromising if the unconsciousness is profound; if there is marked and persistent stertor, shallow breathing, a retarded or irregular pulse, or a sudden rise or sinking of the temperature. A cautious prognosis must be given in regard to the future improvement of the paralysis, for it cannot be determined beforehand how much of the hemiplegia is a direct or an indirect symptom. The chances whether a patient who safely got over one attack of cerebral apoplexy will have another are much against him if his arteries are diseased or his heart or kidneys are affected.

Treatment. The routine practice of venesection in every case of cerebral hemorrhage is now generally abandoned, and for good reasons. Numerous patients recover from the shock for whom nothing has been done in the way of active treatment. There are, nevertheless, exceptional cases in which benefit may be expected from blood-letting, though it requires much tact and judgment to select them. The following symptoms in a young individual may indicate abstraction of blood: a cyanotic appearance of the face, a hot head, injected eyes, a vigorous pulse and a labored respiration. Leeches on the temples or on the mastoid processes may suffice in elderly persons. Active purgation from the effects of a few drops of croton oil mixed with a little syrup or a stimulant enema deserve a trial when the coma is very prolonged. An opposite treatment is advisable when there is great pallor of the face, a low temperature, a feeble pulse, slow and shallow respiration and widely dilated pupils. Patients in this condition are in danger of sinking rapidly, though often nothing can be done to prevent it. Wine, brandy, ether, musk and camphor should be steadily administered

against the threatened collapse. Should the patient be unable to swallow, which is usually the case, some of these remedies may be given with the hypodermic syringe. The failing respiration may be excited by dashing cold water on the face and bare chest; the skin may be rubbed with dry mustard or sinapisms be applied to the insides of the arms and thighs.

It suffices in ordinary cases of cerebral hemorrhage to place the patient in a comfortable position, to raise the head and shoulders and to keep off all disturbances from bystanders. An icebag should be applied to the head.

Formerly a great variety of remedies were employed with the object of promoting the absorption of the clot. The futility of meddling with the clot is now better understood. Confidence in the recuperative powers of nature to restore in some measure the damage done to the brain is fully justified by the improvement of the paralysis which is often witnessed. Much can be done to prevent or retard the renewal of the hemorrhage, with which the patient is always threatened, by measures that invigorate the general system. Patients should be advised to abstain from heavy meals, and from physical strains and mental overwork. The paralysis is often of such long duration that the physician should not neglect to continue an appropriate treatment, which prevents the recurrence of an apoplectic attack. A judicious symptomatic treatment after the shock has passed off relieves a number of distressing symptoms that more or less affect paralytic patients. Troublesome headache is sometimes promptly relieved by a blister behind the ears or nape of the neck. Insomnia may require chloral and bromide of potassium or an occasional small dose of morphia. Sometimes a

nightly rum punch does better in old paralytics, but large and often-repeated quantities of alcohol do mischief.

At the end of about four weeks, after all the initial symptoms have subsided, a systematic course of electric treatment is the only therapeutic measure from which improvement of the paralysis can be expected, although it would be difficult to decide, in favorable cases, what share the electricity had in the improvement. A feeble galvanic current should be cautiously passed transversely through the head for about two minutes in a position corresponding to the hemorrhagic focus. Stroking the paralyzed muscles with the kathode of the galvanic current is also advisable.

HEMIPLEGIA IN CHILDHOOD.

There is much uncertainty in regard to the primary cause of cerebral hemiplegia in children. The post-mortem appearances that are observed after the disease has existed for a considerable time show the effects of a pathological process that led to loss of substance of the brain (porencephalia). The degeneration of brain tissue implicates the motor tract. Probably the origin of the morbid change varies in different cases. It may have been hemorrhage, thrombosis, embolism or a congenital defect. Strümpel describes the disease under the name of "the acute encephalitis of children," and considers it analogous to the acute poliomyelitis of the same class of patients. The disease attacks children between one and six years of age, who had previously been in good health. It is sometimes seen to follow exanthematous diseases.

Clinical History. The hemiplegia in some cases is preceded by grave cerebral symptoms, fever, nausea, vomit-

ing, stupor and general convulsions. After the subsidence of these symptoms the little patient is seen to be paralyzed on one side. The hemiplegia gradually improves, but complete restoration is unusual. The arm is always more affected than the leg, but both limbs are arrested in their growth. The reflexes are exaggerated, contractures develop and the paralyzed muscles atrophy. Sensory disturbances are absent. Symptoms of motor irritation are observed in old standing cases, resembling hemichorea and athetosis. In some cases there is a relaxed condition of the metacarpal articulations that permits the fingers to be placed in positions at right angles with the back of the hand. Epileptiform convulsions develop at a late period. Sometimes impairment of the intelligence occurs, especially on the side of the moral instinct.

Treatment. During the acute stage, when the diagnosis is of course uncertain, the object of treatment is the mitigation of the cerebral excitement. Leeches may be applied to the temples or mastoid process, followed by cold to the head and a calomel purge. After the hemiplegia has become stationary, there is little to be expected from therapeutical measures. Improvement has been claimed in some cases from the iodide of potassium. Electricity and massage may possibly be of benefit.

CEREBRAL EMBOLISM AND THROMBOSIS.

(Softening of the Brain from Occlusion of Cerebral Arteries.)

Etiology. The usual sources of embolism are thrombi of the left auricle, concretions of the arch of the aorta, and very frequently particles of matter that detach from fibrinous masses on the valves of the left ventricle of the

heart dating from a previous endocarditis. The emboli on being washed away by the circulation, are carried to cerebral arteries and occlude them. Thrombi originate in diseased blood-vessels. If they develop in the cerebral arteries they directly produce occlusion. In the latter situation they often crumble and become thus another source of embolism. The blood-vessels in which the thrombi originate are either affected with arterio-sclerosis, atheroma or syphilitic endarteritis. Sluggishness of the circulation, which favors the stagnation of blood, is an important factor in the development of thrombosis.

If the collateral circulation is established which replaces the arterial blood cut off by embolism in a certain vascular territory of the brain no harm results, but if this does not occur it must necessarily happen that the part of the brain deprived of its blood supply softens and breaks down. Emboli are more frequently arrested in the large basal ganglia and the internal capsule than in other parts of the brain. This is due to the circumstance that those regions of the brain are supplied by branches of the middle cerebral artery, which sparingly anastomose. The left middle cerebral artery is rather more frequently affected than the right.

The process of softening in cerebral embolism takes the same course as embolism of the lung, spleen and kidney, but no infarcti are formed. It begins with anæmia of the area of the brain that has been occluded, which is soon followed by disintegration of the affected brain tissue until it is reduced to a pulpy mass. A focus of softening may present a reddish appearance from the intermixture of blood of neighboring vessels, or it is of a white or yellowish color. A recent spot of softening, when

examined with the microscope, is seen to consist of the debris of the destroyed nerve elements and vestiges of neuroglia and vessels. Changes analogous to those taking place in the blood clot of cerebral hemorrhage are observed in the dead brain tissue so that it is difficult to make the distinction in old cases. The disintegrated mass is absorbed and replaced by cicatrical tissue, which hardens and atrophies the convolutions. Deep depressions are found when the softening occurs on the surface of the brain.

Clinical History. An apoplectic attack is often the first intimation of the occurrence of cerebral embolism in individuals whose general health had previously appeared to be good. The loss of consciousness may be as complete and come on as suddenly as in cerebral hemorrhage, though it is more frequently ushered in by general convulsions, delirium or vomiting. But the onset may be slow and the coma incomplete. Patients can be roused for a moment, they look about and may answer questions, but soon fall back again into the former dazed condition. This difference in the severity of the onset probably depends on the size of the artery that is occluded.

The onset in thrómbosis is usually slow and made up of frequent attacks of vertigo, faintness and mental weakness. This condition is generally seen in elderly people who have previously shown evidences of failing health or in those who have exhibited signs of premature senile decay. A deterioration of the physical and intellectual powers may have been noticed for weeks or months before the final breakdown. There is often much headache, dizziness, unsteadiness of gait, a tendency of the body to

lean to one side, now and then a marked incoherence of speech, or rather a misplacement of words, a feeble memory, and sensations of numbness and formication in some of the limbs. These prodromic symptoms may never develop into an apoplectic seizure, but there is a history of occasional paresis of the face, weakness of an arm or of a leg, mental impairment and finally senile dementia. The spots of softening found in the brain in cases of this kind result from thrombosis of atheromatous arteries. Heubner observed obliterated cerebral arteries from thrombosis in syphilitic young persons.

The abrupt development of the apoplectic coma in cerebral embolism and thrombosis does not admit of an easy explanation, and the same applies to the rather frequent occurrence of epileptiform spasms. Probably these symptoms depend on the sudden obstruction of a large arterial branch. A fatal termination may as quickly follow the onset as it does in cerebral hemorrhage, but patients have often been seen to continue for days in an apparent precarious condition of unconsciousness and still recover.

The chronic course of softening requires no separate description, as there is a history of hemiplegia and accessory symptoms analogous to those of cerebral hemorrhage.

Diagnosis. However difficult and often impossible it may be to make the distinction between the clinical history of softening and hemorrhage of the brain, there are certain considerations and points of differential diagnosis that tend to turn the balance in favor of the one or the other. The apoplectic condition is frequently as well pronounced in cerebral embolism as in hemor-

rhage, but its prolonged duration is of less serious import in softening. 2. A severe onset, accompanied by a flushed face and strong pulsation of the carotids, indicates hemorrhage rather than embolism. 3. Coma is more likely to be due to embolism than hemorrhage if the patient be a young person, especially if there is a history of syphilis or inflammatory rheumatism. 4. Mental disturbance is more common in occlusion than in clot. 5. A hemiplegia which disappears in a few days can hardly be due to hemorrhage, for it is far more probable that a paralysis in such a case resulted from embolism that passed off as soon as the collateral circulation was established. 6. Thrombosis may be inferred to exist in syphilitic patients and in senile softening when the physical and mental deterioration slowly develops.

Prognosis. Although patients often recover from the immediate effects of cerebral embolism, they are liable to its recurrence, as the one attack shows the existence of diseased blood-vessels. Even if no other attack follows they enter upon the stage of chronic softening, which may last for years, but tends to a fatal termination.

Treatment. If it were possible to determine with certainty in a case of cerebral apoplexy the existence of embolism or thrombosis, efforts might be made, by means of stimulants to restore the circulation in the affected part of the brain. But it involves a great risk to ply the patient with brandy when a hemorrhage may possibly be going on. Beyond good nursing and paying attention to the secretions, but little can be done, after the subsidence of the initial symptoms. The special treatment of the paralysis is the same as that detailed in the previous section on hemiplegia.

CEREBRAL TUMORS.

Etiology. Intracranial tumors are of the same histological structure as neoplasms in other parts of the body, and their cause is as little known. Adventitious growths develop in persons who have shown no signs of impaired health. Men in the middle period of life are oftener affected than women. The solitary tubercle is the tumor usually found in children.

Varieties of Cerebral Tumor.

1. *Glioma.* This tumor consists of a hyperplastic growth of the connective tissue of the brain, variable in size, of a grayish or reddish color, seldom sharply defined and often very vascular, so as to give rise to hemorrhage. Gliomata usually occur in the medullary substance, and often also in the central ganglia.

2. *Sarcoma.* The various forms of sarcoma generally develop in the dura mater and periosteum of the skull, most frequently at the base, where, in consequence of the irritation and compression exerted on the parts in that region of the brain, very marked symptoms arise. Total blindness of one eye in connection with paralysis of ocular muscles occurs in sarcoma and glioma of the orbit.

3. *Tubercle.* Solitary and multiple tubercle invade different parts of the brain, but more frequently the cortex, the cerebellum and the pons. These tumors are usually of the size of a cherry, but sometimes as large as a hen's egg. Before the discovery of the tubercle bacilli it was difficult to distinguish tubercular masses from gummata.

4. *Carcinoma.* Primary cancer of the brain occurs, but usually it is secondary, developing in association with malignant growths in the breast, lung and pleura.

Tumors of the brain of rare occurrence include *lipoma, cystic growths, hydatids and psamoma.*

Clinical History. A tumor, wherever situated within the cranium and independent of its histological character, gives rise to general symptoms, chiefly due to the degree and amount of compression or irritation it exerts in its immediate vicinity or remote parts of the brain.

General symptoms. Nearly all the cephalic symptoms of brain tumor are the clinical manifestations of crowding and flattening of the convolutions, abnormal tension of the dura mater and circulatory disturbance. The greater the size of the tumor the more pronounced and numerous are these symptoms. Besides, intracranial pressure of the venous trunks causes ventricular effusion.

1. Headache is the earliest and most constant symptom. The pain is generally very severe and marked by exacerbations. There is no uniform relation between the seat of the pain and the particular location of the tumor, but in persistent occipital headache the growth has often been found in the posterior fossa. Patients evince the violence and persistency of the pain by groans and corrugation of the brows even when in a dazed condition. They are sometimes seen to grasp the head with the hands and run to and fro in a frantic manner. Sleep is difficult to procure.

2. Vertigo stands next in prominence as an early symptom. The dizziness is sometimes so aggravated that patients stagger and reel as if they were drunk.

3. Cerebral vomiting is a very troublesome and intractable symptom, and is apt to come on as soon as the patient rises from bed.

4. Remarkable slowness of the pulse is frequently noticed. Passive congestion of the cerebral venous trunks sometimes brings on sudden faintness or a momentary attack resembling apoplexy.

5. A marked symptom in severe cases is mental perturbation, which is exhibited in the expression of the face. The patient appears stupefied, he is slow in answering questions and forgets what he has been saying or doing a moment before.

6. Epileptiform convulsions occurring in brain tumors may be grouped among the general symptoms, but frequently the spasms are due to a tumor in the excitable motor areas of the cortex. Partial spasm affecting facial muscles or an arm also indicates the existence of such a definite focal lesion.

7. There are few cases of cerebral tumor which do not develop optic neuritis. This is a symptom of pressure exerted by the growth wherever located in the brain. It is therefore important to make an ophthalmoscopic examination when a cerebral tumor is suspected to exist. Choked disk is now generally supposed to be caused by the cerebro-spinal fluid being forced into the lymph-sheath of the optic nerve. Sight may be unaffected for a long time, as the atrophy of the disk is often a late process. Amblyopia tending to amaurosis is however in some cases an early symptom of cerebral tumor, and the oculist may be the first to discover the true cause of the disturbed vision.

Focal symptoms of cerebral tumor. A brain tumor may during its whole course excite no other but general symptoms. Such is the history in cases where the tumor is lodged in the centrum ovale or in one of the basal ganglia. It is also a peculiarity of cerebral tumor that symptoms which indicated implication of definite parts of the brain may disappear. A monoplegia, or even a hemiplegia, may pass away and not return. Such an occurrence is explicable on the supposition that a change in the tumor reduced its volume, and thus the pressure was removed from the particular part that had caused the paralysis. Aphasïa in tumor of the left hemisphere may in this manner be only a temporary symptom. Partial spasm of paralyzed limbs, which is rather common in brain tumor, may disappear under analogous circumstances.

It is in tumors at the base of the brain that special focal symptoms always make their appearance. A tumor in this situation affects more or less the cranial nerves where they emerge from the brain. The paralysis of these nerves shows the peripheral type. The nerves which are subject to the paralysis include the different branches of the motor oculi, the trochlearis, the abducens, the facial, the hypoglossal and the trigeminus. Each of these nerves may be separately affected or several of them are simultaneously paralyzed. A large proportion of cases of paralysis of one or more of these cranial nerves, where the existence of a brain tumor is suspected, result from syphilitic gummata.

To describe the special symptoms significant of tumor in other parts of the brain would merely repeat what has already been stated in regard to the pathological diag-

nosis of cerebral lesions. An exception might be made in reference to cerebellar tumor, which is more common in children than adults. The general symptoms are sometimes violent, especially the constancy of the pain in the occipital region and the incessancy of vomiting. Stiffness of the neck is often well marked. Choked disk is nearly always present. Besides, there is not unfrequently unsteadiness of the gait, amblyopia, anosmia and deafness.

Course. At autopsies tumors of the brain have been found that had remained entirely dormant, but such instances are rare exceptions. Sometimes a cerebral tumor abruptly develops symptoms indicating a serious brain trouble. Usually the onset is slow, and the disease runs a chronic course lasting for months or years. The general health is gradually undermined by constant suffering and wakefulness. Towards the close amelioration is brought about by the decline of the mental activities and the deepening of the stupor. A fatal termination sometimes rapidly ensues from a succession of epileptiform convulsions.

Diagnosis. The continuous and progressive course of cerebral symptoms, in which a severe headache that appears to baffle all remedies constitutes a prominent feature, would suggest the existence of a cerebral tumor. Such a diagnosis is greatly strengthened if epileptiform convulsions also occur, and is nearly brought to a certainty if choked disk is discovered. A cerebral abscess may come near to presenting a similar set of symptoms, but this disease can often be traced to a traumatic or some other palpable cause. Its duration is shorter and choked disk is much less frequently found than in tumor.

The implication of cranial nerves is as likely to be due to syphilitic degeneration as to a sarcoma at the base of the cranium. The differential diagnosis would meet here with difficulties, though the association with more of the general symptoms, and especially the presence of choked disk, would tend to decide in favor of tumor. Multiple sclerosis may possibly imitate brain tumor, but the former is the more chronic disease and manifests other symptoms which are not common to cerebral tumor. In very rare instances a chronic hydrocephalus, the result of a circumscribed meningitis, has been found that during life had counterfited a brain tumor. In children, who are frequently subject to headache and convulsions, the probable existence of an isolated or multiple tubercule should be taken into consideration.

The diagnosis in regard to the character of a cerebral tumor is sometimes possible, but far more frequently it is impracticable. A predisposition to tubercular infection in children would influence diagnosis in favor of a tubercular tumor. A history of syphilis would do the same for a gumma. The existence of cancer in a remote part of the body suggests malignant tumor of the brain. Cerebral aneurism, which seldom causes choked disk, is marked by paroxysms of violent headache and frequent attacks of vomiting.

Prognosis. All intracranial tumors, irrespective of their histological nature, tend to a fatal termination. Exception must be made in regard to syphilitic gummata. A guarded opinion is advisable as to the duration of the disease, though two years is about the utmost limit. Death may happen at any moment, either from hemorrhage or epileptiform convulsions.

Treatment. If anything is to be expected from treatment in cerebral tumor it is the iodide of potassium that holds out some prospect of success. This remedy is not only the most proper and efficacious one in gummatous infiltration, but growths of a different nature may sometimes be influenced by its effects. In explanation of the benefit sometimes derived from the remedy, it is supposed that tumors often give rise to symptoms not so much dependent upon their size and location as upon the inflammatory softening and œdema they produce.

There remains still much scope for symptomatic treatment. Anodynes for the relief of pain are indispensable. The bromides exert some control over the convulsive paroxysms. Alcoholic stimulants, tea and coffee should be strictly prohibited.

ACUTE AND CHRONIC ABSCESS OF THE BRAIN.

(Encephalitis.)

Etiology. 1. Traumatism of the skull is one of the chief causes of cerebral abscess. Complicated fractures that destroy brain tissue and permit the ingress of infectious materials invariably give rise to collections of pus within the cranium. It is equally a matter of experience that cerebral abscesses may develop from contusion and laceration of the scalp, although the skull is uninjured. Cases of this kind usually come first under the notice of the general practitioner, for the extension of the inflammation through the bones is often a late process. 2. Disease of the petrous portion of the temporal bone originating from inflammation of the middle ear is next in the order of frequency a cause of cerebral abscess. Children and

youths who have running ears are in constant danger of being either suddenly attacked with acute cerebral abscess or meningitis. The abscess in this class of cases is situated in the parietal lobe, or in one of the hemispheres of the cerebellum, and is usually associated with suppurative phlebitis of a sinus. 3. The metastatic abscess is generally multiple and of small size. It develops during the course of pyæmia and is of subordinate clinical importance. Abscesses of this character sometimes form in purulent bronchitis. There still remains a class of cerebral abscess for which no cause can be assigned.

Anatomical Changes. A cerebral abscess may merely present the appearance of a small spot of softening. If there is only one it is generally of a large size. The greater part of a hemisphere is sometimes found transformed into a collection of pus. The pus is of a greenish-yellow color or reddish from the mixture of blood globules. It may be odorless or offensive and consist of the remnants of destroyed nerve tissue. The cavity in which it is lodged presents irregular walls. The surrounding parenchyma of the brain, to a greater or less distance, is softened and infiltrated with an abundance of granular corpuscles. A large abscess near the surface of the brain may sometimes be recognized by fluctuation. When centrally located it not unfrequently bursts into a ventricle.

A cerebral abscess of old date is usually found encapsulated. The cyst-wall is formed of fibro-cellular tissue. Its inner layer consists of a smooth pyogenic membrane. The contents of a chronic abscess often resemble a thickened cheesy substance.

Clinical History. An acute cerebral abscess, whether directly caused by an injury or any other cause, which

after a period of latency suddenly kindles into activity, gives rise to symptoms that cannot be distinguished from those of acute meningitis. There is a high grade of febrile excitement preceded by a chill or rigor, a violent or deep, dull pain of the head, delirium and finally stupor and coma. Rarely does an acute cerebral abscess run into the chronic stage. Precisely the same group of symptoms of a fatal tendency attends the class of encapsulated abscess, which either rupture into a ventricle or escape to the surface of the brain.

The clinical history of chronic abscess is marked by a course of symptoms of much less severity. There are often attacks of violent headache, probably brought on by incidental exciting causes that disturb the cerebral circulation, and is attended by the irregular occurrence of chills. Nausea is sometimes a troublesome symptom that often terminates in vomiting. The general symptoms in many cases may amount to a feeling of ill health, nervous weakness and a gradual emaciation. But the persistence of pain in the head, the recurrence of febrile disturbance, occasional delirium, the sudden attack of convulsions and a deepening stupor tell the gravity of the underlying disease.

The focal symptoms vary with the location of the abscess. An abscess rarely occupies the basal region of the brain and hence the cranial nerves in that situation are seldom affected. A large abscess in the frontal lobe may give rise to no special symptoms at all if it does not involve the central convolutions. But an abscess in the latter region of the brain is very likely to cause monoplegia or hemiplegia, often attended by epileptiform spasms. The appearance of paralysis affecting the motor oculi, the

hypoglossus or facial nerve, shows the encroachment of the abscess toward the base of the brain. Hemianospia if sought for will probably be discovered if the abscess occupies the frontal fossa. Paraphasia has been observed in abscess of the temporal lobe.

The duration of chronic cerebral abscess varies. Recovery is of exceptional occurrence. The fatal termination may slowly ensue from aggravation of the general symptoms. It rapidly takes place when changes in the abscess develop the symptoms of acute meningitis.

Diagnosis. Cerebral abscess admits of being diagnosed with considerable certainty when cephalic symptoms of a general character are traceable to a previous injury to the skull, or are associated with disease of the ear or the upper nasal cavity. The differential diagnosis between cerebral abscess and tumor is not always easy. The etiology of the individual case affords the most reliable information. Choked disk is far less common in abscess than tumor. Febrile excitement, especially repeated chills, speak in favor of abscess. The sudden occurrence of violent head symptoms that indicates a very grave brain trouble suggest abscess.

Treatment. Since the introduction of systematic anti-septic treatment, surgeons are emboldened to trephine the skull to evacuate an abscess. Whenever such an operation is feasible it holds out the only prospect of relief. But a procedure of this kind is often impracticable in view of the difficulty of locating the abscess with sufficient precision, or to reach it with safety to the patient. Nothing else is left when an operation is not justifiable than symptomatic treatment. Leeches and cold to the head serve to calm the cerebral excitement. The chief

remedies to allay the headache and spasms are morphia and chloroform inhalations.

CEREBRAL SYPHILIS.

Etiology. Syphilis involves the nervous system at a late period, usually when the outward evidences of the constitutional disease have disappeared. It is probably owing to an exciting cause or predisposition to nervous affections that leads to the development of cerebral syphilis.

Anatomical Changes. Two varieties of gummy tumors are recognized, though they frequently occur in combination. 1. The circumscribed syphilitic tumor consists of a dense cheesy mass of a yellowish color. Histologically it is a granular tissue more or less vascular. The gumma generally originates in the dura mater and sometimes in the substance of the brain. In the latter situation it is difficult to distinguish syphilitic granulations from tubercles. 2. The second variety consists of a soft grayish mass, irregular in outline and blending with the surrounding healthy tissue. It is made up of granular cells and is very vascular. This syphilitic infiltration is sometimes transformed into fine cicatrical tissue.

Much importance attaches to the specific changes which the walls of cerebral blood-vessels undergo. Branches of the middle cerebral and posterior cerebral arteries are particularly prone to be attacked. The vascular walls become opaque and gradually assume a whitish color. Spots of a dense consistence form in portions of the vessels and gradually change them into a uniform cartilaginous hardness. This change begins as an endarteritis and finally causes obliteration of the vessels through the for-

mation of thrombi which arrest the circulation in certain areas of the brain. The diseased arteries on yielding to the blood pressure may rupture and produce hemorrhage.

Clinical History. Precursory symptoms, though not characteristic of the specific causation, often make their appearance during a considerable length of time before the complete development of the grave forms of cerebral syphilis. The cephalic symptoms of this order include headache, vertigo, insomnia, neuralgiform pains of the head and face (worse at night) and occasionally slight mental impairment. The occurrence of paralysis affecting cranial nerves is of greater diagnostic significance. Some of the ocular muscles are especially prone to be involved. Ptosis has become notorious as a sign of syphilis. The facial nerve is quite often paralyzed and next in frequency, the sixth.

The onset of cerebral syphilis is often announced by a sudden epileptiform seizure or an apoplectiform attack, followed by hemiplegia. In other cases the initial symptom is a marked somnolence or a peculiar obtuseness of the intelligence.

Severe implication of the higher nerve centers are frequently witnessed during the whole course of the disease and may at any time lead to a fatal termination. It is remarkable, however, how often the grave symptoms of cerebral syphilis disappear without being succeeded by serious consequences, especially if a prompt and energetic treatment be adopted.

One of the marked types of cerebral syphilis presents a group of symptoms characteristic of brain tumor. It is not necessary to go over the same ground again in reference to this subject. General and focal symptoms, as has

already been mentioned, characterize a brain tumor irrespective of its etiology and histological structure. The only point in this connection which is of practical importance is the circumstance that the favorite situation of a gummatous tumor is the basilar region of the brain, where the cranial nerves are exceedingly prone to be involved.

Another type of cerebral syphilis simulates the clinical features of the "general paresis of the insane," though the pathological changes are not extensive. Occasionally syphilis of the brain appears under the guise of multiple sclerosis.

Diagnosis. The chief reliance in diagnosis of cerebral syphilis rests on the previous history of the patient. An accurate search for the objective symptoms of the venereal disease is all the more necessary as its characteristic signs, when the nervous system is affected, are usually not apparent. None of the cerebral symptoms dependent on syphilis are reliable guides of diagnosis. More information is furnished by the order of occurrence of the several symptoms, their peculiar grouping, the presence of isolated forms of paralysis and their irregular combinations. It is not usual to encounter the sequence and assemblage of symptoms peculiar to cerebral syphilis in ordinary gross lesions of the brain. The age of the patient is of some diagnostic importance. An apoplectic attack or hemiplegia in a young person, if embolism, due to valvular disease of the heart, can be excluded, is more likely owing to syphilitic endarteritis than to ordinary cerebral hemorrhage.

Prognosis and Treatment. Of all the grave affections of the nervous system those of a syphilitic origin afford relatively the most favorable prognosis. Permanent destroy-

ing lesions, due to the effects of the venereal poison, are of course as little amenable to curative treatment as those from any other cause. But even in very unpromising cases some measure of improvement is often attainable if the specific treatment is followed up with promptness and perseverance. The only question that can arise in relation to the treatment refers to the choice of the anti-syphilitic remedies, mercury or iodide of potassium. Mercurial inunction is decidedly the quickest and safest way to bring the system under the influence of the specific, though this method is better adapted for hospital than private practice. The continuation of the inunction must be governed by the effects that follow. The potass. iod. is advantageously employed at the same time. This remedy alone often suffices, but it must be given in large doses and for a long time. Hutchinson's plan of using the mercury with chalk is a good one when inunction is objectionable.

CHAPTER XIV.

MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD.

(Disseminated Sclerosis. Sclerose en-plaque.)

Etiology. Multiple sclerosis was formerly confounded with different forms of disease effecting the nervous system. We are indebted to the French school of neurologists for an accurate description of its symptoms. A hereditary influence is evident in some cases. The disease is most frequently seen in young persons between the ages of 20 and 35 years, but it has also been observed in children under 10 years. It occurs oftener in the female than the male sex. The supposed exciting causes are exposure, physical overexertion and emotional disturbances.

Clinical History. In consequence of the very gradual development of the disease, no conspicuous symptoms make their appearance in the early stage, but a very marked group of symptoms characterizes typical cases.

A peculiar tremor constitutes the most prominent symptom. It is owing to this symptom that the disease was formerly confounded with paralysis agitans. But the trembling in sclerosis is only excited when the patient

attempts to carry out a movement. As soon as he relinquishes the effort, the trembling ceases. The tremor in paralysis agitans is of a more limited sweep, and is continuous whether the patient is at rest, or in motion. In sclerosis no tremor is observed, as long as the patient is quiet. When he rises from his seat, or moves a limb the trembling begins. When grasping a glass of water to bring it to the lips, the arm shakes violently, the hand takes a wide excursion and the water is spilt. If the glass reaches the mouth, it clatters against the teeth.

A singular defect of speech, analogous to the tremor is another conspicuous symptom of sclerosis. Words are pronounced in a hesitating, drawling manner, a pause being made between each word or syllable, as in scanning. The voice is monotonous and a tremulous movement of the lips and tongue is observed during speech.

Nystagmus is noticed in about half the cases. The eyelids oscillate in a lateral direction, especially when the patient is intent on looking at a remote object.

The normal muscular power is often well preserved for a long time, but paresis of the limbs is sometimes an early symptom and may terminate in complete paralysis. A spastic condition of the muscles is a far more constant and conspicuous symptom. This motor disturbance is chiefly due to exaggeration of the tendon reflexes. It causes stiffness of the limbs, and when associated with considerable paresis that peculiar gait is witnessed which is characteristic of spastic spinal paralysis. At a late stage, walking is quite impossible. Permanent contractures develop in the lower extremities, so that in well marked cases, the heels touch the nates, and the thighs are firmly flexed on the trunk.

Disorders of sensibility are insignificant or entirely absent. Sometimes there is slight bluntness of the skin, but anæsthesia is of the rarest occurrence. The cutaneous reflexes are normal.

Ocular symptoms are only occasionally present. A serious complication is the development of optic neuritis terminating in atrophy of the disk.

The functions of the bladder, the rectum and sexual organs are not disturbed in a typical case.

Cerebral symptoms sometimes arise during the course of the disease. Patients begin to exhibit mental weakness, and often break out into fits of sobbing without an assignable cause. Headache and vertigo are frequently complained of in the early stage.

Anatomical Changes. According to Leyden, the pathological process of multiple sclerosis is of the nature of an interstitial chronic myelitis, which at first affects the neuroglia and afterwards involves the nerve elements. Rindfleisch believes that the anatomical change begins in the blood-vessels and secondarily implicates the nervous tissue. The nodules are recognized as hard yellowish patches scattered through different portions of the brain and cord, and chiefly occupy the surface of these structures. They are found in great abundance in the spinal cord and pons, and are less numerous in the medulla, the cerebellum and the central ganglia. The nodules, when examined with the microscope, are seen to consist of reticulated connective tissue in which a few nerve fibres are still visible. The coats of the blood-vessels are thickened. The neuroglia is changed into a dense fibrous tissue which surrounds and compresses the

remaining nerve fibres, but their axis cylinders are preserved for a long time.

Atypical cases of Sclerosis. A certain number of cases come under notice which deviate in their symptomatology from the typical form of sclerosis. It has often happened that patients presented symptoms, that gave no intimation of the existence of sclerosis, but which post-mortem examination showed to have been due to this pathological condition. The reason of this anomaly is the localization of the sclerosed patches in special parts of the nerve centers, which would naturally give prominence to corresponding symptoms. It will suffice to give a brief summary of these atypical cases of the disease.

1. Cases resembling loco-motor ataxia, in which the nodules invade the posterior columns of the cord.

2. Apoplectiform attacks followed by hemiplegia, and epileptiform convulsions. In these cases the nodules preponderate in the brain.

3. The symptoms of chronic transverse myelitis, including paraplegia, anæsthesia and implication of the sphincters of the bladder and rectum. Here the patches involve the thickness of the cord.

4. Nodules situated in the lateral columns of the cord give rise to spastic spinal paralysis. Multiple sclerosis in combination with muscular atrophy indicates the extension of the lesion to the anterior gray cornua and the picture of amyotrophic lateral sclerosis is presented. Implication of the medulla oblongata gives rise to symptoms of bulbar paralysis.

Diagnosis. A typical case of multiple sclerosis offers no difficulty to diagnosis, if the peculiar combination of

symptoms characteristic of the disease is borne in mind. Atypical cases are certainly embarrassing, but the very medley of symptoms showing functunal disturbance of different parts of the nervous system is suggestive of multiple sclerosis.

Prognosis. The only encouraging features in the prognosis of multiple sclerosis are the occasional pauses and periods of improvement during the course of the disease. There are no reliable reports of recovery.

Treatment. Therapeutics must be contented with efforts to improve some of the symptoms. Temporary benefit may be expected from electrical treatment. Remedies which are useful in chronic myelitis are indicated.

PARALYSIS AGITANS.

(Shaking Palsy.)

Etiology. Paralysis agitans is a disorder of advanced life, although it is occasionally seen in young people. A hereditary influence is not very evident. Persons, who have passed through much hardship in life are considered especially liable to be affected. An affection resembling paralysis agitans is sometimes seen to develop after injuries and acute febrile diseases.

Clinical History. The essential symptoms of paralysis agitans include tremor and stiffness of muscles. Tremor is the earlier symptom. It usually begins in one hand and gradually becomes general. The patient is, at the commencement, able to control the shaking by a strong voluntary effort or by leaning his body against a support. Mental excitement increases the trembling, but active exertion appears to diminish it. The tremor ceases during

sleep. It may for a considerable length of time be confined to one arm or one side of the body, and when general it is always most apparent in the hands. On closely observing the involuntary movements of the hand, it is noticed that the thumb closes on the fingers (as in spinning wool or rolling pills.) The head is the least affected, and often not at all. Speech is slow and jerky.

Stiffness of the muscles is a very conspicuous symptom, and is the chief cause of the motor weakness. A fixidness of the facial muscles gives a stolid expression to the features. There is a perceptible interference with the act of swallowing, which is probably caused by muscular rigidity rather than by the tremor. The fixed condition of the muscles in advanced cases, causes much difficulty in standing and walking, although the gross-muscular power is little impaired. The patient requires assistance to raise himself from the recumbent position. He is unable in bed to turn from one side to the other. He has to make an extra effort to rise from his seat, though when he is once on his legs he may still manage to walk well enough: but as the disease progresses he needs the aid of a stick to steady himself. At every step the body sags forward, and the patient is unable without help to regain the perpendicular, he is therefore compelled to quicken his gait more and more in order to avoid falling forward upon the face. He is thus forced into a run, which he cannot stop until he meets with some object that offers him a support. If he is still able to keep at a walk he makes the impression of continually seeking his center of gravity. This phenomenon is called "propulsion." Sometimes the patient has a tendency to run backward (retropulsion.) Such individuals are usually

seen to walk with the arms crossed behind the back; the object being to counterbalance the sagging forward of the body.

In well-marked cases of paralysis agitans a peculiar alteration in the attitude of the body develops which is typical of the disease, and by which it can be recognized in those exceptional cases where the trembling is insignificant or entirely absent. The stiffness has not only invaded the trunk, but also the muscles of the neck and some of the flexors and extensors of the arms and fingers. The head strongly bends forward, the trunk is inclined in the same direction; the upper arms are closely held to the side of the chest, the forearms are somewhat flexed so that the elbows stand off from the body, and the position of the fingers resemble that assumed in holding a pen. In rigidity of the lower extremities the legs are slightly flexed on the trunk, the knees are turned inwards and the heels are somewhat raised. In the condition of advanced muscular stiffness, the patient moves as if all the joints had lost their mobility.

In the commencement of the disease, patients sometimes complain of rheumatic-like pains especially in the shoulder. A subjective feeling of internal heat, is more common, though the thermometer shows no elevation of temperature. Frequently a profuse perspiration breaks out. Other incidental symptoms may develop during the course of the disease. Paralysis agitans often comes to a standstill, so that it may last for many years, but recoveries are exceptional.

Pathology. As paralysis agitans usually occurs in aged persons, it has been conjectured that senile decay may be the true cause of the disease. But post-mortem

examinations show no distinctive changes of the nervo-muscular apparatus. Probably the finer anatomical changes that exist escape our methods of investigation.

Diagnosis. The recognition of a typical case of paralysis agitans is easy. In the foregoing section the distinction from the tremor of multiple sclerosis has been pointed out. Senile trembling is less marked and of more limited extent than the tremor of paralysis agitans, and is not attended by muscular stiffness and paresis.

Treatment. It would be unprofitable to enumerate the various remedies that have been tried in this disease. In the early stage it may be possible to control the tremor. The prolonged employment of the subcarbonate of iron in large doses appeared to have been useful in a case reported by Dr. Elliotson. Charcot saw benefit from hyosciamine. Reynolds observed mitigation of the tremor from the wearing of a Pulvermacher chain. Beneficial effects are claimed for arsenic. Galvanism is sometimes of service in mild cases.

CHOREA.

(St. Vitus' Dance.)

Etiology. Chorea is most frequently observed to attack children between the periods of the second dentition and puberty. It predominates in the female sex. A direct hereditary transmission cannot be shown to exist, but it may be assumed that a constitutional susceptibility predisposes to its development. Psychical disturbances, especially fright, act as exciting causes in many cases. A similar mental influence operates in those singular cases resulting from imitation. Chorea often appears during the pregnancy of primiparæ. The disease is prob-

ably of a reflex character when induced by intestinal irritation, especially from worms. A causal relation appears to exist between chorea and rheumatism; or at least chorea sometimes co-exists with valvular disease of the heart which had been preceded by slight rheumatic symptoms.

Clinical History. The initial symptoms of chorea are usually misunderstood by parents and teachers. The affected children are often chided and even punished for having contracted certain objectionable habits and for indulging in silly behavior. These little patients let things drop from their hands, make all kinds of grimaces, continually shrug their shoulders, scribble when required to write and exhibit a constant restlessness. Very soon, however, these irregular movements and contortions are observed to be involuntary and to become general and aggravated as the disease advances. Volitional movements always start the jerks, but they also come on spontaneously. The motor disturbance may make long pauses when the patient is quiet, but in many cases they are continuous or appear in rapid succession. In severe cases, the whole body wriggles and assumes, with short respites, grotesque attitudes. Nearly every voluntary muscle may show incoördinate movements. If the facial muscles are affected, the brow wrinkles and the mouth is distorted. There is occasional winking of the eye-lids. The pupils are usually dilated. The tongue is suddenly thrust forward and as suddenly withdrawn. Speech becomes affected, swallowing is interrupted, strange sounds are produced by spasmodic action of the laryngeal muscles and the respiration is often irregular.

The choreic movements generally begin in the upper limbs and are there most prominent. The arms are flexed, extended, twisted and thrown about in every possible direction. The lower extremities are much less affected, but now and then one leg is thrown forward or sideways, or the knees suddenly bend as if the patient was about to fall. In some cases the trunk is also involved, so that the body leans for a moment to one side or is doubled up.

When the disorder is of a violent character it renders the patient completely helpless. He is unable to keep on his feet, and must be fed and dressed. The constant friction of the elbows and the knees against hard substances produces abraisons of the skin. The tossing and jerking may be so violent that the patient has to be fastened down in bed. There is, however, every conceivable grade of intensity of the disease. Some patients when they are quiet and attention is not directed to their movements appear to be but slightly affected. Choreic movements cease during sleep. In hemichorea, as the name imports, the motor disturbance is unilateral. It is remarkable that choreic patients never complain of fatigue caused by the incessant jactitations.

Increase of sensibility is sometimes noticed at the outset of the disorder, and later in its course a slight paresis and mental obtuseness may be observed in a few cases. Endocardiac murmur in connection with chorea is not always a sign of heart disease for the murmur may be merely a choreic symptom. The reflexes rarely show any alteration. The temperature is not elevated however severe the muscular contractions may be.

The termination is variable. Mild cases may subside in four or six weeks. The average duration of an attack is about three months, but sometimes the disorder is protracted for a year or more.

Pathology. Post-mortem examinations have thus far led no satisfactory explanation of the nature of chorea. There is hardly a doubt that some motor center is involved, but which part of the nervous system is affected, or what the character of the pathological condition may be is undetermined. Chorea must therefore be provisionally considered to come under the category of the neuroses. Foyers of softening, probably due to capillary embolism are occasionally found in the basal ganglia, but it is far from certain that these anatomical changes have any relation with true chorea.

A cerebral origin of the disease may be inferred from the occurrence of hemichorea and mental weakness.

Diagnosis. It is hardly possible to mistake chorea for any other affection. Choreic movements are entirely unlike the tremor of paralysis agitans, mercurial, saturnine or alcoholic trembling.

The **Prognosis** is favorable, but relapses often occur, and in very severe cases the general health seriously suffers.

Treatment. Choreic children should not be allowed to attend school. Any unnecessary allusion to the affection in the hearing of the patient ought to be avoided. The child should be guarded against doing injury to itself. Mild cases require little medicine. Bromide of potassium in rather large doses is decidedly of benefit. An anæmia should be improved by iron. A palpa-

ble intestinal irritation requires appropriate treatment. Arsenic is the approved remedy in acute and protracted cases. Five drops of Fowler's solution may be given three times a day. The arsenic is sometimes advantageously combined with a bromide. Da Costa found hyoscyamine in doses of gr. $\frac{1}{100}$ of much benefit in the insomnia caused by unremitting choreic movements. Valerianate of zinc and nitrate of silver are also recommended. Narcotics according to general experience are not suitable in chorea. Electric treatment very often disappoints. Chorea in pregnancy usually subsides after delivery. Cases are reported in which artificial delivery was necessary.

As an appendix to the subject of chorea a class of cases may be fittingly mentioned characterized by irresistible impulsive movements. The "dancing mania" "tarantism" and "electric chorea," which prevailed from time to time in Europe in an epidemic form belong to this category. Motor disorders of an analogous nature, exhibiting quasi-involuntary movements are occasionally brought under notice. These morbid phenomena totally differ from chorea. They probably belong to the category of hysterical, maniacal or epileptic paroxysms; or perhaps are allied to the phenomena of trance, ecstasy, etc. The following brief notes of a case, which I had opportunity to observe may exemplify the characters of these strange phenomena.

A girl nine years of age had suffered for the past two years as her mother told me, from "nervous attacks." The child in the midst of play would suddenly begin to make a noise like some animal, and then contort its body, throw about the arms in a wild manner, kick the furni-

ture, jump in a leap-frog fashion, scratch the walls, tear its clothes and perform many other strange antics. When I saw the child during one of these attacks, there was a constant discharge of saliva and dribbling of the urine; the pupils were dilated, but consciousness was intact. Any attempt to prevent the child from going through her maneuvres only increased their violence and duration. After their subsidence the child resumed its normal condition.

ATHETOSIS.

Dr. Hammond described under the name of athetosis very peculiar involuntary movements entirely differing from chorea. The affection is almost exclusively confined to children. There is an idiopathic form of athetosis of rare occurrence, which appears to be congenital and may be attended by mental weakness. Athetosis is sometimes a post hemiplegic symptom and may be associated with epilepsy.

The characteristic movements are most conspicuous in the hand and fingers as illustrated in figures 28 and 29. The fingers are never at rest. They are constantly either flexed, extended or intertwined in the most curious manner. The singular positions they assume is due to an irregular elongation from continued stretching of the articular ligaments. The arms and inferior extremities are less affected, but the toes sometimes are involved in movements analogous to those of the fingers. If the facial muscles participate in the movements, they are twisted and contorted. The head is thrown forward, backward and sideways, if the muscles of the neck are implicated.

No anatomical changes are observed in idiopathic athetosis. Probably the source of irritation is in the brain.

Improvements of the movements is sometimes obtained from galvanism, the bromides, or Fowler's solution.

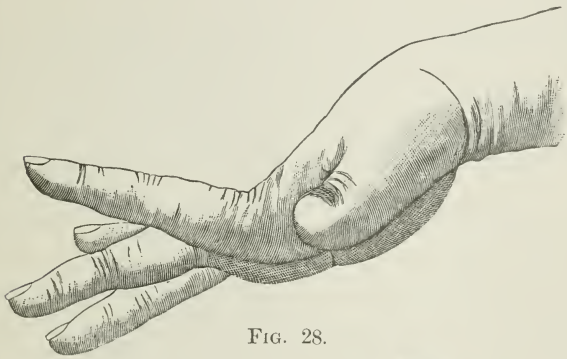

FIG. 28.

FIG. 29.

TETANY.

Etiology. Tetany is characterized by intermittent tonic tonic spasms of certain groups of muscles. The disease occurs in children and young adults. Trousseau found

tetany rather frequent in young nursing women. In many cases, a rheumatic influence is probably the exciting cause. A co-existent diarrhœa has also been observed. The disorder sometimes appears in an epidemic form among girls at school. Tetany has been known to follow the removal of the thyroid gland.

Clinical History. Certain precursory symptoms make their appearance. Pain is felt in the arms and a sensation of coldness and tingling in the fingers. After a few hours the seizure begins in the fingers, they become stiff and the hands the arms and inferior extremities in succession are affected with contractions. Spasm of the flexors predominate. The fingers approximate, the terminal phalanges are extended, the metocarpel ends are flexed, the thumb is strongly abducted and the palm is hollowed. This position of the hand resembles the peculiar form which it is made to assume by the accoucheur, when he introduces the hand into the vagina. In severe cases, the upper arm is pressed against the chest, the elbow is slightly flexed and the forearms are crossed over the epigastrium. In attacks of the inferior extremities, the toes are bent, and the feet are in the position of talipes equinus. The rest of the voluntary muscles are not often involved.

The muscles in the condition of tonic contraction feel very hard and stiff, and even after an attack they do not completely relax. Certain characteristic phenomena are observed during the intervals. The peripheral nerves are found very sensitive to electric excitation, violent contractions being evoked by weak currents. Mechanical stimulation of the muscles show an analogous excita-

bility. Thus, the facial muscles can be made to contract by stroking them with the hand. Trousseau discovered that in many cases energetic contractions can be excited by pressure of the arm in the regions of the median nerve and the brachial artery. As long as "Trousseau's sign" exists the recurrence of the paroxysms may be expected.

The frequency of contractions vary in individual cases. Sometimes there is a long pause between them, and in others they are nearly continuous. An attack generally lasts about two weeks. Prognosis is always favorable. No anatomical lesion has yet been detected to account for the disorder.

Diagnosis. A careful consideration of the symptoms which have been described as characteristic of tetany can hardly fail to render diagnosis easy. As tetany bears considerable resemblance to the very rare disorder called "arthogryposis" a few notes will be added here to point out the peculiarities of the latter disease.

Arthogryposis usually occurs in children, and may be attended by febrile symptoms throughout its whole course. Its essential symptoms consist of tonic spasms of one or of all four extremities. The arms are bent and the fingers flexed so as to assume fixed positions. The legs are either rigidly extended or drawn up to the body. Recovery in mild cases may take place in a few weeks, but a fatal termination usually happens in aggravated cases. The autopsy shows no distinctive lesion.

Treatment. The spasms in tetany are sometimes promptly relieved by applying the anode of the galvanic current to the different nerve trunks distributed to the affected muscles and the kathode to the sternum. Another method consists in passing the current through the spinal cord. The treatment is materially assisted by cold spong-

ing of the back and friction. The bromides, belladona and arsenic give no satisfactory results.

THOMSEN'S DISEASE.

(Congenital Myotonia.)

This disease was first described by Dr. Thomsen, a German physician, who suffered from it in his own person and observed it in several members of his family. A strong hereditary tendency existed in the not numerous cases that have come under notice.

The characteristic symptom of the affection is the occurrence of spasmodic rigidity of voluntary muscles when they are called into action after intervals of rest. The muscles of the inferior extremities are always affected. When the patient attempts to walk he succeeds for a little while, but very soon the muscles of the limbs get stiff, so that he must come to a halt. After a short time of rest the muscles relax, but the longer the muscles have rested the greater will be the difficulty of walking. No outward change in the condition of the muscles is observed; they rather appear to be very well developed.

Erb's recent investigations show that the individual muscular fibers are rounded, having lost their normal polygonal appearance, and that the interstitial tissue is increased. He also noticed that the myotonic reaction outlasts the direct electrical excitation of the muscles. The reflexes and the sensations are normal. No additional symptoms exist. Myotonia is a life-long disease. It baffles all therapeutical measures.

CHAPTER XV.

EPILEPSY.

Definition. The peculiarity of epilepsy consists in an inexplicable tendency of certain nerve centers to manifest, without an assignable cause, at irregular periods, a condition of excitement that gives rise to a typical form of general convulsions in individuals who, although they may have been subject to such attacks during a lifetime, show no distinctive lesion of the nervous system on post mortem examination. According to our present knowledge we must characterize epilepsy as a purely functional neurosis. Convulsive paroxysms occurring in the course of various diseases are symptomatic, and are therefore distinguished by the terms "epileptiform" or "epileptoid."

Etiology. Although the essential cause of epilepsy is unknown, we are acquainted with certain conditions and circumstances which act as predisposing causes. A hereditary influence can be traced in a large proportion of cases. The descent is more frequently on the mother's side. Often the transmission is not direct, but there exists a family tendency, which favors the development of the disease. This tendency is shown by the fact that members of the same family are prone to be affected with allied nervous affections. It may be insanity, dypsomania, hysteria, neuralgia, etc. Independent of a hereditary predisposition, it may be fairly assumed that there

are individuals of a neurapathic constitution in whom this condition is manifested by the development of epilepsy. Alcoholism in the parent is believed to predispose to this disease in the offspring. Genuine epilepsy is not caused by syphilis.

Among the exciting causes of epilepsy are understood those influences which tend to develop the disease in persons who are predisposed to it. The most important is sudden terror or fright. Continued worry of mind, anxiety and disquietude may act in a similar manner. In some cases the occurrence of the first attack has been preceded by declining health, anæmia, sexual excesses, sunstroke or an acute febrile disease.

If epileptic spasms occur after injuries to the head from a blow, fall or cut, they do not imply true epilepsy. In these traumatic forms of epilepsy, there is nearly always a cortical lesion, and the convulsive movements are usually unilateral. (Jacksonian epilepsy.) That form of epilepsy caused by wounds of superficial nerves, from splinters of glass or other foreign substances is the so-called "reflex epilepsy." In cases of this kind it is highly probable that patients manifest a strong tendency to neurotic affections. Reflex epilepsy can be artificially produced in rabbits, as Brown Sequard has demonstrated, by injuring the medulla oblongata or the spinal cord. Epileptic attacks are excited in these animals by irritating certain parts of the skin—"the epileptogenous zone." It is remarkable, that the progeny of the animals so operated upon are sometimes subject to spontaneous epileptic convulsions.

Clinical History. An epileptic seizure is sometimes preceded for hours or days by premonitory symptoms, chiefly

consisting of headache, vertigo, restlessness and psychical disturbance. In the greater number of cases the "warning" is experienced immediately before the attack sets in. Such premonitory symptoms, called auræ, consist of sensory motor, vaso-motor and mental disturbances.

1. The epigastric aura. This is one of the most frequent of premonitory symptoms. It is described by patients as a distressing sensation in the pit of the stomach, attended by nausea and precordial anxiety.

2. An aura of great frequency is a peculiar sensation beginning in the legs or arms, or in the region of the heart and suddenly "going to the head."

3. The auræ referred to the special senses relate to optical disturbances, as flashes of light, colored rings, sudden blindness; or spectral illusions, such as horrible faces, reptiles, but also enchanting scenes and pleasant visions. Hallucinations of hearing are less common. Some epileptics smell foul odors or have a metallic taste in the mouth, before an attack.

4. Motor auræ are of various kinds. An attack may be ushered in by "dragging in the face," a "slap on the head," a choking sensation, or a strain, as if going to stool.

5. The aura of a vaso-motor nature refers to a sudden feeling of chilliness, flashes of heat in the face or pallor, sweating or violent palpitations of the heart.

6. The most frequent psychical aura is a feeling of alarm and anxiety. The patient looks startled, frightened or amazed. Sometimes he is in a dreamy state, the past events of his life, crowding upon his memory.

These premonitory symptoms in individual cases are often variously combined. Sometimes they last long

enough to apprize the patient of the coming attack, but as a rule the convulsions immediately follow.

It is usual in describing the epileptic paroxysm to make the division of: 1. Epilepsia gravior or grand mal; 2. Epilepsia mitior, or petit mal; 3. Epileptoid conditions.

1. *Epilepsia Gravior.* An epileptic attack whether preceded by an aura or not invariably begins abruptly. Many patients utter a peculiar cry at the onset,—"the epileptic cry"—although they are already unconscious, and suddenly fall headlong to the ground. Consciousness and sensibility are completely abolished and tonic spasm of brief duration seizes the whole body. The head is strongly extended. the pupils dilated, the conjunctiva insensible, the jaws firmly closed; the tongue is frequently bitten and bloody froth accumulates at the mouth. Pallor of the face is well marked at the beginning. The back is arched as in aposthotenos, the arms are firmly flexed and the inferior extremities are rigidly extended. The respiratory muscles are involved, the breathing is arrested and the color of the face is bluish or dusky. This stage lasts from 15 to 30 seconds and is followed by the stage of clonic spasms.

The face in this stage assumes a hideous appearance. All the features are contorted; the eyes roll wildly in their orbits, the tongue is thrust out and convulsively retracted, the head beats upon the ground, and the arms, the trunk and the legs are violently convulsed. Sometimes the contents of the bladder and the rectum are evacuated and seminal emissions take place. The pulse and temperature remain normal, but during the venous stasis, the heart is tumultuous and the carotids throb.

Ecchymotic spots appear in the conjunctivæ and upon the skin in the region of the neck. This stage lasts from 1 to 10 minutes.

The third stage is characterized by the disappearance of the cyanosis and the gradual return to a natural respiration. The patient is still unconscious, but he begins to change the position of his body, he opens his eyes, looks about in a kind of a dazed condition and then soon passes into a slumber which may last a few hours. Some patients recover quickly, so as to be able to attend to their usual occupation in less than half an hour. Generally complete recovery from an attack is delayed much longer. For a day or two patients suffer from headache, lassitude and an irritable mental disposition. A large quantity of urine is often passed immediately after a seizure, which contains traces of albumen.

2. *Petit mal.* The mild forms of epilepsy may begin with or without an aura, and be variously manifested. Sometimes the petit mal merely amounts to a momentary confusion of mind, attended or not by slight twitching of facial muscles or contraction of the hands, which quickly subsides. Such a minor attack may stop a patient in the midst of a conversation, or while engaged in his usual occupation. Sometimes it is merely a fit of dizziness or faintness which rapidly passes off and leaves the patient unaware that anything unusual has happened to him. In others the whole attack may only consist of a sudden contortion of the face, or fixidness of the eyes, or a scream, or a reeling of the body. The patient knows nothing of it, and those who happen to witness these phenomena, may think them rather odd, but usually attach no importance to them. Very strange acts are

sometimes unconsciously performed by patients during minor attacks of epilepsy. Trousseau relates the case of a judge, who in open court would leave his seat and answer a call of nature in a corner of the room, then return to his place, entirely oblivious of what he had done. These "faints or spells" as these incomplete seizures are popularly called, rarely excite much alarm in the friends of the patient. The medico-legal bearing of such cases is sometimes very important. One of my patients often fell into the hands of the police for exposing his person in the public streets. He was at times afflicted with grand mal.

3. *The Epileptoid condition.* The symptoms under this head have not been unaptly called the "equivalents of epilepsy." They consist of aberrations of consciousness either of a paroxysmal and transient nature or succeed to a typical or rudimentary fit of epilepsy. Various psychical disturbances may immediately follow an attack which shows the intimate connection between them. But the nature of the mental derangement occurring independently of an attack would not be easily recognized if the individual were not known to be subject to epilepsy. Aggressive and even violent criminal acts may thus be committed in the epileptoid condition. Such a form of transient insanity is recognized by alienists. The diagnosis would hardly remain doubtful, if in addition to the brutal, ferocious, unprovoked act devoid of all known motive and which after its commission appears inexplicable and abhorrent to the accused, there is evidence that he had previous attacks of epilepsy. In children and very young persons, the epileptoid condition is commonly exhibited by unaccountable behavior, vicious

propensities and especially a remarkable tendency to incendiarism.

Course of the disease. Nocturnal epilepsy is a peculiarity of some importance, as the existence of the disease may be concealed for a long time if the attacks only occur during sleep. This condition may be suspected if patients continually suffer from sore tongue or frequently wet the bed. There is no uniformity in regard to the frequency of attacks. Some have only one or two during the year, others have them every month or daily. A similar difference obtains in regard to the severity of the seizures. Many patients have none but the grand mal; some have also minor attacks; and in another class, the very mildest form of the disease or the epileptoid condition predominates.

Genuine epilepsy develops in youth, rarely after thirty. The general health and the intellectual faculties may remain unimpaired through a life-long existence of the disease. When mental deterioration occurs, the memory is the first to suffer, which ultimately terminates in hopeless dementia. In very rare cases, the seizures become violent and recur in rapid succession. This "status epilepticus" is a very dangerous condition and usually ends in death. Various influences favor the recurrence of attacks. The baneful effects of alcoholism, venereal excesses, physical overwork and mental excitement are undeniable. On the other hand a peaceful life, moderation and genial surroundings exert a beneficial influence in reducing the repetition of the seizures. Epileptic girls sometimes improve, when they arrive at the menstrual period: more frequently this epoch develops

the disease. That marriage exerts a favorable influence on epilepsy, is not sustained by experience.

Pathology. Anatomical changes of the cerebral meninges and blood-vessels are not unfrequently found in long standing cases of epilepsy, but such morbid appearances are of a secondary character and are not uniformly present. Atrophy of the hemispheres is sometimes seen in subjects who died of epileptic dementia. Induration of the pes-hippocampi minor is more frequently seen than any other sort of lesion in epilepsy, but it is not a constant lesion. In the absence of a distinctive anatomical change that might give a clue to the nature of the disease we may perhaps come nearer a solution of the problem, if the starting point of the epileptic paroxysm could be determined. It was for a long time supposed, that the medulla oblongata, which is the general reflex center, is the region where the epileptic commotion originates. Nothnagel in more recent times, thought that he had found a "convulsive center" in the pons. The cortical origin of epilepsy finds at present most favor among neurologists. This view certainly agrees best with clinical facts. In the first place, the loss of consciousness, which is one of the essential symptoms of the epileptic attack, is a psychical phenomenon, and therefore a cortical disturbance. And again, some of the auræ are of a purely mental nature. In the second place, the spasmodic movements which are epileptic phenomena of equal importance occur in lesions of the cortex. "Cortical epilepsy" artificially produced in animals attacks the muscles in the same order which is observed in the epileptic seizure in man. This whole subject has been admirably discussed by Hughlings Jackson. But the

nature of that morbid irritation which spontaneously initiates the convulsive paroxysm of epilepsy is still a mystery.

Diagnosis. It is very unusual to mistake a case of epilepsy. Although the epileptiform convulsions in cerebral tumor, abscess and syphilis greatly resemble those of true epilepsy, yet the intermediate clinical history of organic brain disease decides the differential diagnosis. "Jacksonian epilepsy" is generally unilateral and always depends on a cortical lesion. Hysterical convulsions as already pointed out are never confounded with epilepsy, by the experienced physician. In uræmic spasms there is permanent albuminuria.

Treatment. In the rare cases of "reflex epilepsy" permanent benefit may follow surgical procedures. Trephining of the skull in superficial tumor or abscess has achieved some brillant cures. In genuine epilepsy no indications for causal treatment exist. There is no doubt that the frequency of attacks is lessened, if the patient observes a proper diet and regimen and practices regular habits of life. Alcoholic stimulants are decidedly injurious, even coffee and tea are better avoided. Indigestible food, heavy meals and in fact excesses of all kinds favor the occurrence of the attacks. A strict vegetable diet has many advocates.

Among the remedies employed in the treatment of epilepsy, there are certainly none which equal in efficacy the several bromides. The bromide of potassium is the one usually preferred. It should be given in large doses and continued for months and even much longer, if it be found of service. One drachm a day is the usual dose to begin with, but may be increased to the maximum dose

of 2½ dr. a day if the attacks are severe and frequent. Smaller doses suffice should improvement follow. The remedy should be much diluted in water to prevent irritation of the stomach, and for the same reason it is advisable to give the daily quantity in divided doses. The other bromides, the sodic, the ammonic and lithic may be advantageously combined with the bromide of potassium. One part of each of the sodic and ammonic with two parts of the bromide of potassium form a good combination. The bromide of sodium agrees better with delicate stomachs. The bromide of lithia contains the largest percentage of bromine, but is not superior to the other bromides. The quantity of these medicines must be diminished or wholly omitted for a time if brominism appears. Its symptoms are an eruption of acne, tremor, dyspepsia, impotence, cardiac weakness and dejection.

Belladonna enjoyed formerly a high reputation in Epilepsy, but is far inferior to the bromides. It is given in pills of the extract or in the form of atropia in equivalent doses of gr. $\frac{1}{120}$ three times a day. Dr. Gowers recommends a combination of belladonna with one of the bromides.

The oxide of zinc is an old remedy for fits. From 2 to 3 grains combined with gr. ½ of belladonna extract and 15 grains of valerian root powder, three times a day, is a favorite prescription with good observers after failure of the bromide treatment.

Borax has lately been much lauded in doses from 10 to 15 grains three times daily.

It is needless to do more than allude to other remedies that are recommended for epilepsy, such as nitrate of silver, arsenic, indigo and preparations of copper, as they

are rarely of benefit. Electricity does not appear to exert any influence in epilepsy.

Various popular measures are in use to prevent an attack when the patient has a "warning," such as slapping the palms of the hands, a brisk walk, and stuffing the mouth with salt. Nothing should be done during the attack except to guard the patient against injury. If the tongue is caught between the teeth, a gentle effort should be made to release it.

MÉNIÈRE'S DISEASE.

Aural Vertigo.

Etiology. Chronic disease of the ear, affecting the labyrinth and semi-circular canals is supposed to be the cause of the peculiar disease to which attention was first drawn by Ménière. It is sometimes attended by otorrhœa and deafness.

Clinical History. The chief symptoms of the disease are tinnitus aurium and excessive vertigo. It begins with shrill voices heard in one or both of the ears, accompanied by dizziness, nausea and vomiting. Later, the paroxysms occur frequently, at short intervals, and finally the vertigo is so continuous and severe that the patient has to keep to his bed. During the exacerbations of the vertigo, the patient has a sensation as if his body were whirled about in every possible direction, and with such a force that he clutches at surrounding objects to prevent falling to the ground. Towards the end of the attack a very distressing feeling of nausea comes on, the face is pallid and cold, and the body is covered with perspiration.

After having progressed for several years complete deafness ensues, and with its appearance all the other

symptoms cease. Ménière's disease may be confounded with epilepsy and disease of the cerebellum.

Treatment. Special treatment of the ear, when called for is of primary importance. Quinia appears to give always relief. From 10 to 15 grains should be given daily. Politzer reports remarkable results from hypodermic injections of pilocarpin.

HYSTERIA.

Etiology. The multifarious and bizarre phenomena, which cannot be referred to a definite pathological change, but which bear a stamp and present a physiognomy that the experienced physician recognizes at a glance lend to hysteria the character of a singular disorder but one of great clinical importance. Symptoms, capricious, erratic and anomalous, ranging from a mild group of functional nervous disturbances up to an apparent formidable implication of important nerve centers, and frequently disappearing suddenly without leaving a trace behind, show that no tangible morbid condition affects a particular organ or tissue. The fact that hysterical manifestations are eminently excited by psychical influences strongly supports the view that the nervous system, and especially the higher centers are affected with a peculiar irritability or with defective inhibitory power. Hysterical persons certainly exhibit an impressible disposition which is either hereditary or acquired. This neuropathic condition greatly preponderates in the female sex. It is not difficult to understand why the delicacy of the nervous apparatus in women readily reacts in an undue manner to exciting causes that hardly affect the stronger sex. Hysterical symptoms in girls frequently

make their first appearance at the period of pubescence. Of a similar influence is the recurrence of the menstrual molimen. Uterine and ovarian troubles, chlorosis and anæmia often lay the foundation of a life-long hysteria. But the prominence formerly attributed to disturbances of the sexual organs in the female as exciting causes of hysteria is not sustained by experience. That boys are not unfrequently affected with hysterical symptoms cannot be disputed. Even men under the influence of shock or strong emotional excitement sometimes go through a paroxysm of genuine hysteria. A tendency to the development of hysteria must be assigned to the injurious effects of mental strain, especially of a depressing nature. To this category of exciting causes belong deep anxiety, concealed sorrow, disappointment and harsh treatment. It is alleged, that the frivolities and artificial excitement in which females in affluent circumstances indulge augment the number of hysterical patients. But women in humble walks of life are certainly not exempt. A deleterious influence must also be attributed to the ill-directed training of little girls, who give intimation of a precocious disposition, or betray odd or morbid susceptibilities. Very remarkable and puzzling are the histories of hysterical individuals, who nurse and pet without a sinister motive some spurious ailment, that subjects them to a miserable state of existence and actual deterioration of health in order to keep up the rôle they assume. It would be difficult to account for such phenomena were it not for the fact, that they represent phases of hysteria. Probably some of this class of hysterical persons might be brought to confess that the undue anxiety and slavish ministration spent

upon them, has fostered their morbid craving for sympathy and their desire to become the objects of interest and solicitude.

The hysterical element is also strongly developed in cases where a slight hurt or insignificant accident concentrates attention upon a particular part of the body. It is usually a large joint or the spine where the hysterical neurosis locates.

It would be an error to suppose that hysteria is confined to anæmic, feeble and broken down women. The exhibition of decided hysterical symptoms in women otherwise healthy and vigorous is a matter of common experience.

Clinical History. Considering the diversity of hysterical symptoms even in the same patient it would be a questionable undertaking to give a description of a typical case of the disorder. But the recurrence of a uniform order of symptoms or a particular one in the same patient is quite often observed.

Hysterical convulsions. Not every hysterical female is subject to convulsions, though an attack is prone to develop under the influence of strong mental excitement. In some cases there is a brief prodromic stage characterized by a feeling of lassitude, epigastric uneasiness, a choking sensation, fluttering in the region of the heart and vague pains in different parts of the body. Sometimes these symptoms stop short and terminate in a fit of spasmodic crying or laughter.

The convulsive movements in hysteria are of a tumultuous character. The limbs are either wildly thrown about, or the arms and hands are contorted and twisted. The facial muscles are not affected, but the patient

makes all kinds of grimaces, expressive of anger, fright or terror. There is sometimes unconsciousness but the mind is usually unimpaired. The pupils are normal.

In another class of cases, tonic contractions are prominent. The arms are bent, the fingers semiflexed, and the inferior extremities extended. An attack often consists of partial spasm of a limb or some group of muscles. Laryngeal spasm interferes with breathing. Sometimes the respiration is remarkably rapid, and the heart beats violently. Spasm of the pharynx and of the œsophagus is believed to give rise to "globus hystericus," which patients compare to a ball rising or sticking in the throat. Spasm of the diaphragm causes incessant hiccough, often accompanied by severe pain in the pit of the stomach. Hysterical cough has a harsh, brassy sound, unattended by expectoration. The loud movement of gases in the intestines causes the annoying "borborygmæ." A great variety of other forms of hysterical spasm occur which hardly need mention.

A case comes now and then under notice in which the hysterical and epileptic elements seem to be blended. French authors give frightful pictures of "hystero-epilepsy." They are seldom witnessed in this country.

A genuine hysterical fit is easily distinguished from epilepsy. There is seldom complete coma in hysteria, no initial cry, the pupils are not dilated, the face is not cyanotic, there is no biting of the tongue, and the spasmodic contractions have not the rhythmical jerk of epilepsy. Besides, the duration of hysterical convulsions is indefinite.

Psychical characteristics of hysterical individuals. It is especially in the sphere of emotional life, the sensibilities,

the mood, temper and disposition, that the peculiarities of the hysterical element become manifest. The unbalanced state of the emotions is exhibited in sudden transitions from gayety to sadness. There is a passiveness to the play of fancy and whim; singular likes and dislikes arise; sometimes an unaccountable apathy and stolid indifference or an overcharged enthusiasm and a highly wrought pathetic sentimentality is exhibited. There may be a morbid desire to create interest and to attract sympathy. Slight impressions produce exaggerated effects. The inclinations and the will seem to be perverted, as shown in uncontrollable obstinacy and waywardness. Hysterical individuals who are more or less affected in this manner are an enigma to their friends and acquaintances, and despots in their families. But hysterical females can render themselves very agreeable if they like. There is sometimes a fine languor in the expression of their eyes and a supplicating tone of the voice, which novelists are fond of depicting. Hysterical individuals are usually of a bright intelligence. They are often well aware of the oddities and fancies that frequently possess them, but they ingeniously explain them away by a high-colored description of their manifold complaints.

Hysterical paralysis. The paralysis in hysteria is generally of the paraplegic type, but hemiplegia is not rare. Most frequently the paralysis suddenly develops after a mental shock or a strong emotional disturbance. A peculiarity of hysterical paralysis is the ability of the patient to move the limbs well enough when in bed or reclining, but when requested to rise she immediately sinks to the floor. One is tempted to suggest that the patient might easily get up and walk by an effort of

the will, but this command of the will is just what is wanting. The paralysis is evidently of cerebral origin. Another peculiarity of hysterical paralysis when only one limb is affected is the characteristic gait. The paralyzed limb does not describe an arc when carried forward, but the patient makes a long stride with the sound limb and the other is dragged along.

Hysterical aphonia is often observed. The paralysis of the vocal cords comes on suddenly. Its existence is revealed by the laryngoscope. The reflex excitability of the pharynx is sometimes lowered and the glottis cannot be completely closed. In other hysterical individuals, usually in young girls, the alteration of the voice consists in the utterance of shrill, harsh sounds.

Hysterical dysphagia probably depends on spasm rather than on paralysis of the œsophagus. The tube can be passed after a brief trial.

Contracture of groups of muscles is sometimes an isolated hysterical symptom, or it is associated with paralysis. Most frequently one arm or the hand is affected. The arm is rigidly contracted and the fingers are intertwined in a curious manner. Hysterical contractures are readily relieved by morphia injections or chloroform inhalations.

Hysterical anæsthesia. One of the most remarkable symptoms of hysteria is complete hemianæsthesia generally of the left side. The loss of sensibility is accurately defined by the median line of the body. All the qualities of common sensation are abolished. The patient does not feel the nearness of a lighted candle, nor the prick of a needle. This analgesia explains

the mutilations of the body, which hysterical individuals have been known to practice with the purpose of eliciting sympathy or for a sinister motive. In another class of hysterical patients, the anæsthesia is confined to limited areas of the skin, affecting also the neighboring mucous membranes. Disturbances of the special senses are often associated with anæsthesia ; — amblyopia, amaurosis, achromatopsia and narrowness of the field of vision. There may be impairment of hearing, of taste and smell on the affected side. A very singular form of paralysis of the muscular sense has been described by Duchenne. The patient is unable to tell the position of a limb when her eyes are closed.

Hyperæsthesia is a very frequent accompaniment of hysteria. It is often associated with spasmodic contractions. Touch or pressure in many parts of the body elicits pain. The head, the shoulders, the chest and the abdominal walls appear to be highly sensitive, which suggests to the patient the existence of some serious internal disease. Usually the patient exaggerates this sensitiveness, as is discovered when her attention is distracted. Whether neuralgia, which may be present, is hysterical or independent of the disorder is difficult to decide. Intercostal pain is the most constant form of hysterical neuralgia and ovarialgia is sometimes intense. A species of pain peculiar to hysteria is the so-called "clavus hystericus." It is usually limited to a small spot along the sagital suture. Hyperæsthesia of special sense exists when the patient complains of painful sensitiveness to light or of acute hearing. A perversion of the gustatory sense in hysterical girls explains their relish for unsavory substances and their dislike to ordinary articles of food.

Hypnotism. Hypnotic phenomena which can be artificially produced in a certain class of people occasionally occur spontaneously in hysterical individuals. The very obscure nature of these phenomena surrounds them with much mystery. Since Braide's investigations in hypnotism medical men have begun to overcome their aversion to a subject which had been so much mixed up with charlatanry and deception. A variety of strange conditions of the nervous system are induced by different methods, such as stroking the head with the hand, requesting the person to count numbers, to look continually at a bright object, and the like. A condition of lethargy is produced in which the subject is semiconscious and exhibits a remarkable contractile irritability. A faint blow upon a nerve causes a spasmodic contraction of the corresponding muscle. Charcot in his cases of this description observed a cataleptic condition in which he could fix the limbs and position of the body in every conceivable way. Of greater interest are the cases of "suggestion." The hypnotized person under this influence is affected with hallucinations in which he assumes attitudes and goes through movements in correspondence with the special suggestion. By a change of the manipulations the hypnotized person passes into a state of somnambulism. He is half conscious, describes visions and does whatever he is bid.

A number of nervous disorders affecting different organs remain to be mentioned. Frequent attacks of palpitation are very alarming to hysterical patients. They can usually be traced to a previous mental excitement. The pulse is occasionally exceedingly rapid without an apparent cause. Attacks resembling angina pectoris

have been repeatedly noticed. "Hysterical hæmoptisis" and "hysterical hæmatemesis" belong also to this category of anomalous symptoms; and, stranger still, blood escapes into the skin (the so-called "stigmata"). These spontaneous hemorrhages cause great apprehension, though leading to no serious consequences. Digestive disturbances are often well marked. "Nervous dyspepsia" is extremely obstinate, and seems to baffle all remedies. "The phantom tumor" is merely a partial distension of the abdominal walls, and if very extensive may simulate pregnancy. Ischuria is a very remarkable hysterical symptom. Days pass by and no urine is discharged, though there is no retention. Sometimes violent vomiting comes on, and examination of the contents of the stomach discovers the presence of urea. It is highly probable that many cases of obstinate dysmenorrhœa are of a hysterical origin.

Course of the Disease. It will be observed from the foregoing enumeration and description of the medley of symptoms recognized as hysteria, that the clinical history of this affection must include a host of other disorders, dependent as they are on an unstable and vulnerable nervous system. For the same reason it cannot be predicted what different ailments will arise during the course of the disease. Experience has taught that whilst hysterical symptoms may entirely disappear for an indefinite period, an exciting cause, usually some emotional disturbance, rekindles the morbid tendency. It may happen that none of the more severe forms of the disorder are present in some patients, but they are continually harassed by some of its minor ailments. We see hysterical women who are never satisfied with their state of

health; they are always invalids. Some special complaint may predominate. It may be nervous dyspepsia. dyspnœa, palpitation or a worrying pain in some part of the body. On the other hand, it is peculiar to hysteria that severe symptoms, such as paralysis, contractures, hemianæsthesia or convulsive paroxysms, disappear suddenly and spontaneously.

Diagnosis. Hysteria presents a physiognomy of symptoms that betrays their true character. Their very puzzling nature, that does not conform to the etiology, clinical history and course of well-known individual diseases, is significant. Hysterical paralysis as pointed out differs from ordinary forms of paraplegia and hemiplegia dependent on gross nervous lesions. It should however be recollected that hysterical people are subject, like other mortals, to diseases which bear no relation to their nervous trouble.

Treatment. A protest must be entered against the cruel indifference with which the ever returning ills and ailments of the hysterical are generally regarded. The physician who shares this error is certainly blamable. Banter and ridicule are entirely out of place. Hysteria is not a dangerous malady, it does not kill, but it is a most distressing affliction. To deal with it successfully requires a tact on the part of the physician, which not every one has at command. If he succeeds in gaining the confidence of the patient, which is best accomplished by unaffected sympathy combined with firmness he will have a comparatively easy task before him.

Moral therapeutics therefore takes the first rank in the treatment of hysteria. Seeing that mental excitement is often the *vera causa* of the most severe forms of the disorder,

such prophylactic measures should be recommended that guard against the influences which develop and keep up hysterical manifestations. In the management of inveterate cases it may become necessary to change the surroundings of the patient and to secure rest by seclusion in which a rational treatment can be systematically carried out.

Some of the popular measures for cutting short a hysterical fit are often quite effectual, but stuffing the mouth with salt or throwing a pailful of water over the head of the patient should not have the sanction of the physician. It will suffice to splash some cold water in the face, or if the patient is very violent to irrigate the head for a few minutes at a time. Severe measures are at any rate not called for in a disorder which ceases of its own accord and is unattended with danger. What may be called "habitual hysterical fits" are best controlled by stern reproof. Firm pressure in the region of the hyperæsthetic ovary sometimes succeeds in checking an attack. Hysterical paralysis yields better to moral treatment, when conducted with tact and perseverance, than to any other measure. The patient is to be persuaded to make at least some effort to overcome her muscular weakness. An intelligent nurse can be of far more assistance than any one of the family of the patient. As soon as the patient has gained some confidence in her ability to help herself, improvement will soon follow. This treatment can be advantageously aided by electricity, massage and cold douches to the spine.

Hysterical aphonia is often successfully treated by electricity. The electrodes of a galvanic current are to

be placed to the sides of the neck, or Mackenzie's electrode is introduced into the larynx.

Gynecologists claim good results in hysteria from the treatment of uterine disturbances. It may readily be admitted that benefit has been derived from correcting a displaced uterus, or from the relief of ovarian trouble, but in numerous cases such treatment has utterly failed to cure the hysteria.

The employment of remedies against the multitude of nervous disorders in pronounced hysteria is a thankless task. But even under the unpromising condition of having to deal with a constitutional diathesis, the physician can be of great service to his patient if he succeeds in inspiring confidence in his effort to afford relief. The effects of moral therapeutics is sometimes seen in the "cures" from some highly advertised "infallible remedy," or the passes and manipulations of the "electro-magnetizer."

In cases associated with anæmia or a decline of the general health a tonic and an invigorating treatment is indicated. Cold baths and douches and general galvanization will often be found of service.

Among the "anti-hysterical" remedies it is only valerian that sustains some sort of reputation, because it is not quite as offensive as assafœtida. The bromides are sometimes very useful. Narcotics should be sparingly given, or better not at all if not urgently called for. Paraldehyde from ʒss to ʒi is occasionally quite effective in calming hysterical excitement. Hemianæsthesia is best treated with the faradic brush.

HYPOCHONDRIASIS.

Etiology. Hypochondriasis chiefly affects men, but typical examples of the malady are occasionally observed in women. It is always a chronic affection, though it never endangers life. Apparently there seems to be an affinity between hypochondriasis and melancholia, inasmuch as the alteration common to both is characterized by a depressed mental condition, but it would be erroneous to class hypochondriasis with insanity, for the intelligence remains clear in the former, and it is only an excessive anxiety and apprehension manifested by the hypochondriac in regard to his ill-defined symptoms which develop his fanciful notions.

Clinical history. The essential clinical feature of hypochondriasis is a morbid disposition to fix the attention on slight ailments, which are magnified and falsely interpreted by the patient. The anomalous sensations of which he constantly complains are undoubtedly real; they absorb his thoughts, leave him no rest, and render him sad and peevish. Besides these indistinct sensations, which probably originate in the abdominal viscera, there are other symptoms of a more palpable nature. They chiefly consist of an uneasiness felt in the epigastric region, a burning sensation about the umbilicus, flatulence, disordered bowels, palpitations, flushes of heat in the face and frontal headache. Each of these morbid sensations, as they more or less bother the patient, is a source of alarm to him. He watches, studies and bewails them. By and by he locates them in a particular organ or some part of the body, and becomes convinced that he is the victim of a serious disease. The shifting character

of the vague feelings is probably the reason why hypochondriacs imagine they are successively affected by a variety of diseases. Now it is the stomach, then the liver, next the heart, the lungs, the bowels, the bladder, the rectum or spinal marrow. This change in the situation of the fancied disorder is often suggested by the names of diseases about which these patients hear or read. They continually change their medical advisers and are voracious consumers of drugs. Some are in fear of losing their sexual power, and if ever they have had syphilis or imagine they had it, become confirmed syphilophobes.

Treatment. Although hypochondriac people are exceedingly fond of taking medicines and sometimes declare they are benefited, yet they never acknowledge a cure. There is no use to direct treatment against any special symptom, for ten others will take its place. Attention should nevertheless be paid to the digestive organs, which are frequently disordered. Vegetable tonics, such as calisaya bark, cascarilla, columbo and gentian, with the addition of a mineral acid, answer very well. As torpidity of the bowels is a standing complaint of these patients, it is advisable to prescribe an occasional pill of aloes and nux vomica, or the compound liquorice powder if hemorrhoids exist. An active life, devoted to a regular occupation which involves considerable physical exertion, is often of greater service than all medication. Good results are occasionally obtained from "moral therapeutics." An effort may be made to wean the patient from his gloomy thoughts and brooding over his ailments. According to circumstances, he may be urged to enter a political career, or take an interest in a popular philanthropic

cause, attend to vestry meetings, engage in a scientific pursuit, or cultivate one of the fine arts. Extensive traveling, which breaks in upon the monotonous routine of life, is sometimes beneficial. Such strategy has in some instances proved successful.

Griesinger relates the case of a young woman who fancied that her intestines protruded through the partly opened abdominal walls. Her physician did not dispute this, but told her that it is not unusual for the abdominal muscles, when they are weak, to permit the intestines to come to the surface. He ordered an apparatus to support the parts, and had the satisfaction by these means not only to cure her of her false idea, but also relieved the constipation of the bowels, from which she had suffered for a long period.

CHAPTER XVI.

PARETIC DEMENTIA.

(General Paresis of the Insane.)

It is of great practical importance to the general physician to be familiar with the peculiarities of this disease, which, under the disguise of mental weakness and alteration of the moral tone, affects individuals in the prime of life, and tends to progressive deterioration of both body and mind. Outside of the profession the fully established disease is called by the ominous name of "softening of the brain."

Etiology. Paretic dementia makes the heaviest inroads among those classes of society where the feverish activity of modern civilization is conspicuous. The disease attacks men between the ages of thirty and forty years. Women are much less liable to be affected. A hereditary tendency if at all existing is not evident. Of greater etiological importance is the connection of a history of syphilis with a large proportion of cases. It is a significant fact that the disease mostly occurs in men living in large cities. Its victims constitute a high percentage of the inmates of our insane asylums.

Clinical History. Certain periods may be distinguished in the course of paretic dementia which mark its several stages, but the order of occurrence of the essential symptoms in numerous cases is too variable to permit of such

a strict division of its clinical history. The simultaneous and successive pathological changes of important nerve centers give rise to psychical, motor and sensory disturbances that observe no uniformity of development. Nevertheless a typical case presents ample diagnostic points.

The disease always begins with a precursory stage. Prodromic symptoms develop so insidiously that often no satisfactory information can be obtained in regard to the time when they first attracted notice. The condition of impaired mental capacity is chiefly manifested by a commencing failure of the memory and laxity of the moral sense. This alteration is a puzzle to the family and associates of the patient. He exhibits an unusual levity, neglects his business, becomes fond of spirituous liquors, visits disreputable houses and gives various indications of changed habits. He mislays valuable papers, is inattentive to important appointments, gives orders to his assistants which he forgets and thus disarranges his affairs. Delinquencies of which he is guilty at this period are partly due to his thoughtlessness and partly to his moral debasement. He may for this reason commit a theft or forge a check with little compunction. The sedate *paterfamilias* shocks by his loose language and rakish demeanor. The promising young man violates the ordinary rules of propriety and decorum. He enters a course of alcoholic and sexual excess. If he is reproached for his conduct he takes little pain to set himself right, or offers some unplausable reason in extenuation.

The paretic dement is generally intolerant of contradiction, flies into a passion about trifles, but shows a remarkable equanimity at the miscarriage of his affairs.

He engages in foolish and ruinous enterprises, spends his money lavishly, and readily enters into objectionable matrimonial alliances. It would be difficult to prove that such a person is insane, but he is certainly fast drifting into it.

Symptoms of a different order frequently make their appearance during this period. The patient is troubled with headache, dizziness, momentary confusion of the mind and a feeling of general illness. He becomes alarmed concerning the state of his health and is vaguely conscious of some deterioration of his former mental capacity. He is depressed, emotional, or a true melancholia develops. The physician, who is consulted at this stage would probably misinterpret the symptoms, if the family, who know more about the condition of the patient did not give the proper information.

The development of very marked psychical and somatic symptoms may be considered the second stage. A close examination might have noticed before this a peculiar defect of speech, but this symptom is now more apparent. A tremulous movement of the lips and tongue attends the utterance of words, as if they had to overcome an impediment: the words come out with an explosion. The brow corrugates, the zygomatic muscles are fixed, the nostrils alternately contract and dilate. Very characteristic are the patient's blunders in pronouncing difficult words, as "artrallirary" for artillery, "cletircal" for electrical. He makes use of wrong words. His handwriting has also deteriorated. It is coarse and does not keep to the line, single letters and whole syllables are omitted. A similar incoördination affects his hands and fingers, especially noticeable in mechanical employments.

If the patient is an artist, he has lost nicety and delicacy of touch. There is an unsteadiness of the gait. The patient throws his limbs too wide apart. Dancing, skating, swimming and the like accomplishments are awkwardly performed, or no longer practicable. If the patient is tested in reckoning or in drawing up accounts his lapses and glaring mistakes are of diagnostic value.

Pupillary changes or paralysis of external ocular muscles constitute significant initial symptoms. They are rarely absent in advanced cases. Inequality of the pupils or reflex immobility (Argyle Robertson pupil) and ptosis are often observed.

If in connection with the symptoms that have been described, there is loss of the patellar reflex, migraine, and neuralgia of the extremities, the co-existence of locomotor ataxia may be suspected.

As the mental alienation progresses, there is a development of expansive delusions and extravagant projects corresponding to fancies of exaltation and grandeur. This character of the delusions is not peculiar to general paresis, but is observed in this disease with great frequency and distinguished by an absurdity that overleaps all bounds of the possible. The monomaniac may fancy that he is a king, an apostle, or a millionaire; he acts the assumed rôle and maintains his pretensions against all comers. But the paralytic dement declares that he is King of all Kings, the possessor of all the gold mines in the world, Napoleon, or God Himself—all in one breath. He is never struck by the bare incongruity of these fictions, personates them but indifferently and is easily persuaded to drop them—though only to take up others

just as senseless and ridiculous. Even when melancholia tinges the delusions, they partake of the absurd. Such a patient may say that he only measures an inch in height, that he is three-cornered and died yesterday. The delusions of the female paretic turn upon matters congenial to her sex. She boasts of possessing thousands of silk dresses, is confined with twins every day and the like.

The paretic dement not only braggs of his enormous wealth and high distinction, but also of his muscular strength and fabulous sexual power. He can lift the roof off the building with the greatest ease; he has married the finest women in town and his genital is two feet long. Erotic delusions are particularly common in male patients. The subjective feeling of an exuberance of health and well-being is another of the marked features of the disease. If the paretic dement is asked how he feels—he answers "first-rate," "never felt better in his life," he is "all right." Generally he is good-humored and cheerful and appears to be one of the happiest mortals. Some are, however, ill-natured and occasionally combative. The position in life and education of the patient modifies the delusions. A driver of a furniture wagon, whom I examined before his admission into the hospital, told me that his credit at the Savings Bank would keep him and his family for ten life-times, and then related with great gusto, how he "used up" a man with whom he had had a quarrel. This tendency to give revolting details of imaginary encounters has often been noticed in the paretic dement.

The extravagant projects of patients frequently involve delusions of ludicrous and impossible schemes of amassing untold wealth.

Paroxysmal attacks of mania and epileptoid and apoplectiform seizures constitute notable features of the disease.

The maniacal paroxysms generally develop at a late period, and sometimes even at an early date. In very rare cases, they are nearly continuous and may prove fatal from nervous exhaustion, but usually they resemble acute delirium. The mental excitement in many cases is that of simple mania, of a violent, outrageous and malevolent character. Patients sing, shout and are destructive. They are filthy, constantly spit and besmear themselves with their excrements. In their imbecile fury they make desperate assaults, and are considered the most troublesome inmates of asylums. Their brutality is sometimes exhibited in outbreaks after release from their confinement. They use violent and abusive language and threaten those who had a hand in their incarceration with cruel penalties. A maniacal paroxysm may last for hours or days and is succeeded by a brief stupor.

Epileptoid attacks, appear towards the termination of the disease. Usually the spasms are unilateral and rarely present the violence of a true epileptic fit. A patient often remains in this condition for days and recovers from it. Consciousness is not always impaired.

Apoplectiform attacks like the epileptoid seizures occur towards the close of the disease, though in some cases they are witnessed at an early period. The sudden loss of consciousness may happen whilst the patient is engaged in conversation. The head is hot and the face flushed like in cerebral hyperæmia. But apoplexy from cerebral hemorrhage may also occur and prove fatal. I once attended a man who had apparently improved after hav-

ing exhibited unmistakable symptoms of paretic dementia. He became a frequent visitor of restaurants and club-rooms, from which places he was several times carried home in a condition of congestive apoplexy. He died eighteen hours after the last seizure during which time he did not recover his consciousness. Although patients usually get over these episodical attacks of mania and epileptoid and apoplectiform seizures, they constantly lose ground after each attack.

Before the last stage of the disease is reached, very remarkable remissions of prominent symptoms are occasionally witnessed. Improvement may be so evident, that the family of the patient conclude to remove him from the asylum under the impression that complete recovery has taken place. Such a patient may again be capable of attending to ordinary affairs and enjoy the liberty and comforts of home. But experience has taught that relapses are common, and that these invalids are not only liable to give way when undertaking the management of complicated transactions, but are apt to become involved in enterprises that may sink a fortune.

The downward tendency of the disease is marked by progressive obscuration of the intelligence and continued increase of the paretic condition, until the patient is reduced to an utter wreck of body and mind. When this third and last stage is reached he may be carried off in a few months by the "galloping" form of the disease. The fatal termination is sometimes postponed for two or three years. Death is often hastened by the development of malignant decubitus.

Anatomical Changes. Very marked post-mortem appearances are found in subjects, who have died in the

advanced stage of the disease. Symmetrical parts of the brain and spinal cord are involved in a degenerative process, that probably originated in a low inflammation. The skull in many places is thickened. Bony plates are sometimes found in the adherent dura mater. Hemorrhagic pacchymeningitis quite often exists. Some portions of the brain are entirely atrophied, leaving gaps between the unaffected parts. Cortical areas are slightly softened or indurated. The ganglion cells in these regions show marked degeneration. A cystic formation is very frequently noticed, consisting of minute cavities, which are due to dilatation of perivascular spaces in the gray and white substance. The basilar portion of the brain is affected to a much less extent than the convexity, but the pons and the medulla show partial atrophy. The axis cylinders of the nerve fibres are coarse and distended. Very decided changes are constantly found in the neuroglia, from slight increase of connective tissue to complete sclerosis. The blood-vessels of the affected parts are twisted and tortuous. The adventitia and the muscular coat have undergone degeneration. To this condition of the vessels must be attributed the venous stasis and thrombic coagulations in the substance of the brain. Nuclear or "free bodies" are seen in the lymph spaces, some of which are transformed into spider cells. Analogous changes are found in the spinal cord. The adhesions of the arachnoid and dura mater to the subjacent parts of the cord present the appearance of a meningo-myelitis. A fascicular degeneration often involves the posterior columns as in loco-motor ataxia. Sometimes the sclerosis is diffuse and has implicated the cells of the anterior cornua.

The gross changes in the brain and the spinal cord explain the general nature of the symptoms, but what can be said in regard to those cases of the disease, in which no such morbid alterations are found. It has been suggested, that disturbance of the cerebral circulation initiated by vaso-motor influences which leave no traces, originates the symptoms, and that the degeneration of important nerve-centers takes place in the more advanced cases of the disease.

Diagnosis. In the matter of diagnosis the great practical importance of recognizing the early signs of the disease is evident. Alteration of the mental condition, especially on the moral side is significant. It is not usual for a man of settled habits of life to change them. The evidences of such a change vary in different individuals, but they all indicate the complication of an enfeebled memory, shallow reasoning, moral laxity and weakness of the will. Palpable errors in reckoning, a deteriorated hand-writing, tremulous lips in the act of speaking and pupillary changes are valuable diagnostic points. Of much importance are the episodical paroxysms of mania and the epileptoid and apoplectiform attacks. Usually the diagnosis of a typical case of paretic dementia causes no embarrassment. Uncertainty will be felt in cases resembling ordinary mania, brain tumor, cerebral syphilis and especially atypical examples of multiple sclerosis.

The Prognosis is exceedingly unfavorable. Cases of recovery do occur, but hundreds of demented paralytics die yearly in our hospitals.

Treatment. Therapeutics holds out but faint hopes in a disease dependent on destroying lesions of important

nerve centers. In the early stage it may be possible to arrest the disease in some cases, and the effects of iodide of potassium should at least be fairly tested. Alcoholic stimulants and sexual excesses should be strictly prohibited. Above all, it is the bounden duty of the attending physician to apprise the family or friends of the patient of the true state of the case, and of the risk he runs of raising scandal and effecting his pecuniary ruin, if the proper precautions be not taken. There should be no hesitation to recommend the seclusion and care of an asylum as soon as the disease is recognized. The prompt and systematic treatment at an early period in such an establishment offers the best chance of benefiting the patient.

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FORMULÆ.

Hypodermics. Tablets of morphia gr. $\frac{1}{8}$ to $\frac{1}{4}$ and of atropia gr. $\frac{1}{120}$ to $\frac{1}{60}$ are now conveniently employed for hypodermic injections. They readily dissolve in water. Magendie's solution of morphia containing one grain in 30 minims is also well adapted for hypodermic use.

The cerebral effect of morphia is antagonized by combining it with atropia. Tablets containing the proper proportions of these remedies are now offered for sale by reliable druggists. The following formulæ are recommended:

℞ Morphiæ Sulphatis - - - gr. vi
Atropiæ Sulphatis - - - gr. $\frac{1}{5}$
Aquæ Destillatæ - - - ℥ ss M
Sig. Inject 10 M. or more according to circumstances.

℞ Strychniæ - - - - - gr. i
Aquæ Destillatæ - - - ℥ ss M
Sig. Inject from 5 to 10 minims.

℞ Codeiæ - - - - - - gr. iv
Aquæ Destillatæ - - - - ℥ ss M
Sig. Inject M xxx

Headache. Insomnia. ℞ Antipyrini - - - - - ℈ ij
Ft. in capsulas no. iv.
Sig. one every 3 hours or one at bedtime.

or

Headache. Insomnia.

℞ Antipyrini - - - - - ℥ ii
Syr. Aurantiæ cort. - - - ℥ ss
Aquæ Aurantiæ flor. ad - - ℥ ij M
Sig. A dessert spoonful every one or two hours until three to six doses are taken.

℞ Tinct. Cannabis Indicæ - - gtt. xv
Chloral Hydratis - - - gr. x
Potass. Bromid. - - - gr. xx
Elix. Smpl. - - - - ℥ i M
Sig. Take before going to bed.

Hemicrania.

℞ Tinct. Cannabis Indicæ - - gtt. x
Eth. Sulph. - - - - - gtt. xxiv M
Sig. Take in a tablespoonful of water.

℞ Urethani - - - - - gr. viii
Confect. Rosæ - - - - gr. xv M
Ft. massa et. in pil. No. xxiv div.
Sig. From one to three pills.

Insomnia.

℞ Paraldehydi - - - - ℥ iii
Alcoholis - - - - - ℥ iss
Spir. Lavandul. Comp. - - ℥ ii
Syr. Smplicis - - - ad ℥ iv M
Sig. One dessertspoonful every hour until sleep is produced.

℞ Sulfonali - - - - - gr xx—xxx
Sig. Take in black coffee or in syr. aurant.

Insomnia.

℞ Camphor Monobromatis - - ℈ i
Make into 10 pills covered with gelatine.
Sig. Take one or two pills every two hours.

Headache. Insomnia.

℞ Caffein Citratis - - - ℥ ss
Syr. Limonis - - - - ℥ ss
Aquæ - - - - ad ℥ ii M
Sig. One teaspoonful every three hours.

℞ Efferv. Bromo-Caffeini
Sig. One teaspoonful in a wineglassful of water every hour or two.

℞ Guaranæ - - - - - gr. x—xx
Pulv. Aromat - - - - - gr. v
Sig. Take every 2 hours until relieved.
Hemicrania.

℞ Phenacetini - - - - gr. iii
Caffein Citratis - - - gr. iss
Sacch. Lacti - - - - gr. v M
Sig. Take every two hours until the headache is relieved.

℞ Tinct. Opii Deodorat - - - gtt. xxx
Tinct. Digitalis - - - - gtt. x M
Cerebral Congestion

℞ Pulv. Opii - - - - gr. i
Quiniæ - - - - gr. iii M
Make into two pills.
Cerebral Anæmia.

℞ Ext. fl. Conii - - - M xx
Sig. At one dose to be followed by from five to ten minims every hour until the excitement is subdued.
Maniacal Excitement.

℞ Chloral. Hydratis
Potass. Bromidi - - āā ℥ ss M
(One full dose acts better than frequent small doses.)

Headache. Insomnia.

℞ Ammonii Chloridi - - - ℥ iii
Morphiæ Acet. - - - gr. i
Caffein. Citratis - - - - ℥ ss
Spirt. Ammoniæ Aromat - ℥ i
Elix. Guaranæ - - - - ℥ iv
Aquæ Rosæ - - - - ℥ iv M

Sig. One dessertspoonful every quarter hour until relieved.

Bilious headache after debauch.

Debout's Pills.

℞ Quiniæ Sulphatis - - - ℥ i
Pulv. Digitalis - - - - gr. viii

Make into twenty pills.

Sig. One every four hours.

Neuralgia.

℞ Quiniæ Sulphatis - - - ℥ i
Morphiæ Sulphatis - - - gr. ii

Ft. massa in pill No. xxx div.

Sig. One pill thrice daily and gradually increased until nine are taken.

℞ Quiniæ Sulphatis - - - ℥ i
Morphiæ Sulphatis
Acid Arseniosi - - - āā gr. iss

Ft. massa in pill No. xxx div.

Sig. Taken as the foregoing.

℞ Fl. Ext. Gelsemini
" " Black Cohosh - āā ℥ i M

Sig. Four drops every hour. The dose should be diminished if drooping of the eyelids is noticed.

℞ Exalginæ - - - - - gr. v to x

Taken in capsule.

BORSON'S PILLS.

Neuralgia. ℞ Pulv. Moschi - - - gr. x
Pulv. Digitalis - - - - gr. v
Opii - - - - - - gr. iv
Make into ten pills.

Sig. One every four hours.

℞ Aconitini (*Duquesnill*) - - gr. $\frac{1}{10}$ to $\frac{1}{6}$
Glycerinæ
Alcoholi - - - - āā ℥ i
Aqua Menthæ pip q. s. ad ℥ ii M

Sig. One teaspoonful two or three times a day.

Facial Neuralgia.

℞ Phenacetini - - - - gr. x
In capsule.

Sig. Every hour until relieved.

℞ Antipyrini - - - - - gr. LXXV
Aquæ Destillatæ - - - - ℥ ijss
Spts. Lavandulæ Comp. - - ℥ iii
Syr. Limonis - - - q. s. ad ℥ iv M

Sig. One dessert-spoonful three times a day.

℞ Croton Chlorali - - - ℥ i
Glycerinæ - - - - -
Syr. Aurantiæ - - - - āā ℥ i M

Sig. One teaspoonful per dose.

℞ Ol Terebinthinæ - - - - ℥ ii — iii
Mucilage Accaciæ - - - q. s.
Ft. Emulsio et adde
Syr. Zingeber - - - - ℥ i
Aquam - - - - - ad ℥ iv M

Sig. One tablespoonful every four to six hours.

Sciatica.

Neuralgia. ℞ Strychniæ - - - - - gr. ss
Aquæ - - - - - - ℥ ss M
Sig. Eight drops in a dessert-spoonful of water three times a day.

Sciatica.

℞ Ext. Nux Vomicæ - - - gr. i
Mannæ - - - - - ʒ i
Ft. massa et in pil. No. viii div.
Sig. One pill three times a day.

℞ Ammon. Chloridi - - - ℥ ij
Ext. Fl. Glycyrrhizæ - - - ℥ ss
Syr. Aurantiæ - - - - ℥ i
Aquæ - - - - - ad ℥ vi M
Sig. One tablespoonful three times a day.

Epilepsy. ℞ Potass. Bromidi - - - ℥ ss
Sig. In a wineglassful of water.

℞ Potass. Bromidi - - - gr. xv
Sodæ Bromidi
Ammoniæ Bromidi - - āā gr. v M
Sig. In a tablespoonful of water.

℞ Potass. Bromidi - - - - ʒ i
Tinct. Digitalis - - - gr. v — x
Elix. Simplicis - - - - ℥ ss M

℞ Potass. Bromidi - - - - ʒ i
Sol. Fowleri - - - - gtt. iii M

℞ Potass. Bromidi - - - - ʒ i
Tinct. Belladonnæ - - - gtt. x M
Sig. Three times a day.

℞ Sodæ Boracis - - - - gr. xv — xxx
Glycerinæ - - - - gtt. v
Aquæ Menth. pip. - - - ℥ ss M
Sig. Three times a day.

Nervine Stimulants.

℞ Spirt. Aetheris Comp.
Tinct. Valerianæ Ammon. - āā ℥ i M
Sig. One teaspoonful with a tablespoonful of water.

Hysteria.

℞ Spirt. Ammon. Aromat. - - ℥ i
Syr. Aurantiæ - - - - ℥ ii M
Sig. One dessert spoonful every hour.

Nervousness.

℞ Spirt. Vini Gallici - - - ℥ ss
Tinct. Opii Deodorat. - - gtt. xv — xxx M

Nervous Shock.

℞ Aetheris Chlor. - - - ℥ i
Pulv. Acaciæ - - - - ℥ ss
Aquæ - - - - - ℥ iv M
Sig. One tablespoonful every two hours.

Hysteria.

℞ Spirt. Lavandulæ Comp.
Tinct. Cardamom. Comp. - āā ℥ ss
Syr. Aurantiæ - - - - ℥ i M
Sig. One dessert spoonful in a wineglassful of water.

Restorative.

℞ Camphoræ - - - - gr. xxiv
Sapo-medicat - - - - q. s
M et divid in pillulas - - viii
Sig. One three times a day.

℞ Camphoræ - - - - gr. xxiv
Aetheris Sulph. - - - - ℥ ss M
Sig. Twenty drops in a wineglassful of water every half hour.

Nervine Stimulants.

℞ Ext. Quebracho - - - ℥ j
Sig. Twenty to thirty drops three times a day.
Hysterical Asthma.

Stimulant and Sedative Applications.

℞ Linimentum Chloroformi - ℥ iii
Tinct. Iodini
Tinct. Aconitii - - - āā ℥ ii
Tinct. opii - - - - ℥ss M

℞ Methyl Chloridi pulv. - - - ℥ i
Sig. Apply with a brush, or a pledget of lint to the painful part.

℞ Menthol - - - - - gr. iiss
Cocaini Muriatis - - - - gr. viiss
Chloral Hydratis - - - gr. iv
Vaselini - - - - - ℥ iiss M
Sig. Apply to painful part and cover with a strip of court-plaster.
Supraorbital Neuralgia.

℞ Carbonis Bisulphidi - - - ℥ iv
Pulv. Camphoræ q. s. ad solut. satur.
Sig. Apply with a brush to the painful parts.
Lumbo-abdominal Neuralgia.

℞ Ol. Terebinthinæ - - - ℥ viii ss
Chloroformi
Tinct. opii - - - - āā ℥ ii M
Sciatica.

℞ Ext. Hyosciami
Ext. Balladonnæ - - āā ℥ i
Glycerinæ - - - - ℥ i M
Irritable Testicle.

℞ Methyl chloridi
Sig. For a spray to the vertebral column.
Spinal Irritation. (Huchard.)

Stimulant and Sedative Applications.

℞ Atropiæ Sulphatis - - - gr. v
Aquæ Destill. - - - ℥ iii M
Sig. Soak a compress with some of the solution and apply for an hour.

Neuralgia (Trousseau).

℞ Ext. Belladonnæ - - - - ℥ i
Tinct. opii - - - - ℥ i
Glycerinæ - - - - - ℥ iii M
Sig. A piece of lint wet with this mixture is applied to the neuralgic nerve. Cover the part with oil silk.

℞ Veratriæ - - - - - gr. xxiv
Glycerinæ - - - - - ℥ ii
Spir. Rectific - - - - ℥ vi M

℞ Collodii Cantharid. - - - ℥ iv
Sig. Paint the skin.

Miscellaneous.

℞ Magnesiæ Sulphatis - - - ℥ iv
Acidi Sulphurici dil. - - ℥ i
Ferri Sulphatis - - - - gr. xv
Syr. Aurantiæ - - - - ℥ ss
Aquæ Menthæ pip. - - - ℥ vii M
Sig. Two tablespoonfuls twice daily.

Tonic Aperient.

℞ Ergotin (Beaujou) - - - ℥ ss
Ft. pillulas no. x.
Sig. One ter. in die.

℞ Strychniæ - - - - - gr. i
Tinct. Ferri Chloridi - - ℥ ss
Tinct Colombæ - - - - ℥ i
Aquæ - - - - - ad ℥ iv M
Sig. One teaspoonful three times a day.

Nervous Dyspepsia.

Miscellaneous. ℞ Tinct. Digitalis - - - - ℥ iss
Ext. Fl. Ergoti - - - - ℥ iiss
Syr. Aurantiæ - - - - ℥ ss M

Sig. One teaspoonful every eight hours.

Essential Vertigo.

℞ Auro-Chloridi - - - - gr. i
Ext. Gentiani - - - - gr. xv
Ft. massa et in pill, No. xxx div.

Sig. One three times a day.

Loco-motor Ataxia.

℞ Acid Arseniosi - - - - gr. i
Mass. Ferri Carbonatis - - ℥ i
Quiniæ Sulph. - - - - ℥ ss
Ft. massa et in pill. No. xl div.

Sig. One pill three times a day.

Nervous Dyspepsia.

℞ Hydrarg. Chloridi Corrosivi - gr. i
Glycerinæ - - - - - ℥ i
Tinct. Cinchon. Comp. - - ℥ ii
Spirt. Ol. Menthæ pip. - - - M xxiv M

Sig. One teaspoonful in a wineglassful of water ter. in die.

Vertigo of the aged.

MEGLIN'S PILLS.

℞ Ext. Hyosciami
Zinc Oxidi - - - - āā ℈ ii M
Divide into forty pills.

Sig. Begin with one pill and increase by one pill every other day.

Tic doloreux.

Miscellaneous.

FOURNIER'S PILLS.

℞ Zinci Valerianæ - - - - - gr. v
Ext. Opii - - - - - - gr. iss
Ext. Hyosciami - - - - - gr. iiss
Confect. Rosæ - - - - - q. s.

Divide into six pills.

Sig. One pill every three hours until three are taken, then omit the pills, and take them again in the same way the next day.

Neuralgia.

PROF. S. GROSS' PILLS.

℞ Quiniæ Sulph. - - - - - ℈ ii
Morphiæ Sulph. - - - - gr. i
Strychniæ - - - - - - gr. $\frac{2}{3}$
Acid Arseniosi - - - - - gr. i
Ext. Aconitii - - - - - gr. x

Divide into twenty pills.

Sig. Take one pill three or four times a day.

Neuralgia.

BROWN-SEQUARD'S PILLS.

℞ Ext. Belladonnæ - - - gr. $\frac{1}{6}$
" Stramonii - - - gr. $\frac{1}{5}$
" Cannabis Indicæ - - - gr. $\frac{1}{4}$
" Aconitii - - - - - gr. $\frac{1}{3}$
" Opii - - - - - - gr. $\frac{1}{3}$
" Hyosciami - - - - gr. $\frac{2}{3}$
" Conii - - - - - - gr. i
Pulv. Glycyrrhizæ - - - q. s.

Make into one pill.

Sig. Take from three to four pills a day.

Brown Sequard has observed no great constitutional disturbance from the use of these pills (?)

Inveterate Neuralgia.

GENERAL INDEX.

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V

ERRATA.

Page 26—4th line from top, for "nerves" read *cornua*.

Page 180—"Lockhart Clarke" (one name).

Page 209—3d line from top, for "hemianospia" read *hemianopsia*.

Page 310—7th line from top, for "Trosseau" read *Trousseau*.